Tim Mair, Sandy Love,
Jim Schumacher,
Roger Smith & Grant Frazer

Equine Medicine, Surgery and Reproduction

Second Edition

SAUNDERS

Equine medicine, surgery and reproduction

Senior Content Strategist: Robert Edwards
Senior Content Development Specialist: Catherine Jackson
Senior Project Manager: Sukanthi Sukumar
Senior Designer: Greg Harris
Illustration Manager: Jennifer Rose
Illustrator: Antbits Ltd

Equine medicine, surgery and reproduction

Second Edition

Edited by

Tim S Mair

Bell Equine Veterinary Clinic, Mereworth, Maidstone, Kent, UK

Sandy Love

Chair of Equine Clinical Studies, School of Veterinary Medicine, University of Glasgow, Glasgow, UK

Jim Schumacher

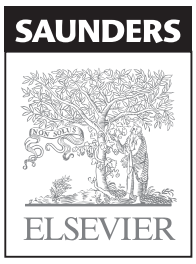
Department of Large Animal Medicine and Surgery, University of Tennessee, Knoxville, Tennessee, USA

Roger KW Smith

Department of Clinical Science, The Royal Veterinary College, Hatfield, Hertfordshire, UK

Grant S Frazer (deceased)

Department of Large Animal Theriogenology, College of Veterinary Medicine, The Ohio State University, Ohio, USA



Edinburgh London New York Oxford Philadelphia St Louis Sydney Toronto 2013

SAUNDERS
ELSEVIER

© 2013 Elsevier Ltd. All rights reserved.

No part of this publication may be reproduced or transmitted in any form or by any means, electronic or mechanical, including photocopying, recording, or any information storage and retrieval system, without permission in writing from the publisher. Details on how to seek permission, further information about the Publisher's permissions policies and our arrangements with organizations such as the Copyright Clearance Center and the Copyright Licensing Agency, can be found at our website: www.elsevier.com/permissions.

This book and the individual contributions contained in it are protected under copyright by the Publisher (other than as may be noted herein).

First edition 1998
Second edition 2013

ISBN 978 0 7020 2801 4

British Library Cataloguing in Publication Data
A catalogue record for this book is available from the British Library

Library of Congress Cataloging in Publication Data
A catalog record for this book is available from the Library of Congress

Notices
Knowledge and best practice in this field are constantly changing. As new research and experience broaden our understanding, changes in research methods, professional practices, or medical treatment may become necessary.

Practitioners and researchers must always rely on their own experience and knowledge in evaluating and using any information, methods, compounds, or experiments described herein. In using such information or methods they should be mindful of their own safety and the safety of others, including parties for whom they have a professional responsibility.

With respect to any drug or pharmaceutical products identified, readers are advised to check the most current information provided (i) on procedures featured or (ii) by the manufacturer of each product to be administered, to verify the recommended dose or formula, the method and duration of administration, and contraindications. It is the responsibility of practitioners, relying on their own experience and knowledge of their patients, to make diagnoses, to determine dosages and the best treatment for each individual patient, and to take all appropriate safety precautions.

To the fullest extent of the law, neither the Publisher nor the authors, contributors, or editors, assume any liability for any injury and/or damage to persons or property as a matter of products liability, negligence or otherwise, or from any use or operation of any methods, products, instructions, or ideas contained in the material herein.

ELSEVIER your source for books, journals and multimedia in the health sciences
www.elsevierhealth.com

Working together to grow libraries in developing countries
www.elsevier.com | www.bookaid.org | www.sabre.org
ELSEVIER BOOK AID International Sabre Foundation

The publisher's policy is to use **paper manufactured from sustainable forests**

Printed in China

Last digit is the print number: 9 8 7 6 5 4 3 2

Contributors

Alistair RS Barr MA VetMB LLB PhD DVR CertSAO DEO DipECVS MRCVS
Professor of Veterinary Surgery, School of Veterinary Science, University of Bristol, UK

William V Bernard DVMDiplomate ACVIM
Internist, Owner/Manager, Lexington Equine Surgery and Sports Medicine, Lexington, Kentucky, USA

Thomas J Doherty MVB MSc Dip ACVA
Professor, Department of Large Animal Clinical Sciences, College of Veterinary Medicine, University of Tennessee, Knoxville, Tennessee, USA

G Barrie Edwards (deceased) BVSc DVetMed FRCVS
Professor, Department of Veterinary Clinical Science and Animal Husbandry, University of Liverpool, South Wirral, UK

Kira L Epstein DVM DACVS DACVECC
Associate Professor, Department of Large Animal Medicine, College of Veterinary Medicine, University of Georgia, Athens, Georgia, USA

Nicholas Frank DVM PhD DACVIM
Professor of Large Animal Internal Medicine and Chair, Department of Clinical Sciences, Tufts Cummings School of Veterinary Medicine, North Grafton, Massachusetts, USA; Associate Professor of Medicine, School of Veterinary Medicine and Science, University of Nottingham, Nottingham, UK

Grant S Frazer (deceased) BVSc
Department of Large Animal Theriogenology, College of Veterinary Medicine, The Ohio State University, Ohio, USA

Kelsey A Hart DVM PhD DACVIM
Assistant Professor, Large Animal Internal Medicine, Department of Large Animal Medicine, College of Veterinary Medicine, University of Georgia, Athens, Georgia, USA

Marcus J Head BVetMed MRCVS
Senior Associate, Rossdales Diagnostic Centre, Suffolk, UK

Kristopher Hughes BVSc FACVSc DipECEIM
Senior Lecturer in Equine Medicine, School of Animal and Veterinary Sciences, Charles Sturt University, Wagga Wagga, New South Wales, Australia

Leo B Jeffcott MA BVetMed PhD FRCVS DVSc
Professor, Department of Clinical Veterinary Medicine, University of Cambridge, Cambridge, UK

Raphael Labens Mag. Med. Vet MVM DEC-EQ CertES(Orth) DACVS
Large Animal Hospital, College of Veterinary Medicine, North Carolina State University, USA

J Geoffrey Lane BVetMed DESTS FRCVS
Cedars Surgical Services, Axbridge, Somerset, UK

Sandy Love BVMS PhD MRCVS
Chair of Equine Clinical Studies, School of Veterinary Medicine, University of Glasgow, Glasgow, UK

Tim S Mair BVSc PhD DEIM DESTS DipECEIM AssocECVDI MRCVS
Bell Equine Veterinary Clinic, Mereworth, Maidstone, Kent, UK

Margaret C Mudge VMD DipACVS DipACVECC
Associate Professor of Equine Emergency and Critical Care, College of Veterinary Medicine, The Ohio State University, Ohio, USA

Yvette S Nout DVM PhD DipACVIM DipACVECC
Assistant Professor – Equine Sciences, Department of Animal and Veterinary Sciences, College of Agriculture, California State Polytechnic University, Pomona, California, USA

Robert Pascoe
Veterinary Surgeon, Bell Equine Veterinary Clinic, Rochester, UK

Carlos Pinto MedVet PhD DACT
Associate Professor and Service Head, Theriogenology and Reproductive Medicine, The Ohio State Veterinary Medical Center, Ohio, USA

Patrick J. Pollock BVMS CertES(Soft Tissue) DipECVS MRCVS
European and RCVS Recognised Specialist in Equine Surgery, Weipers Centre Equine Hospital, School of Veterinary Medicine, University of Glasgow, Glasgow, Scotland

Bonnie R Rush DVM MS DACVIM
Professor, Equine Medicine, Department of Clinical Sciences, Kansas State University, Manhattan, Kansas, USA

Contributors

Michael CA Schramme **DrMedVet CertEO PhD HDR DipECVS DipACVS LA Associate of the European College of Veterinary Diagnostic Imaging**
Professor of Equine Surgery, University of Lyon, VetAgro Sup – Veterinary Campus of Lyon, Department of Equine Studies, France

Jim Schumacher **DVM MS DipACVS MRCVS**
Department of Large Animal Medicine and Surgery, University of Tennessee, Knoxville, Tennessee, USA

John Schumacher **DVM MS DipACVIM**
Department of Clinical Science, College of Veterinary Medicine, Auburn University, Auburn, Alabama, USA

Colin C Schwarzwald **DrMedVet PhD DipACVIM DipECEIM**
Professor of Equine Internal Medicine, Section Head, Internal Medicine Section, Equine Department, Vetsuisse Faculty, University of Zurich, Zurich, Switzerland

Debra C Sellon **DVM PhD DipACVIM**
Professor of Equine Medicine, Department of Veterinary Clinical Sciences, Washington State University, Pullman, Washington, USA

Charlotte Sinclair **BVSc Cert EP Cert ES (Orth) MRCVS**
Staff Clinician in Equine Diagnostic Imaging, Department of Clinical Science, The Royal Veterinary College, Hatfield, Hertfordshire, UK

Roger KW Smith
Department of Clinical Science, The Royal Veterinary College, Hatfield, Hertfordshire, UK

Joseph S Spano (deceased) **DVM PhD**
Department of Pathobiology, College of Veterinary Medicine, Auburn University, Auburn, Alabama, USA

David GM Sutton **MA VetMB CertEM(IntMed) DECEIM PhD MRCVS**
Senior University Clinician in Equine Medicine, Weipers Centre Equine Hospital, School of Veterinary Medicine, University of Glasgow, Glasgow, UK

Ramiro E Toribio **DVM MS PhD DACVIM**
College of Veterinary Medicine, The Ohio State University, Ohio, USA

Alexander Valverde **DVM DVSc Dip ACVA**
Associate Professor, Department of Clinical Studies, Ontario Veterinary College, University of Guelph, Guelph, Ontario, Canada

Linda J Vogelnest **BVSc (Hons) MACVSc FACVSc**
Specialist Veterinary Dermatologist, Hospital Superintendent Camden, The University of Sydney, Veterinary Teaching Hospitals, Camden & Sydney, Australia

Martin P Weaver **BVMS PhD DVR DrMedVet(Munich) MRCVS***
Senior Lecturer in Equine Surgery and Orthopaedics, Royal (Dick) School of Veterinary Studies, Easter Bush Veterinary Centre, Roslin, UK

Renate Weller
Senior Lecturer in Large Animal Diagnostic Imaging, Department of Clinical Science, The Royal Veterinary College, Hatfield, Hertfordshire, UK

*Deceased

Preface

The art is getting longer and longer, the brain of the student not bigger and bigger

Hippocratic aphorism, c.400 BC

It's over ten years since the first edition of *Equine Medicine, Surgery and Reproduction* was published. Over that time there have been tremendous advances in many areas of equine veterinary science. It is timely, therefore, for a new and up-dated edition of the book to be published. Our aims in producing this book are the same as they were for the first edition – a single volume user-friendly text covering the major areas of equine medicine, surgery and reproduction. As before, all of the authors are highly experienced in clinical teaching and are internationally-recognised experts in their fields. The book is aimed at veterinary undergraduates and general practitioners, but we hope that others, including more specialist equine clinicians and professionals in allied fields will find it of use.

In addition to up-dating all of the original chapters, the scope has been increased by the addition of new chapters on wound management, sedation and anaesthesia, diagnostic imaging, and critical care. This book has had a long gestation, and, sadly, two of our authors died prior to the book being published. Both Professor G Barrie Edwards and Dr Grant S Frazer were dear friends as well as highly respected clinicians and teachers; this volume is dedicated to their memory.

Chapter 1

Upper alimentary system

J. Geoffrey Lane, Robert Pascoe

CONTENTS

1.1 Normal upper alimentary tract function: deglutition 2
Oral, pharyngeal and oesophageal phases of deglutition 2
Prehension 2
Mastication 2
Lingual function 2
Elevation of palate 2
Pharyngeal constriction 2
Laryngeal protection 2
Crico-pharyngeal relaxation 2
Primary and secondary oesophageal peristalsis 2
1.2 Diagnostic approach to cases of dysphagia 2
History – signs of dysphagia 2
Physical examination, external and oral inspection 3
Endoscopy *per nasum* 3
Radiography and fluoroscopy 4
Oral examination under general anaesthesia 4
1.3 Aetiology of dysphagia: oral phase abnormalities 4
Facial palsy and lip lesions 4
Temporo-mandibular joint and hyoid disorders 4
Lingual abnormalities 5
Dental disorders 5
Congenital and acquired palatal defects 5
Other oral conditions: foreign bodies, neoplasia 5
1.4 Aetiology of dysphagia: pharyngeal phase abnormalities 5
Pharyngeal paralysis 5
Pharyngeal compression: strangles abscessation 5
Pharyngeal cysts, palatal cysts 5
Epiglottal lesions, including sub-epiglottic cysts 5
Laryngeal abnormalities 6
Fourth branchial arch defects (4-BAD) 6
1.5 Aetiology of dysphagia: oesophageal phase abnormalities 6
Megaoesophagus 6
Oesophageal obstruction ('choke') 6
Oesophageal strictures/stenosis 6
Intra-mural oesophageal cysts 6
Oesophageal rupture 6
Neoplasia 6
'Wind-sucking' 6
Grass sickness 7
1.6 Oral trauma, mandibular fractures etc. 7
1.7 Oesophageal obstruction 7
1.8 Anatomy of the oral cavity 8
Oral cavity 8
Normal dental anatomy 8
Triadan system 9
Eruption of teeth 10
1.9 Abnormalities of wear – abrasion and attrition 10
1.10 Periodontal disease 12
1.11 Dental caries 13
1.12 Endodontic disease including dental abscessation 14
1.13 Tumours of the upper alimentary tract 15
Odontogenic tumours 15
Other tumours of the jaw 15
1.14 Diagnostic approach to dental disorders 15
Ageing of horses by dentition 15
Clinical signs of dental disease 15
Oral examination 16
Radiography of teeth 16
Other ancillary diagnostic techniques 17
Indications for dental extraction 17
Options for the extraction of incisors, canines and wolf teeth 18
Options for the extraction of cheek teeth 18
Further reading 20

© 2013 Elsevier Ltd
DOI: 10.1016/B978-0-7020-2801-4.00004-3

1.1 NORMAL UPPER ALIMENTARY TRACT FUNCTION: DEGLUTITION

Normal deglutition comprises the prehension and mastication of ingesta followed by its transfer from the oro-pharynx to the stomach.

Oral, pharyngeal and oesophageal phases of deglutition

Deglutition is divided into three stages:

1. The **oral phase** – which includes the gathering of food, movements within the oral cavity, mastication and the formation of boluses of ingesta at the base of the tongue – is under voluntary control.
2. The presence of a bolus gathered at the tongue base triggers the sequence of reflexes, collectively known as swallowing, which propels the ingesta from the pharynx – the **pharyngeal phase** – into the oesophagus. The glosso-pharyngeal nerve (IX) and the pharyngeal branches of the vagus (X) innervate the pharynx and larynx, and their afferent and efferent pathways are co-ordinated in the swallowing centre in the brainstem.
3. Waves of peristalsis convey the ingesta along the oesophagus to the stomach – the **oesophageal phase** of deglutition.

Prehension

Prehension in the horse relies on the incisor teeth to grasp and section herbage and on the lips to pick up smaller pieces of ingesta as well as to contain it within the mouth and to manipulate food towards the cheek teeth.

Mastication

The molar and premolar teeth are responsible for the mechanical crushing of the fibrous diet.

The tongue and buccal musculature assist in manipulating the ingesta between the maxillary and mandibular dental arcades.

Mastication requires free opening and closure of the temporo-mandibular joints (TMJs) through the action of the masticatory muscles – the masseter, pterygoid and temporal muscles close the jaws, and gravity, assisted by the digastric muscles, opens them. The masticatory muscles receive their innervation through the mandibular branch of the trigeminal nerve (V).

The shape of the articular surfaces of the TMJs together with the presence of menisci permit lateral movements by the mandibular teeth across the wearing surfaces of the upper cheek teeth.

Lingual function

The tip of the tongue assists in prehension and moves the ingesta between the cheek teeth.

Contraction of the tongue base helps in the formation of boluses and, once collected, each bolus is driven caudally; this triggers the involuntary phases of deglutition by driving food and fluid caudally from the oro-pharynx.

The tongue is attached to the hyoid apparatus, and free movement at the tympano-hyoid articulation is required for the craniocaudal tongue motion which facilitates bolus formation in the oro-pharynx.

The glossal musculature receives its motor supply via the hypoglossal nerve (XII).

Elevation of palate

The action of the levator palatini muscles draws the soft palate dorsally to close off the naso-pharynx and prevents the nasal reflux of ingesta; this marks the onset of the involuntary stages of deglutition.

The horse has an intra-narial larynx at all times other than during the momentary disengagement for deglutition. (See 5.18 and 5.21.)

The levator palatini muscles lie parallel with the drainage ostia of the auditory tube diverticula (ATDs) so that when they contract the ostia open to allow exchanges of air for pressure equilibration across the ear drum.

Pharyngeal constriction

The constrictor action of the circular muscles of the pharyngeal walls embraces both oro-pharynx and naso-pharynx – the latter can be appreciated during endoscopic examinations of the naso-pharynx. A wave of constriction follows the contraction of the tongue base and passes from rostral to caudal efficiently to empty the oro-pharynx – the pharyngeal 'stripping' wave – leaving minimal quantities of ingesta at the base of the tongue.

Laryngeal protection

Aspiration of food and fluid through the rima glottidis is prevented primarily by the tight adduction of the vocal folds and arytenoid cartilages and to a lesser extent by the retroversion of the apex of the epiglottis.

Crico-pharyngeal relaxation

The upper oesophageal sphincter is formed by the thyro- and crico-pharyngeus muscles, and these are maintained in a state of contraction to prevent involuntary aerophagia, especially during forced exercise. Relaxation of the crico-pharynx simultaneous with the pharyngeal stripping wave permits the food and fluid boluses to pass caudally into the proximal oesophagus.

Primary and secondary oesophageal peristalsis

After each bolus has passed through into the proximal oesophagus, primary peristaltic waves are initiated by active closure of the cricopharynx.

Primary oesophageal peristalsis carries individual boluses to the cardia, but the process is not completely efficient and small quantities of ingesta are left at variable levels in both the cervical and thoracic oesophagus, even in normal horses. These ingesta are either picked up in the bolus of a subsequent primary wave or by locally generated secondary peristalsis.

1.2 DIAGNOSTIC APPROACH TO CASES OF DYSPHAGIA

History – signs of dysphagia

The signs of dysphagia include:

- an unwillingness to eat.
- slow, messy feeding.
- halitosis.
- rejection of semi-masticated food onto the ground (quidding).

Figure 1.1 Nasal reflux of saliva and ingesta in a case of acute oesophageal obstruction.

- productive coughing.
- nasal reflux of saliva, ingesta and fluids.

Obviously, horses that are unable to eat and swallow food are likely to lose weight rapidly, but this process is accelerated if the horse develops secondary inhalation pneumonia, which is a common sequel to dysphagia. A moist cough is typical of animals aspirating food or saliva into the rima glottidis. In addition to a clear case history, careful observation of the patient's attempts to eat and drink should be made.

If the horse shows return of ingesta from its mouth, the site of the lesion causing the dysfunction must lie in the oral cavity or oropharynx.

Nasal reflux of ingesta points to an abnormality of the pharyngeal or oesophageal phase of deglutition (Figure 1.1).

Physical examination, external and oral inspection

Evidence of systemic and/or toxic disease, including *Streptococcus equi* infection, botulism, grass sickness, rabies, upper motor neuron disease, lead poisoning and tick paralysis should be sought.

The external assessment should check for evidence of concurrent neuropathies such as facial palsy, Horner's syndrome or head tilt.

Thoracic auscultation should check for signs of inhalation pneumonia.

Local lymphadenopathies and firm distension of the oesophagus to the left side of the trachea are changes which might be found during palpation of the throat area.

Useful information can be obtained by attempting to pass a nasogastric tube. This should determine whether pharyngeal swallowing reflexes are still present or whether the upper alimentary tract is physically obstructed.

Under sedation and with a Hausmann gag in place, a detailed inspection of the oral cavity with the aid of a dental mirror should look for evidence of dental malalignment, enamel pointing of the cheek teeth, fractures of the dental crowns, periodontitis, soft-tissue lesions of the buccal cleft and palate, oral foreign bodies and lesions of the tongue. The structures involved may require digital manipulation to complete the examination, and a tell-tale foul smell points to the presence of stale entrapped ingesta.

Defects of the palate cannot always be appreciated from a conscious examination of the mouth because they are often restricted to the caudal section of the soft palate.

Endoscopy *per nasum*

The presence of ingesta in the nasal meati, nasopharynx, larynx or trachea is never normal and confirms the broad diagnosis of pharyngeal or oesophageal phase dysphagia. Such contamination may range from clods of green grass to tiny flecks of chewed ingesta. The latter may be appreciated only on examination of tracheal aspirate samples.

Endoscopy *per nasum* is necessary to confirm whether pharyngeal paralysis is present (Figure 1.2). The usual findings consist of:

- a mixture of saliva and ingesta on the walls of the nasopharynx.
- persistent dorsal displacement of the palatal arch.
- poor constrictor activity during deglutition.
- failure of dilation of one or both ATD ostia during swallowing.

Where functional pharyngeal paralysis is diagnosed, many horses are afflicted with pharyngeal hemiplegia, i.e. the pharyngeal neuropathy is unilateral, for example in cases of guttural pouch mycosis (see 'Aetiopathogenesis' in 5.6).

True pharyngeal paralysis may be seen in cases of botulism.

Conchal necrosis may accompany prolonged dental suppuration and may be seen on endoscopy of the nasal chambers (see 'Conchal necrosis and metaplasia' in 5.16).

Figure 1.2 Pharyngeal paralysis in a case of guttural pouch mycosis. Note the asymmetric larynx – the GPM is right-sided; the dorsally displaced soft palate; and the saliva and ingesta on the pharyngeal walls.

Figure 1.3 Midline cleft of the soft palate in a foal exhibiting the nasal reflux of milk. Most clefts are confined to the soft palate in horses, and the hard palate is rarely involved. Diagnosis is best confirmed by endoscopy *per nasum*.

Provided that an endoscope with a diameter of 8.0 mm or less is available, the diagnosis of a palatal defect by inspection of the floor of the nasopharynx *per nasum* presents no difficulties, even in quite young foals (Figure 1.3).

Other abnormalities which may cause dysphagia and which can be confirmed by endoscopy of the pharynx and larynx include:

- epiglottal entrapment, with or without a sub-epiglottic cyst (see 5.22 and 5.23).
- epiglottal hypoplasia.
- iatrogenic palatal defects after 'over-enthusiastic' staphylectomy.
- fourth branchial arch defects (4-BAD syndrome) (see 5.25).
- evidence of sub-epiglottic foreign bodies, usually in the form of unilateral oedema in the region of the ary-epiglottic folds.
- intra-palatal cysts (see 'Palatal defects' in 5.28).
- nasopharyngeal cicatrix.
- iatrogenic hyper-abduction of the arytenoid cartilage from prosthetic laryngoplasty or other evidence that 'tie-back' surgery has triggered dysphagia.
- arytenoid chondropathy (see 5.24).
- pharyngeal neoplasia (see Pharyngeal and laryngeal neoplasia in 5.28).
- pharyngeal distortion by external compressive lesions such as neoplasia or abscesses.

The extent of tracheal aspiration of ingesta which accompanies the dysphagia can be deduced by advancing the endoscope into the trachea to the level of the thoracic inlet. This is particularly helpful to assess compromised swallowing after 'tie-back' surgery.

Oesophagoscopy is often unrewarding in the investigation of equine dysphagia simply because physical or functional obstructions of the oesophagus invariably lead to a build-up of ingesta and saliva in the lumen which, in turn, inhibits a satisfactory field of view. However, when the patient has been starved prior to endoscopy, evidence of conditions such as oesophagitis, megaoesophagus, stricture, rupture, tracheo-oesophageal fistula, diverticulum, intra-mural cyst, dysautonomia and neoplasia may be found.

Radiography and fluoroscopy

Plain lateral radiographs of the pharynx, larynx and cervical oesophagus are used to investigate the relationships between normal anatomical structures and to identify intra-luminal, mural and extra-mural soft-tissue swellings. Contrast media can be helpful to outline these structures. Fluoroscopic studies – again, using contrast media – are required for the dynamic investigation of deglutition.

Lateral radiographs of the chest are a useful aid to monitor the progress of aspiration pneumonia which shows a characteristic pattern of consolidation in the dependent lung lobes.

Other imaging techniques

Ultrasonography always has a potential role in the investigation of the soft tissues of the throat but also has a specific value in the assessment of the temporo-mandibular joint, which is a difficult structure for radiography.

Scintigraphy has a specific role in the investigation of early cases of dental apical infections and can also confirm bony activity in the area of the TMJ.

The role of advanced techniques of MRI and CT in the investigation of dysphagia, and the advantages over traditional methods such as palpation and endoscopy, is not yet established.

Oral examination under general anaesthesia

General anaesthesia is necessary to complete the inspection of the oral cavity. The tendency for soft tissues to obscure the view, particularly towards the base of the tongue, can be overcome by the use of an endoscope passed through a polypropylene mare gynaecological speculum. Again, general anaesthesia is required for a more detailed manual or endoscopic examination of the caudal oral cavity, especially in the region of the epiglottis and ary-epiglottic folds.

1.3 AETIOLOGY OF DYSPHAGIA: ORAL PHASE ABNORMALITIES

Facial palsy and lip lesions

Facial paralysis inhibits the ability of a horse to prehend and retain ingesta in the oral cavity (see Chapter 11).

A laceration at the commissures of the lips can arise when a horse becomes hooked at the corner of the mouth and a major avulsion injury follows. Careful anatomical reconstruction of the lip margins is required.

Sarcoids may develop at the lip margins, especially the commissures, and the method of therapy (excision, cryosurgery or chemical cautery) which is used to ablate the lesions must also take regard of the acceptance of a bit after healing.

Temporo-mandibular joint and hyoid disorders

The temporo-mandibular joints (TMJs) lie very superficially immediately ventral to the zygomatic arch and are vulnerable to direct trauma, including penetrating wounds. However, TMJ disorders are rare in the horse but when they do occur they cause marked pain and a rapid loss of bodily condition. Disuse leads to obvious atrophy of the masticatory muscles, most obviously the masseters. Clinical examination shows resentment of attempts to open the mouth and even under general anaesthesia the range of opening may be severely reduced. A diagnosis of TMJ disease is difficult to confirm by radiography and other techniques such as ultra-sonography, scintigraphy, MRI or CT scanning, depending on availability, will provide superior images.

Ankylosis of the joint between the stylo-hyoid and petrous temporal bones is often a feature of temporo-hyoid osteoarthritis (THO) in horses and may limit a horse's ability to move the tongue (see 'Temporohyoid osteoarthropathy, THO' in 5.2)

Lingual abnormalities

The well protected location of the hypoglossal nerve in horses is such that injuries to the nerve with lingual paralysis are rare.

Trauma, either in the form of lacerated wounds or tongue-strap strictures, accounts for the majority of tongue lesions of the horse. A horse with a severely injured tongue may be unable to manoeuvre ingesta around the mouth and is inclined to drop food or to collect it in the buccal cleft.

Horses which lose the rostral portion of the tongue through incisive wounds can maintain normal bodily condition, albeit showing messy feeding patterns.

Foreign bodies may become buried in the lingual tissues, and the painful suppurative response can reduce a horse's inclination to eat.

Dental disorders

Dental disorders are discussed at length later in this chapter, but the reason why dental diseases cause dysphagia is through oral pain. There is little evidence that horses are afflicted with 'tooth-ache' per se, but soft-tissue pain through ulceration of the oral mucosa, particularly at the gingival margins in the form of periodontitis, is a major contributor to an inability to masticate food adequately and to reject partly masticated food to the ground – 'quidding'.

Congenital and acquired palatal defects

The presence of a defect in the soft palate prevents an effective seal between the oral cavity and the naso-pharynx during the lingual propulsion of ingesta towards the tongue base and during pharyngeal contraction. The result is that food and fluids are refluxed via the nasal chambers to the nares.

Simple midline linear defects of the soft palate are the most common cause of the nasal reflux of milk by foals in early life (Figure 1.3). Rarely, the midline cleft extends rostrally into the hard palate.

Other forms of palatal defect include unilateral hypoplasia and pseudo-uvula formation which can escape confirmation until the patient is considerably older. They often occur in association with epiglottal entrapment.

Palatal reconstructive surgery using mandibular symphysectomy has been described, but the results are invariably disappointing.

Excessive palatal resection (staphylectomy) (see 5.21) in the treatment of dorsal displacement of the soft palate (DDSP) is irreparable.

Inadvertent splits in the palate have been reported after the relief of epiglottal entrapment by section with a hooked bistoury passed *per nasum* in the standing horse (see 5.22).

Other oral conditions: foreign bodies, neoplasia

The most common foreign bodies in the oro-pharynx are brambles/thorned plant material which become lodged in the sub-epiglottal area or in the lateral food channels causing acute-onset dysphagia. Endoscopy *per nasum* will show oedema in the ary-epiglottic folds, even when the foreign body itself cannot be seen. Such an endoscopic finding is an indication for an oral examination, either by palpation or with a guarded endoscope, under general anaesthesia. Other foreign bodies such as wire fragments may become wedged between teeth.

Squamous cell carcinomas, lymphosarcoma and connective tissue tumours arise sporadically in horses, and they tend to cause dysphagia simply by virtue of space occupation.

1.4 AETIOLOGY OF DYSPHAGIA: PHARYNGEAL PHASE ABNORMALITIES

Pharyngeal paralysis

Paralysis or paresis of the pharyngeal constrictor muscles arises when the function of glosso-pharyngeal nerve (IX) is compromised. When food and fluids are not propelled into the upper oesophagus they may be returned via the nostrils, aspirated into the laryngeal airway, or spilled out of the mouth. The most common causes of pharyngeal paralysis are:

- guttural pouch mycosis (see 5.6) (Figure 1.2).
- ATD diverticulitis (see 5.5).
- botulism (see Chapter 11).
- heavy-metal poisoning (see Chapter 22).

It is always correct to investigate the possibility of ATD disease in cases of pharyngeal dysfunction. When there is marked inhalation of ingesta leading to broncho-pneumonia or evidence of dehydration, the condition of the patient demands euthanasia on humane grounds. However, some horses with partial pharyngeal dysfunction may survive without distress and simply show an occasional cough and nasal discharge without progress to aspiration pneumonia. Restoration of pharyngeal function may occur, but this takes many months.

Nasopharyngeal cicatrization inhibits the efficiency of pharyngeal constrictor function, but horses with this disorder are more likely to present for the investigation of respiratory noises and/or exercise intolerance.

Pharyngeal compression: strangles abscessation

The presence of extra-mural soft-tissue swellings adjacent to the pharynx may cause dysphagia because of external compression of the pharynx and also, in the case of an abscess because of the pain associated with the movement of food boluses past the lesions (see Chapter 19).

Pharyngeal cysts, palatal cysts

The origin of pharyngeal wall cysts is not known, but intra-palatal cysts may well develop from salivary tissue which is normally distributed widely in the palatal mucosa. Diagnosis of both conditions is by a combination of endoscopy and radiography. While cysts on the pharyngeal walls can be excised or ablated by trans-endoscopic laser application, lesions within the substance of the soft palate are not as easily treated because fistulation of the palate is a likely sequel to surgery.

Epiglottal lesions, including sub-epiglottic cysts

Peracute epiglottitis, with oedema and cellulitis may occur as a complication of upper respiratory tract viral infections and can be so severe that a potentially fatal airway obstruction occurs. Emergency tracheotomy intubation may be necessary to prevent asphyxiation.

A less severe form of epiglottitis with swelling and distortion of the epiglottis may have a similar aetiology and causes dysphagia and

Figure 1.4 Sub-epiglottal cyst in a foal. Note that in this case there is no associated entrapment of the epiglottis by the glosso-epiglottal mucosa.

coughing presumably through discomfort during deglutition. Afflicted horses also produce untoward respiratory sounds at exercise. The diagnosis is established by endoscopy, but it can be difficult to differentiate this form of epiglottitis, which is likely to be responsive to vigorous antibiotic therapy, from a para-epiglottic foreign body.

The increased mass of the epiglottis arising in entrapment by the glosso-epiglottal mucosa (see 5.22) or by a sub-epiglottic cyst (Figure 1.4) (see 5.23) causes dysphagia because of space-occupation and a restriction of the freedom for epiglottal retroversion. Secondary persistent dorsal displacement of the palatal arch may occur. Persistent DDSP is an indication for oral endoscopy and lateral radiography possibly using contrast medium. Both conditions are amenable to successful excisional surgery.

Laryngeal abnormalities

Compromised glottic protection leading to the aspiration of ingesta into the lower airways may arise spontaneously in cases of arytenoid chondropathy, or through iatrogenic causes such as complications of prosthetic laryngoplasty or partial arytenoidectomy (see 5.20). The precise cause of post-laryngoplasty dysphagia is not known, but over-abduction of the arytenoid cartilage, the physical presence of the implants themselves and nerve injuries are amongst the suggestions which have been proposed. Removal of the prosthesis is often, but not always, effective in control of the dysphagia, but, of course, the respiratory obstruction for which the surgery was originally performed can be expected to return as the arytenoid cartilage reverts to its collapsed state.

Fourth branchial arch defects (4-BAD)

Approximately two thoroughbreds per thousand born are afflicted with defects of the structures which derive from the fourth branchial arch, specifically the wings of the thyroid cartilage, the crico-thyroid articulation, the crico-thyroideus muscle and the crico- and thyro-pharyngeus muscles (see 5.25). The fourth branchial arch defect (4-BAD) syndrome may arise unilaterally or bilaterally, and any or all of the structures may show partial or complete aplasia. When the 4-BAD syndrome includes aplasia or hypoplasia of the crico- and thyro-pharyngeal muscles the proximal oesophageal sphincter remains permanently open. Horses afflicted with 4-BAD usually present with abnormal respiratory noises at exercise, and it is most unusual for them to show dysphagia unless they are badly afflicted or there is another concurrent defect. Horses with 4-BAD may show bizarre eructation-like noises at rest and may be confused with wind-suckers.

1.5 AETIOLOGY OF DYSPHAGIA: OESOPHAGEAL PHASE ABNORMALITIES

Megaoesophagus

Megaoesophagus has been reported sporadically in the horse, sometimes as a primary congenital disorder and sometimes secondary to other conditions causing restriction of oesophageal function such as vascular ring strictures. Coughing, nasal reflux of ingesta and distension of the cervical oesophagus may all be features. Confirmation of the diagnosis is easily achieved by contrast radiography.

Oesophageal obstruction ('choke')

Obstruction of the oesophagus is discussed in greater detail at the end of this chapter (see 1.7). Impaction of dry fibrous material to occlude the lumen of the oesophagus, typically in the cervical segment, is the commonest cause of acute dysphagia in the horse. Older horses seem to be more susceptible but this may relate to the diets offered to horses which are not being fed for competitive exercise. In contrast, foals which are beginning to take herbage occasionally plug the oesophagus with a bolus of dry grass.

Oesophageal strictures/stenosis

Oesophageal strictures are thought to be the sequel of episodes of acute obstruction, and horses with this condition are presented with recurring 'choke'. Confirmation of the diagnosis is best achieved by contrast radiography. The most common site for stricture development is at the thoracic inlet. Resection of the stenosed segment of oesophagus may be attempted, but a guarded prognosis is indicated because recurrence of the stricture at the site of anastomosis is a frequent complication.

Intra-mural oesophageal cysts

Congenital intra-mural cysts may be encountered in young horses and cause dysphagia through restricting peristalsis and obstruction to the passage of oesophageal boluses. The lesions may be seen as swellings in the oesophageal wall at endoscopy or by ultrasonography or contrast radiography.

Oesophageal rupture

Ruptures of the oesophagus may arise through external trauma by kicks or stake wounds or by misuse of a nasogastric tube. Regardless of the cause, the condition carries a poor prognosis unless affected horses are presented for treatment soon after the injury has occurred because of the rapid advance of contamination and cellulitis into the surrounding tissues.

Neoplasia

Tumours of the oesophagus are very rare in the horse, but squamous cell carcinoma at this site has been reported.

'Wind-sucking'

Horses performing this stereotypy do not swallow air, and deglutition is not part of the sequence of events. The muscles of the upper neck contract to create a pressure gradient across the soft tissues of the throat so that the walls of the pharynx and upper oesophagus are

pulled apart. The sudden in-rush of air through the open cricopharynx coincides with the gulping sound which is typically heard during attacks of wind-sucking, but the air is returned from the oesophagus to the pharynx immediately.

Grass sickness (See Chapter 3)

'Grass sickness' produces dysphagia in its acute form but colic in the sub-acute and chronic forms. The condition is seen in horses of all ages throughout the United Kingdom and northern Europe. Afflicted horses are generally severely depressed with patchy sweating, elevated pulse and ileus. The dysphagia arises as a part of total gastro-intestinal stasis, and nasal reflux of ingesta adds to the pitiful appearance of the patients. There is currently no reliable in vitro diagnostic test, but the radiographic demonstration of oesophageal stasis and the endoscopic identification of ulceration of the oesophageal mucosa are helpful pointers to the likely diagnosis.

1.6 ORAL TRAUMA, MANDIBULAR FRACTURES ETC.

Pathogenesis

Fractures of the teeth and jaws are not unusual and arise from falls, kicks by other horses and by bitting injuries and stick-and-ball trauma in polo. Fractures of the teeth themselves often lead to pulp exposure and devitalization and periapical suppuration. Restoration may be considered with early diagnosis but extraction becomes the preferred course for chronic cases.

Clinical signs

In many instances, particularly with horses at pasture, the traumatic incident is unwitnessed. Occasionally compound fractures of the mandible or maxilla are sustained with an obvious external wound. Horses with these injuries may present with sudden onset swellings of the facial and mandibular areas or inability to close the mouth with failure of effective prehension of ingesta and drooling of saliva. Neglected cases of oro-facial trauma may show external evidence of suppuration from devitalized tissue and weight loss from an inability to prehend and masticate food.

Diagnosis

Fractures of the mandibular rami should always be considered in cases of acute onset swelling of the jaw. External palpation, a judicious intra-oral examination and a careful radiographic investigation are used for damage assessment.

Treatment

Fortunately, many fractures of the mandible and maxilla show little displacement of the bone fragments, and surgical fixation is often not necessary. However, teeth may be devitalized either by infection through the fracture line or by disruption of their vascular supply. The decision whether or not to extract depends on radiographic findings, but such surgery should be delayed at least until a firm fibrous union of the fracture site is present. At this time sequestra may also be located and removed.

Open wounds over the sinuses may necessitate removal of loose bone fragments and debridement of gross contamination. An implanted irrigation catheter can be a useful measure to flush away blood and debris and should always be used when the sinus wall has sustained a full thickness penetration. The provision of broad-spectrum antibiotic cover during the immediate post-traumatic period should be routine.

Most palpable bone fragments, or those seen on radiographs, retain periosteal or endosteal attachments and heal uneventfully, but occasionally devitalization leads to sequestration with a discharge to the nose or skin surface. These should be dealt with by surgical removal as they arise.

Grossly deforming depression fractures should be treated by elevation as soon as possible after the trauma otherwise a fibrous union will form in a matter of days.

Molar and premolar involvement in upper or lower jaw fractures is best managed on a 'wait-and-see' basis because generally it will not be possible to determine whether or not a tooth is devitalized in the acute phase, and, in any event, the procedure of dental extraction will simply exacerbate the original trauma.

Fractures of the incisor quadrants can be repaired effectively by in-and-out wire fixation of the loosened teeth to secure neighbours and/or to the mandible or premaxilla. Alternatively, the canine teeth can be used to anchor the fixation wires.

Prognosis

In general terms the outlook for horses which sustain traumatic injuries to the jaws is favourable because there is often little displacement of bone in cases of fracture, and the devitalization of soft tissue and teeth is limited.

1.7 OESOPHAGEAL OBSTRUCTION

Aetiology

Acute obstruction of the oesophagus by impacted, dry ingesta ('choke') is typically associated with the ingestion of inadequately soaked sugar beet pulp in the United Kingdom. Equivalent offending possibilities occur in other parts of the world. The dry fibrous material swells with the absorption of saliva and an expanding bolus occludes the oesophageal lumen. Subsequent boluses compound the obstruction.

Clinical signs

Horses with 'choke' present in an acutely distressed state with copious reflux, particularly of saliva, to the nostrils and mouth (Figure 1.1).

Diagnosis

The initial diagnostic challenge in cases of oesophageal obstruction is to differentiate this condition from other causes of acute dysphagia most notably pharyngeal paralysis.

The cervical oesophagus may be palpably distended with firm ingesta, and the passage of a nasogastric tube beyond the pharynx is generally not possible.

Inhalation tracheitis is invariably present, and inhalation pneumonia may follow if treatment is not promptly instituted.

Endoscopy of the pharynx and ATDs may be helpful when the case history convincingly rules out access to the common causes of obstruction.

Treatment

Many 'chokes' can be relieved by heavy sedation, the repeated administration of spasmolytics and prophylactic use of broad spectrum antimicrobials. This treatment can be continued for several hours or even days, but the distressed state of the patient and the likelihood of serious respiratory complications demand that conservative management should not be prolonged.

If conservative therapy fails to relieve the obstruction after 24 hours, most clinicians advocate lavage under sedation or general anaesthesia.

The author's preference is to institute vigorous lavage, under anaesthesia, by stirrup pump through a nasogastric tube. Obviously the patient's trachea should be intubated, and the head is positioned over a supporting bag with the nose inclined downwards. In this way the impacted ingesta is progressively washed back out at the nostrils.

Other clinicians prefer to attempt relief using a similar lavage technique with the patient standing but sedated. It is important that the horse's head and neck are maintained in a lowered position during the procedure to prevent aspiration of food and lavage fluid. Excessive pressure on the oesophageal lavage must be avoided.

Prognosis

The prognosis for a complete recovery after the relief of an oesophageal obstruction is good. The possibility of recurrence of the 'choke' or of long-term stricture development can be reduced by withholding dry fibrous foods for at least 72 hours as oesophageal motility is likely to remain weak for several days. Instead, wet 'sloppy' foods, mashes and fresh grass should be fed. Oesophageal motility is likely to remain weak for several days. The inhalational tracheitis or bronchitis is usually self-limiting, but broad-spectrum antibiotic cover should be maintained for 7–10 days.

1.8 ANATOMY OF THE ORAL CAVITY

Oral cavity

The oral cavity of the horse forms the first part of the alimentary tract. The horse is an obligate nasal breather so the oral cavity takes no part in respiration in contrast to many other species. The role of the oral cavity is to prehend food, particularly forage, and processes this into a form that allows for efficient digestion in the hind gut. This is different to the digestive process in ruminants, which allows for repeated cycles of mastication and digestion via regurgitation of partially digested boluses of food in a process known as rumination. In horses, the processing of food occurs in one attempt and has resulted in development of a specialized hypsodont dental anatomy. This feature of equine dental anatomy and function should always be borne in mind when carrying out corrective dental procedures that may adversely affect the masticatory function of the oral cavity as they may, indirectly, have a significant effect on the function of the entire digestive tract. The teeth are comprised of three main types of hard dental tissue, cementum, enamel and dentine (divided into primary and secondary type) which are exposed at the occlusal surface with wear (Figure 1.5a, b & c).

Normal dental anatomy

The dentition of the adult horse comprises six upper and lower incisors set within the premaxilla and mandible respectively. The teeth have a hypsodont conformation, divided into three regions, the visible clinical crown, a sub-gingival reserve crown and an apical region. These teeth function as a unit whose primary role is prehension of food. The anatomy of the incisors, and its change with age, has historically been used as a guide to ageing the horses. The enamel infolds on the erupting crown creating an infundibulum which is filled with cementum. As the teeth come into wear this becomes isolated, forming the 'dental cup'. The teeth at this stage are said to have an oval shape to the occlusal surface. As wear continues the pulp cavity lays down a protective tissue known as secondary dentine. This is more porous than the surrounding primary dentine and as it is exposed at the occlusal surface it becomes stained by oxidized plant material creating the 'dental star' which lies labially to the infundibulum. Continued attrition at the occlusal surface reaches the base of the infundibulum made solely of enamel and is termed the 'enamel mark'. Eventually the enamel is worn away completely and the dental star takes a more central position within the occlusal surface which now has a more triangular appearance.

The adult incisors are all preceded by deciduous dentition, which is lost at approximately 2.5 years for the central incisors, 3.5 years for the middle incisors and 4.5 years for the corner incisors. The deciduous incisors are smaller in appearance to their permanent counterparts and viewed from rostrally have a semicircular shape in contrast to the more rhomboid or oblong shape of the permanent dentition. At birth the central incisors are erupting, and usually present within the first week of life. The deciduous middle incisors erupt at approximately 6 weeks of age, with the corner incisors appearing around 6 months of age.

The canine teeth, sometimes referred to as 'tushes', are most commonly seen in stallions or geldings. The upper and lower canine teeth erupt from the premaxilla and mandible respectively. They may be present in mares as well, although are often vestigial, unerupted and/or variable in number. The canine teeth are said to erupt around five to six years of age, although this can be extremely variable.

The incisors and canines are separated from the remaining dental arcades by the 'interdental space'. This is often misnamed as the 'diastema' – a term which refers to a pathological condition often responsible for periodontal disease. In some horses, wolf teeth are found within the interdental space. These vestigial first premolar teeth are found most commonly in the maxillary arcades, lying rostral to the first cheek teeth. These teeth are commonly blamed for riding or bitting problems; however, in many cases it is hard to attribute the clinical signs shown to their presence. More commonly other pathological conditions of the oral cavity can explain the clinical signs shown and, therefore, removal of the wolf teeth, which lie just lateral to the palatine artery, is not always indicated.

The cheek teeth lie within the caudal oral cavity, comprising two upper and two lower batteries of teeth. The cheek teeth can be subdivided into premolars, the first three teeth in each battery, and molars, the last three teeth in each battery. The premolars of the horse are unique in that they are 'molarized' and as such are morphologically identical to the molar teeth. The teeth have a hypsodont conformation, divided into three regions, the visible clinical crown, a subgingival reserve crown and an apical region. As these teeth erupt they are angled in such a way as to move towards each other creating a tight junction between adjacent teeth, in a process known as 'mesial drift'. The premolar teeth each have a deciduous precursor which erupt in the first 7 days of life and are shed at approximately 2½ years, 3 years and 4 years for the 2nd, 3rd and 4th premolars respectively. The molar teeth have no deciduous precursors, and erupt at 1 year, 2 years and 3½ years for the 1st, 2nd and 3rd molars. The eruption of the molars corresponds with lengthening of the head as the horse grows.

The cheek teeth are continually wearing down due the presence of abrasive silicates within the diet. The relative hardness of the different dental tissues results in a naturally roughened surface which is important in mastication. The enamel, though brittle, is slightly harder than the surrounding cementum and dentine resulting in small ridges over the surface of the tooth. The mandibular teeth are oblong in shape and have a single enamel ridge with marked folding creating a millstone-like surface. The maxillary are square in shape and have two infundibulae creating an enamel ridge around the periphery of the tooth and two small enamel rings in the centre of the tooth. The centre of these two smaller enamel rings contains cementum which is often hypoplastic, and may predispose to caries (see dental caries later).

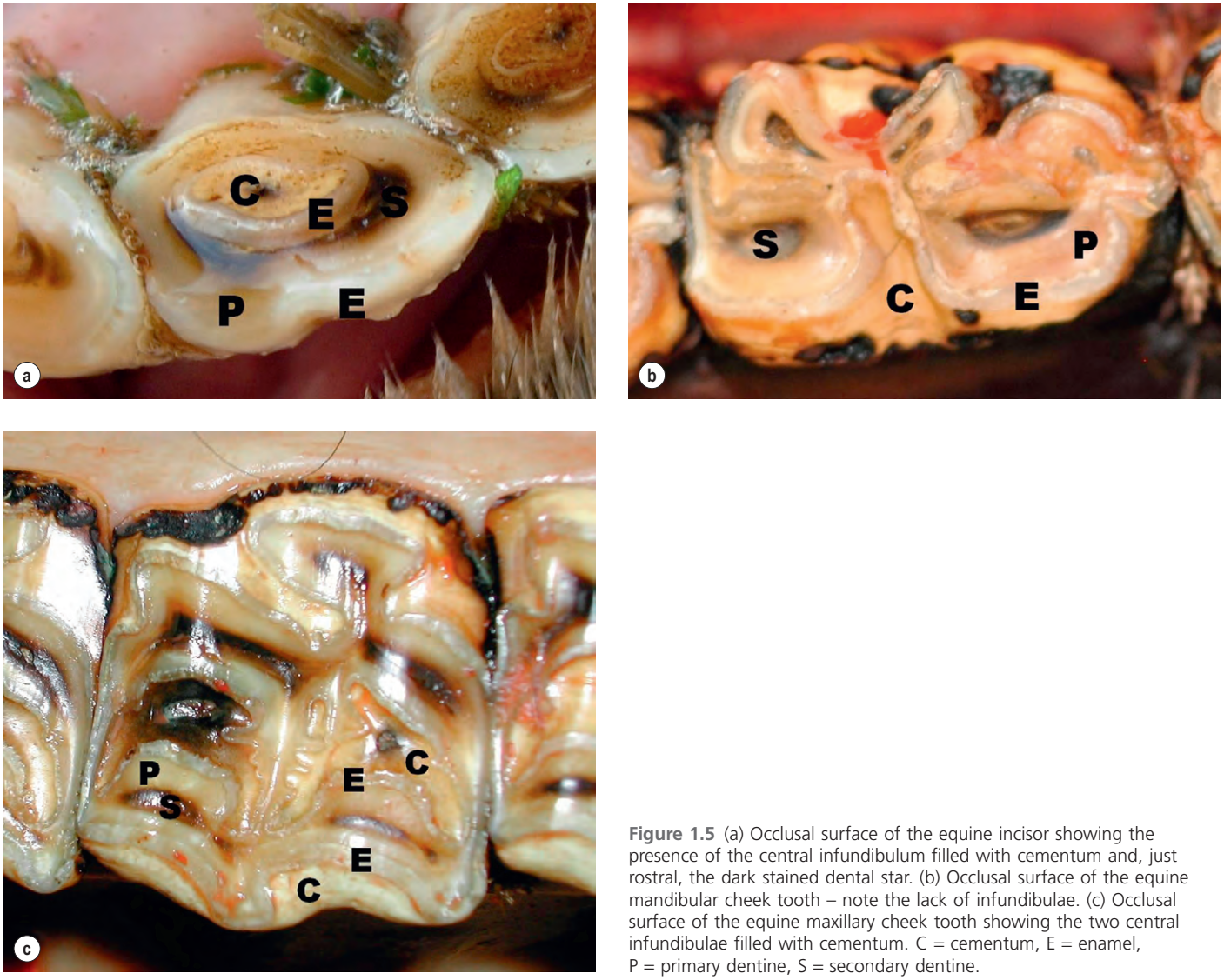


Figure 1.5 (a) Occlusal surface of the equine incisor showing the presence of the central infundibulum filled with cementum and, just rostral, the dark stained dental star. (b) Occlusal surface of the equine mandibular cheek tooth – note the lack of infundibulae. (c) Occlusal surface of the equine maxillary cheek tooth showing the two central infundibulae filled with cementum. C = cementum, E = enamel, P = primary dentine, S = secondary dentine.

The overall contour of the occlusal surface of each tooth is not flat but contains a pair of transverse ridges which interdigitate with the opposing arcade to increase the surface area of the arcades. The height of these ridges varies between individual horses. Individually enlarged ridges, more appropriately termed transverse overgrowths, may arise opposing diastema (see 1.10) and should be reduced to prevent further food impaction between the opposing teeth.

The pulp is the central vascular tissue surrounded by the hard tissues of the tooth. The pulp is responsible for the innervation of the tooth as well as for the production of dentine. The odontoblast cells form the outermost layer of the pulp and are intrinsically linked to the dentine by means of odontoblast processes. It is believed that wear on the occlusal surface stimulates the odontoblasts via their processes which are responsible for the production of secondary dentine. The pulp anatomy of the incisors and canines are relatively similar with a single common pulp chamber. The pulp chamber is more complex in the cheek teeth with a common pulp chamber at the apex of the tooth in the young horse and five pulp horns separated by primary dentine extending towards the occlusal surface. In the most rostral cheek tooth in each arcade an extra pulp horn is present on the rostral margin, and in the most caudal cheek tooth in each arcade an extra one or two pulp horns are present at the caudal aspect. Awareness of the location of these is essential when performing reductions of overgrowths and also in identifying pathology affecting the pulp canal. The occlusal extremity of each pulp horn can be recognized as a dark stained area due to the porous nature of secondary dentine which uptakes food particles. These subsequently oxidize, creating a brown stain on the tooth surface.

Triadan system

In equine dentistry it is essential that all practitioners use the same terminology when discussing diseases affecting the teeth. The length of name required to identify an individual tooth, as well as the confusion that arises from the cheek teeth being made up of both premolars and molars, has led to the establishment of a system of nomenclature adapted from human and small-animal dentistry. This is termed the

modified Triadan system and has become the standard used by practitioners working in the field of equine dentistry. The numbering system assigns a three-digit code which can be used to identify any normal tooth found in the equine mouth, and comprises two parts. The first digit denotes the arcade number, and the next two digits the tooth number.

When viewed from rostrally, each quadrant in the adult horse is assigned a number, starting at 1 for the upper right moving clockwise assigning the next number to each arcade in turn. Thus, quadrant 2 is the upper left, quadrant 3 the lower left and quadrant 4 the lower right. This is used for adult permanent dentition. For the deciduous dentition, the quadrant numbering starts at 5 for the upper right and moves round sequentially as for the permanent dentition so that quadrant 8 would be the lower right deciduous quadrant.

The teeth are then numbered sequentially starting from the central incisor, which is assigned 01, moving distally assigning a new number for each tooth that might be present. Thus, the incisors are 01 to 03; the canines are 04; the wolf teeth 05; the premolars 06 to 08 and the molars 09 to 11. This can be seen on the dental chart (Figure 1.6). It should be noted that extra numbers are not assigned for supernumary teeth, the extra tooth is merely noted as a supernumary and assigned the number of the adjacent normal tooth, e.g. a supernumary tooth at the caudal aspect of the upper right maxillary arcade would be a supernumary 111, not a 112. Likewise, numbers are not removed for absent teeth, e.g. a mare with no canines or wolf teeth will not start its cheek teeth arcades with 04s.

Eruption of teeth

The teeth of equidae are unique in their hypsodont from, which has arisen due to their diet. The abrasive nature of their diet results in wear on the occlusal surface. In order to compensate for this wear the teeth continually erupt. The rate of eruption varies with age; in younger individuals it is more rapid – it may be up to 10 mm per year. As the horse gets older the eruption rate slows down; a horse over 20 may erupt their teeth at only 1 mm per year. The eruption rate, in balance with the rate of wear, ultimately determines how quickly wear disorders will arise when mouths are left untreated, and also how quickly occlusion will be re-established following corrective procedures.

1.9 ABNOMALITIES OF WEAR – ABRASION AND ATTRITION

Abnormalities of wear are the most common dental pathology encountered by the equine veterinary surgeon. They include minor sharp points through to large unopposed overgrowths.

Pathogenesis

Wear disorders include problems of both insufficient and excessive attrition of the teeth. The most simple wear disorders are the production of sharp buccal points on the maxillary cheek teeth and sharp lingual points on the mandibular cheek teeth. They arise due to lack of attrition on these edges when horses feed on diets which are more easily masticated. The maxillary arcades are set wider apart than the mandibular arcades, and the reduced lateral movement associated with most domestic horses' diets results in a lack of wear on the aforementioned edges. These sharp points can lead to abrasion and ulceration of the cheeks and tongue. This causes discomfort during eating which results in further reduction of the lateral movement of the jaw.

Most wear disorders of concern arise from a lack of attrition of the occlusal surface. The lack of wear combined with the continued eruption of the cheek teeth results in formation of an overgrowth. The reasons for overgrowth should always be ascertained. The overgrown tooth is seldom abnormal and is merely a normal erupting tooth that isn't being worn down. Evaluation of the arcade opposing the overgrowth will often reveal the underlying pathology. Common causes include: missing teeth, malerupted teeth, fractured teeth, overall disparity in the apposition of the arcades, e.g. parrot mouth and diastemata. The shape and size of the overgrowth will be dictated by the nature of the abnormal wear. Large overgrowths if left untreated can result in soft-tissue trauma, tooth displacements and diastemata.

Wear disorders may also arise due to lack of normal lateral movement of the mandible during mastication. The most common causes are painful conditions of the oral cavity, such as diastemata, which result in a reduction in the lateral excursion of the opening phase of mastication. This, in turn, results in a lack of wear on the buccal aspect of the maxillary arcades and lingual aspect of the mandibular arcades. In time, the occlusal angle of the entire upper and lower arcades of teeth on the affected side steepens, a condition known as 'shear mouth'.

Clinical signs

Clinical signs vary considerably between cases. Many cases are clinically silent. Eating problems, such as spilling hard feed, are often unnoticed by the owner. Large overgrowths resulting in significant soft-tissue trauma may cause quidding of forage. Riding problems may also be noticed, particularly in horses expected to work in an outline.

In cases with shear mouth, the problems of mastication giving rise to the wear disorder may be apparent in the form of quidding. In cases where quidding is not seen, careful observation may reveal mastication occurring only on the unaffected side.

Diagnosis

Diagnosis relies on a thorough clinical examination. Examination of the oral cavity by palpation and visual inspection and a good awareness of normal anatomy should quickly identify overgrowths. Care should be taken to avoid misinterpreting normal anatomical features such as transverse ridging and ventral curvature of the mandible ('curve of spee') as overgrowths requiring correction. Identification of shear mouth relies on awareness of the normal angle of occlusion, which varies between individuals and throughout each arcade. Recognition of the primary pathology which has resulted in the overgrowth or shear mouth should also be completed.

Treatment and prevention

Treatment of sharp points consists of rounding the buccal aspect of the maxillary arcades and lingual aspect of the mandibular arcades. This can be accomplished using a range of hand rasps or motorized instruments. Care should be taken not to remove excessive width from the teeth being rasped by ensuring that the instruments are kept between 45 and 60 degrees and only the sharp points are removed and rounded.

Overgrowths of the occlusal surface require careful assessment. Reduction is often most easily accomplished using motorized instruments. These allow precise reduction of overgrowths on the tooth surface that would be impossible with hand instruments. It should be remembered that teeth are living anatomical structures. A large proportion of the occlusal surface is made of dentine (both primary and secondary) which is intimately connected to the pulp and, therefore, should be considered as a living tissue. Motorized instruments, if used, should feature continuous water cooling to prevent thermal trauma to the pulpal tissues. Reduction should be performed visually using a headlight to illuminate the mouth, with particular attention paid to the dark stained secondary dentine on the occlusal surface.

Equine Dental Chart

Clinician:_____

Owner/Trainer:_____ Date:_____

Address:_____ Horse:_____

_____ Breed:_____

_____ Age:_____ Sex:_____

_____ Location:_____

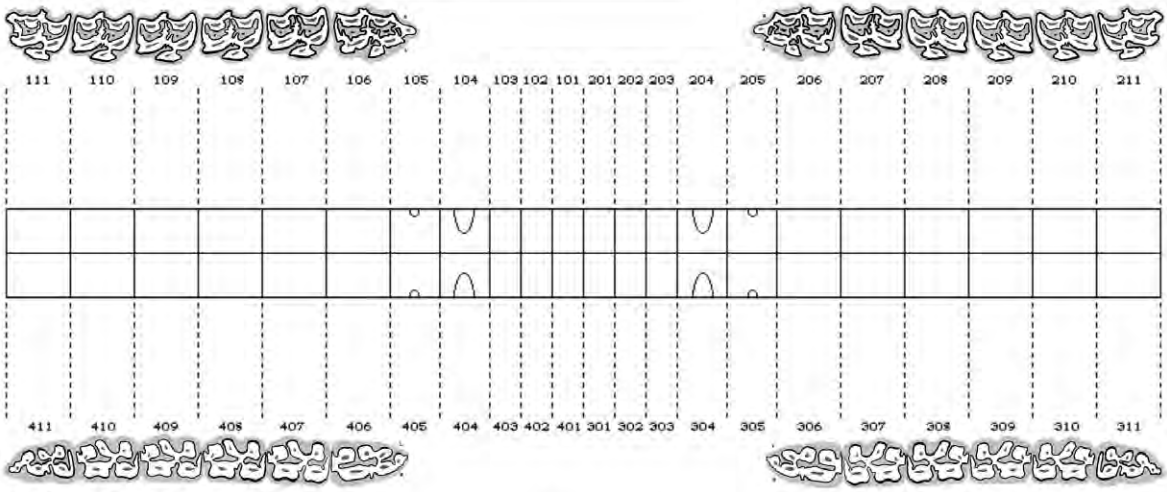
Tel._____ _____

Referring Vet/EDT:_____

Problems:_____

Tetanus status:_____ Sedation:_____

Lateral Excursion: Before_____ After_____



Soft Tissues

Arcade#1_____

_____

Arcade#2_____

_____

Arcade#3_____

_____

Arcade#4_____

_____

Key:

SP(B/L) - Sharp points (buc./ling.)
CH - Caudal hook (S/M/L)
RH - Rostral hook
St - Step
Wa - Wave
WC - Worn crown
TC - Tall crown
ROT(B/L) - Rotated (buc./ling)
 # - Fracture
O - Missing tooth
P/E - Partially erupted
U/E - Unerupted
CAL - Calculus
X - Extracted
DT/r - Deciduous tooth/ root sliver
SN - Supernumary tooth
BU - Buccal ulceration
LU - Lingual ulceration
PD - Periodontal pocket
Dias - Diastema

TMJ:_____ Occlusal Angle: Left_____ Right_____

Body Condition: OW VG G AV P VP

Further notes:

_____

_____

_____

_____

_____

_____

_____

_____

_____

Please make your next appointment in ____ months for a dental checkup.

Signed:_____

Figure 1.6 Dental chart using the Triadan system, *with kind permission of the Bell Equine Clinic.*

This thin layer of porous dentine lies over the pulp horns of the tooth, and as the overgrowth is reduced the protection of the pulp is removed. Monitoring the change in colour of the secondary dentine, as the superficial stained layer is removed, gives a guide to the location of the pulp horns, and once the stained layer is completely removed, further reduction should be avoided. When reducing an overgrowth it should be done in a manner to maintain the normal lateromedial angulation of the occlusal arcades.

In cases with shear mouth, correction can present a significant challenge. Initially, treatment of the underlying pathology is indicated. In mild cases this may be sufficient for normal mastication to resume which, in turn, will result in more normal wear forces. In more severe cases it should be remembered that the pathology has taken many months, and in most cases years, to arise. As such the chewing motion and musculature is adapted to the abnormal masticatory action, so corrections should be done gradually. Correction should begin by addressing the normal sharp buccal and lingual points, followed by reduction of the highest side of the arcade being treated. Attempts to re-establish normal occlusal angle starting from the lower side of the tooth should be avoided as this area is excessively worn, and would result in excessive removal of clinical crown.

Prognosis

The prognosis for most overgrowth corrections is good. Excessive removal of occlusal surface or heating of the tooth structure using uncooled instruments can result in post-procedure quidding, either through dentine sensitivity, lack of differential wear or lack of occlusion between opposing arcades. These can take many months or years to resolve, and in geriatric patients may never fully recover so care should be taken to identify these risks and ensure procedures are done in an appropriate manner.

1.10 PERIODONTAL DISEASE

Pathogenesis

Periodontal disease is defined as pathology affecting the periodontal ligament and associated structures. In comparison to man, plaque retention is not believed to be as important an initiating factor as food stasis. Most periodontal disease arises as the result of food stasis in the region of the interproximal spaces between cheek teeth or incisors, and is often associated with pathological spaces occurring between the teeth, known as diastema. Food accumulation in the spaces results in a localized periodontal disease (Figure 1.7). Localized periodontal disease may arise with lower canine teeth in association with tartar accumulation.

Clinical signs

In many instances mild periodontal disease appears clinically silent, with localized diastemata not being immediately apparent without detailed oral examination under sedation, aided by use of a dental mirror and/or dental endoscope. In more severe cases, the predominant sign is oral phase dysphagia, particularly when masticating long-stem forage ('quidding'). This can be quite dramatic with large piles of partially masticated forage building up on the floor of the stable.

Diagnosis

Clinical cases presenting with signs of quidding forage should be sedated and receive a detailed oral examination using a bright headlight and dental mirror or an oral endoscope to establish a correct diagnosis. Diastemata between the caudal mandibular cheek teeth are easily missed on a cursory dental examination, and frequently in these cases quidding forage is incorrectly attributed to sharp enamel points, a rare cause of dysphagia of forage. The relationship of diastemata to dental overgrowths and displacements should not be overlooked when examining the oral cavity. Caudal overgrowths on the last mandibular cheek teeth are commonly associated with diastemata due to caudal displacement of the overgrown teeth, widening of the interproximal space and impaction of food compounded by development of opposing transverse overgrowths. As such, the primary pathology, as well as the secondary, needs to be addressed.

Figure 1.7 Diastemata with localized periodontal disease between mandibular cheek teeth.

Treatment

Following detailed examination and accurate diagnosis, the food impacted around the interproximal space needs to be removed. This is usually accomplished by careful use of a combination of fine dental picks and lavage using low pressure water picks, or three-way syringes found on some dental machines. Care should be taken to avoid causing more damage to the periodontium. High-pressure, air abrasion units, in particular, must be used with great care to avoid forcing food material deeper into the periodontal tissues.

Once the impacted food has been removed, further assessment of the interproximal space is required. The depth of periodontal pockets should be assessed using fine periodontal probes, again ensuring care is taken not to cause further damage to the periodontium. The degree of gingival recession should also be noted which will reduce the measured depth of periodontal pockets. Radiography, using either intraoral or open-mouth oblique views, should be used to assess the loss of interproximal bone and also to assess the interproximal conformation. The periodontal pocket should be cleaned to remove any necrotic tissue and the interproximal tooth surfaces debrided using hand scalers and/or high-speed burrs where necessary.

In some cases, removal of the impacted feed and necrotic tissue from the periodontal pocket and correction of the related dental malocclusions are sufficient alone to resolve the clinical condition.

If the interproximal conformation, assessed using clinical examination and radiography, is such that the space between adjacent teeth is narrower at the occlusal surface than at the gingival margin a diagnosis of 'valve diastema' can be made. In these cases widening of the interproximal space at the occlusal surface using a fine right-angled burr may be indicated. The aim is to prevent food trapping between the

teeth; however, care should be taken to prevent inadvertent pulp exposure by excessive removal of dental tissue and thermal trauma by using a continuously irrigated instrument. In some cases, especially in the caudal oral cavity, it can be very difficult to ensure that the burr is accurately located within the interproximal space; the use of instruments with an adjustable head angle and assessment of the instrument placement with a dental mirror or endoscope are indicated.

Once the periodontal pocket has been cleaned and any interproximal widening required has been completed, then periodontal pocket can be protected using polyvinylsiloxane impression material dispensed using an automix syringe over the top of the pocket. Some practitioners advocate the use of calcium sulphate ('Plaster of Paris') within the base of the pocket to act as an osteoconductive material to promote regeneration of interproximal bone and/or antibiotic gel preparations as an aid to treat the bacterial infection associated with periodontal disease.

Prognosis

The prognosis for treatment of periodontal disease varies depending on the extent of the disease at time of treatment. Localized low-grade periodontal disease can often be effectively treated with conservative management and routine rasping and carries a good prognosis. Extensive periodontal disease with concurrent loss of bone in multiple locations can prove very frustrating to treat and carries a guarded to poor prognosis.

1.11 DENTAL CARIES

Pathogenesis

Dental caries occurs as a result of acid erosion of the calcified dental tissues of the teeth. In the horse, this most commonly involves the cementum, either on the periphery of the tooth, so called peripheral caries, or within the infundibulae of the maxillary cheek teeth, termed infundibular caries. The exact pathogenesis is at present undetermined; however, it is believed to be similar to that found in other species, where oral bacteria metabolize simple carbohydrates in food to form acid which dissolves the inorganic salts from the cementum.

It has been suggested that hypoplasia of the cementum found within the infundibulae of maxillary cheek teeth, a common finding in anatomical studies, may predispose to food accumulation within the cavity and thereby allow the disease process to begin. The reasons for cemental hypoplasia are not clear. It has been proposed that early removal of deciduous caps may prematurely disrupt the blood supply to the infundibular cementum thereby causing cessation of cementum production, this doesn't explain why the most commonly affected teeth are the first maxillary molars, which have no deciduous precursors.

The pathogenesis of peripheral caries is also poorly understood. Low-grade pitting is commonly found; however, more extensive disease resulting in almost complete loss of peripheral cementum, predominantly affecting the mandibular molars, in some individuals often has no clear cause. Undoubtedly the make up of an individual's own dentition will have some bearing, but factors such as dietary carbohydrates and acids may also play an important role. The authors have observed cases where the excessive feeding of sugary sweets to horses on box rest or feeding a haylage has resulting in rapid onset of peripheral caries which has responded to removal of treats or alteration of the forage diet and resulting in cessation of the disease process; however in many cases there is no clear cause that can be identified. The peripheral cementum in the horse is a dynamic tissue integral to the periodontium. A dramatic increase in the thickness of cementum occurs at the alveolar crest and providing the ongoing disease process can be stopped it may be possible for the diseased peripheral cementum to be replaced as the tooth erupts. Peripheral cemental caries may also be linked to periodontal disease, although at present it is unclear which disease is the initiating problem.

Clinical signs

Clinical signs vary between cases but often cases of dental caries are clinically silent. In some cases extensive peripheral caries on the caudal mandibular arcades can result in lingual ulceration which can cause hypersalivation or dysphagia although this is rare. Infundibular caries is almost always clinically silent until the point of fracture at which point sudden onset dysphagia can occur.

Diagnosis

Accurate diagnosis relies upon a detailed oral examination. Food must be washed from the mouth using copious water or disinfectant solution as a clear view of the tooth surfaces is required. The use of a dental mirror and bright headlamp is essential to recognize lesions within the caudal oral cavity, and in some instances an oral endoscope may be preferable. The diagnosis of dental caries is not solely made on the appearance of discoloration, although this is a helpful guide. The use of a dental explorer to assess the relative hardness of the tooth surfaces also aids diagnosis with active caries lesions being softened by the disease process. The extent of caries lesions should also be assessed. This is not always immediately apparent, especially in cases with infundibular disease. Radiographs, and, if available, computed tomography, may help in ascertaining the extent of involvement and depth of lesions. In cases where there is extension into the pulp canal the lesion is likely to result in apical infection and, as such, extraction of the tooth is likely to be required. Care should be taken to avoid confusion in young horses with recently erupted permanent dentition where the infundibulum has a very open shape, frequently confused as dental caries. Also, it is imperative that the practitioner understands the occlusal surface anatomy in detail to avoid confusion between infundibular caries (which initially involves cementum only) and secondary dentinal defects which can be the consequence of endodontic disease.

Treatment

A number of practitioners have tried to treat dental caries using restorative dentistry techniques adapted from human dentistry. It is essential in cases where this treatment is being considered that the extent of the caries process is accurately known. The nature of many infundibular lesions is such that the disease process may not be completely apparent from the occlusal surface. This, in combination with the curved anatomy of the infundibulae and the depth from the occlusal surface of the active lesion in many cases, makes complete restoration of the lesion impossible.

In those cases where restoration is considered suitable it is important that owners are made aware that cases will require careful monitoring. The initial process consists of debridement of the carious dental tissue using a combination of high- and low-speed dental handpieces and, in some cases, high-pressure air abrasion using aluminium oxide powder. It should be remembered that the infundibulum is a central structure within the tooth and is surrounded by multiple pulp horns; therefore, debridement should be done carefully to avoid excessive removal of dental tissue and inadvertent pulp exposure. Following debridement, the cavity is lavaged and dried using a three-way syringe and then etched using phosphoric acid gel for 10–20 seconds. This dissolves the inorganic calcium salts from the superficial layers of the cavity. The acid gel is removed by lavage and then air-dried briefly to remove the majority of the moisture. A bonding agent is then applied to the walls of the cavity and thinned using air

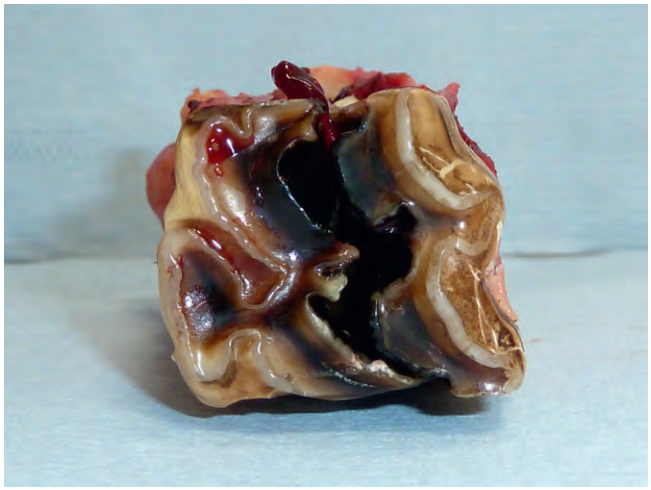


Figure 1.8 An extracted maxillary cheek tooth showing a sagittal fracture due to coalescing infundibular caries.

followed by curing using a high-intensity blue light from a specially designed curing light. It should be remembered that infundibular lesions primarily involve cementum, which is different to human dentition, where caries involves enamel and dentine, which means that the bonding of dental materials is unproven in the horse. For this reason the use of combined etch and bonding materials has not been recommended. Following bonding, the cavity is then filled using restorative materials in thin layers applied to the individual walls of the cavity to build up the restoration. Following restoration, cases require careful monitoring. Once a restoration is in place, it can be difficult to assess whether caries is ongoing underneath the restoration. For this reason, follow-up examinations and radiography every 6 months are essential to prevent subsequent endodontic involvement.

In those cases where the caries process has progressed to the point of fracture (Figure 1.8) the only viable treatment option is extraction of the affected tooth.

Prognosis

The prognosis for horses with extensive caries lesions involving both infundibulae is guarded, with many cases resulting in sagittal fracture of the tooth. These cases require extraction of the affected tooth. The prognosis for horses with peripheral caries, for horses with mild to moderate infundibular caries, and for horses with caries where restoration has been attempted remains undetermined.

1.12 ENDODONTIC DISEASE INCLUDING DENTAL ABSCESSATION

Pathogenesis

Endodontic disease is defined as disease affecting the pulp and periapical tissues of the tooth. Once within the pulpal tissues, bacteria cause infection and ultimately necrosis of pulp. The pathogenesis varies between cases but includes extension of periodontal disease around the tooth to allow bacteria to affect the apical region, extension of dental caries from the cementum, through the enamel and dentine into the pulp canal, dental fracture and, less commonly, maxillary or mandibular fracture. In many cases the exact cause is unclear, and in these cases, anaechoresis associated with dental eruption is a possible pathogenesis.

Clinical signs

Dental pain is rarely seen in cases of endodontic disease in horses. Occasionally the owner may notice a brief period of dysphagia associated with initial onset of the disease, but in the absence of other clinical signs, this is rarely investigated if it resolves in a couple of days.

Cheek teeth are most commonly affected by endodontic disease. Clinical signs noted are mandibular swelling with the lower cheek teeth, maxillary swelling with the rostral upper cheek teeth, and unilateral purulent nasal discharge with caudal upper cheek teeth.

Diagnosis

Early diagnosis of endodontic disease is rare in the horse.

Detailed oral examination using a bright headlamp, dental mirror and dental explorer may find defects in the secondary dentine of the affected tooth. These arise due to the continual eruption of the teeth and attrition of the occlusal surface. In normal teeth the secondary dentine is laid down continuously by the odontoblasts within the pulp; however, with pulpitis the odontoblasts are impaired, so that production of secondary dentine is insufficient to compensate for occlusal surface wear.

Radiography is the most commonly used diagnostic imaging modality in equine dentistry. Changes associated with endodontic disease include disruption of the lamina dura denta, periapical sclerosis and blunting of the dental apices. Where a draining tract is present it may be possible to gain further information by placing a probe within the tract during radiography. In many cases though, dental radiographs are hard to interpret, making early diagnosis difficult. It may be necessary to repeat radiographs over several weeks to assess changes around teeth and avoid misdiagnosis.

Computed tomography allows more detailed imaging of the teeth and associated structures, without the problems associated with superimposition. Until recently this required general anaesthesia of the patient; recently developed techniques now allow CT to to be used to image the head of horses with the horse sedated and standing.

Treatment

Treatment of teeth with endodontic disease primarily consists of extraction of the affected tooth. The complex nature of the pulp canals of equine cheek teeth make endodontic therapy difficult, if not impossible. There have been many studies looking into the success rates of total root canal therapy in the horse, using a variety of different materials and techniques. Short-term success is often good, around 70%; however, the outcome in the medium to long term is usually poor, requiring extraction of the affected tooth. Recent advances in our understanding of the unique anatomy and physiology of equine cheek teeth have revealed why failure is so common. The apical and coronal seal is a common source of technique failure in brachydont teeth and with the continual wear and complex anatomy of the pulp canals in equine cheek teeth breaching of this seal is likely to occur in most cases.

Early diagnosis of tooth fracture or recognition of iatrogenic pulp exposure, may allow the use of pulp capping techniques, with or without partial pulpotomy to preserve the tooth; however, these techniques have yet to be evaluated with research to be scientifically evaluated.

Prognosis

The prognosis for individually affected teeth is poor as most will require extraction. The likelihood of complications occurring after extractions is very technique-dependent; however, the long-term outlook is good following tooth extraction.

1.13 TUMOURS OF THE UPPER ALIMENTARY TRACT

Odontogenic tumours

Pathogenesis

Neoplasia may arise from any of the dental tissues, including the cementum and periodontium, although odontomas and ameloblastomas, derived from dentine and enamel respectively, are more common. In general terms the greater the level of differentiation of the tumour tissues the more identifiable mineralized dental material will be present and the better the prospects of successful surgical extirpation. In contrast, poorly differentiated odontogenic tumours have no mineralized elements and are extremely aggressive.

Clinical signs

Although these tumours are rare in horses, the possibility of an odontogenic tumour should be considered whenever an animal develops a localized swelling of either jaw, and particular suspicion should be raised when the patient is young, coinciding with the time of greatest activity by the primordial dental tissues. However, ameloblastomas are more prevalent in older horses.

Diagnosis

A definitive diagnosis of odontogenic neoplasia can be made only after biopsy of the lesion, but histological interpretation is both difficult and confusing. In addition, the result of the biopsy investigation may not be known until long after extirpation surgery has been performed because samples must undergo demineralization before they can be fixed. Thus, the major diagnostic challenge in the case of a horse presented with a swelling of either jaw is to differentiate between dental periapical infection, post-traumatic reaction including sequestration, dental impaction, cysts and tumours not related to dental tissues (see 1.8) and odontogenic neoplasia.

Treatment

Some benign, well-differentiated lesions are amenable to surgical removal, but this may necessitate the use of an osteoplastic flap through the mandibular cortex or maxilla to expose and withdraw the tumour piecemeal. Postoperative radiographs are advisable to establish that all aberrant dental tissue has been removed. It may be necessary to close defects into the mouth with a sealant such as dental impression compound while closure by granulation takes place.

Prognosis

Some odontogenic tumours are not amenable to surgical removal by virtue of their size or invasive nature, and euthanasia may be necessary. The surgical removal of a benign odontogenic lesion may necessitate the loss of a permanent tooth. Although the original lesion may cause considerable swelling, bony remodelling should improve the cosmetic result in the months which follow surgery.

Other tumours of the jaw

Pathogenesis

The non-dental tumours of the mandible or maxilla include osteoma, osteosarcoma, ossifying fibroma, fibrous dysplasia and juvenile mandibular ossifying fibroma. Horses in any age group may be afflicted by osteomas, and mature animals are more likely to be the subject of malignant bone tumours, but the other three tumours listed: '(ossifying fibroma, fibrous dysplasia, and juvenile mandibular ossifying fibroma)' occur predominantly in foals. In addition bone cysts may arise in the upper or lower jaws.

Clinical signs

Apart from swelling of the affected jaw, horses with non-dental tumours or cysts may show dysphagia with quidding if there is mechanical interference with mastication, or they may not accept the bit if the interdental space is involved. Expansive lesions of the maxilla may obstruct the flow through the nasolacrimal duct leading to an overflow of tears at the medial canthus.

Diagnosis

In the differential diagnosis of swellings of the mandible and maxilla, suppuration at the roots of the cheek teeth predominates, but bone cysts and non-dental neoplasms should be considered. An extensive firm swelling of either jaw of a foal represents an indication for further evaluation particularly by radiography. A definitive diagnosis requires histological confirmation.

Treatment

Localized lesions may lend themselves to excision on similar lines to those described for odontogenic tumours (see 1.13). Bone cysts should be opened and drained before curettage of the lining. Small cysts may be packed with cancellous bone but larger defects are better packed with medicated gauze which is steadily withdrawn in the postoperative period.

Prognosis

Similar prognostic guidelines apply to these lesions as to odontogenic tumours.

1.14 DIAGNOSTIC APPROACH TO DENTAL DISORDERS

Ageing of horses by dentition

The patterns of eruption and attritional wear of the incisor teeth can be used to estimate the age of horses. The age of horses up to 6 years of age can be determined with some confidence based on the sequence of eruption of the incisors, which is approximately 2.5 years for central incisors (01s), 3.5 years for the middle incisors (02s) and 4.5 years for the corner incisors (03s). Thereafter, the presence or absence of cups, marks and stars, the shape of the occlusal surfaces and the angle of occlusion, and the existence of Galvayne's groove can be used to provide clues upon which to base a 'guestimate'. The variation in individual anatomy, wear rates, diet and the presence of cribbing may lead to a result which is in error by many years.

The permanent cheek teeth also erupt in a constant sequence, 09s (~1 year), 10s (~2 years), 06s (~2.5 years), 07s (~3 years), 11s (~3.5 years) and 08s (~4 years). Thus, the 09s are the oldest teeth in each arcade, and on radiographs, have the shortest crown; the 08s are the last to erupt into the middle of the arcade and have the longest reserve crown.

Clinical signs of dental disease

Many cases with findings of dental disease on oral examination have no clinically discernible signs of the disease from the outside. Spilling of hard feed can often be associated with sharp enamel points causing soft-tissue trauma; however, equally, it may simply be a habit in a particular horse. Riding abnormalities are commonly attributed to dental disease, and in many cases improvement seen following treatment can justify such assumptions; however, the significance of dental

disease in riding problems should not be overestimated or used to justify excessive or unnecessary treatments. In many cases the problems are just as likely to be behavioural, rider performance or related to another non-dental disease.

More significant signs that are likely to represent severe dental disease include halitosis, oral phase dysphagia (particularly involving forage – 'quidding'), unilateral nasal discharge, facial or mandibular swelling and very occasionally, oesophageal obstruction.

Oral examination

A good oral examination is the key to accurate diagnosis of equine dental disease, and it should be done in an ordered manner. An oral examination is indicated as part of a routine health check at least every year, and in younger horses may be required every six months. It can often be included alongside vaccinations as part of a wider routine health check.

The first part of any examination is taking an accurate history. This will include details such as previous dental care history, reasons for examination (e.g. routine check-up, dysphagia, facial swelling), and age and breed of the horse. Much of this history will be taken over the phone so it is essential that individuals within a practice taking phone calls are briefed on the information required.

Clinical examination is best performed in a systematic manner in every case. This may vary between individual practitioners, but a routine of work should be established, which can then be applied in each case to achieve an accurate diagnosis and treatment plan. Initial examination of the horse should be done at a distance, noting demeanour and eating behaviour, as well as using this time to obtain further clinical details from the client.

Once close examination of the horse begins, the horse should be restrained with a headcollar and lead rope. Headcollars which feature adjustable nose bands are particularly useful as they can be adjusted during the course of the examination once a speculum is placed. A basic clinical examination to assess heart, lungs and body temperature, as well as examining other systems for signs of disease, should be undertaken initially to avoid misdiagnosis of non-dental conditions. Examination of the head should start with examination of the external surfaces, looking for any swellings, facial symmetry, signs of nasal or ocular discharge, or lymph node enlargement. Nasal and oral malodour should be noted. Done carefully, this early examination can often calm a nervous horse and/or owner.

After initial examination, the decision to use sedation can be made depending on the response of the patient. It may be that the previous history from the owner has already indicated that this is required, due to the horse's temperament or severity of clinical signs. The use of an alpha 2 agonist, such as detomidine or romifidine, in combination with an opioid, such as butorphanol, is most commonly used and will usually be sufficient. For prolonged examinations or procedures, then infusions of detomidine or romifidine can be administered using an intravenous catheter.

Examination of the teeth begins with assessment of the incisors. Their number, relative location to each other and any overgrowths should be noted. Teeth with extensive periodontal disease or fracture should be assessed carefully. Placement of a conventional speculum for examination of the cheek teeth may be contraindicated in cases where severe incisor disease is present and the speculum is likely to result in further tooth damage.

Careful palpation of the interdental space prior to placing an oral speculum can reveal information regarding the presence of canines and wolf teeth as well as bitting injuries. However, care should be taken as it can place the practitioner's digits at risk of serious injury from being bitten. For this reason it is often preferable to undertake examination of this area using an oral speculum.

Placement of an oral speculum should be done with care. They are large heavy, usually metal, objects, and there is significant potential for injury to the practitioner and other personnel in close proximity. Care should be taken to maintain control of both the horse and the speculum during its placement and use. A handler who can aid restraint is often useful, but in many cases sedation will be required. The use of a twitch is contraindicated due to the proximity of this to the practitioner performing an examination. The incisor plates of the speculum need to be securely located on the incisor teeth. In cases with incisor malocclusions, such as parrot mouth, the use of specially designed plates which can rest on the bars of the mouth may be necessary. These can cause severe injury to the bars if the horse chews so sedation is usually required. It is important to ensure that once in place the poll strap is done up securely, centred, and the speculum is opened gradually ensuring that each side is opened equally to prevent excessive stresses on either the horse's temporomandibular joint or the speculum itself.

Once the speculum is placed and open, a brief manual examination will reveal major pathology and location of food within the mouth. It is sensible for protection of the practitioner's hands and prevention of disease transmission that thin nitrile examination gloves are worn for examination. Following initial examination the mouth should be rinsed using a large flushing syringe to remove all loose food from the mouth.

Palpation of the mouth should be done in a methodical manner, paying attention to the occlusal, buccal and lingual/palatal aspects of each arcade in turn. The soft tissues adjacent to each of these areas should also be palpated. Palpation should be followed up with a visual examination using a bright head light (Figure 1.9). The use of a rigid dental mirror can aid clear visualization of the anatomy of the caudal oral cavity, and is essential for accurate assessment of lesions of the occlusal surface such as caries or secondary dentinal defects and periodontal lesions within the interproximal spaces (Figure 1.10). Dental endoscopy can be useful for recognition and documentation of subtle lesions of the caudal aspect of the oral cavity as well as for documentation of cases, but the equipment can be expensive.

Radiography of teeth

Radiography is the most useful diagnostic tool for imaging teeth. The high contrast afforded by the teeth, bones and surrounding air spaces

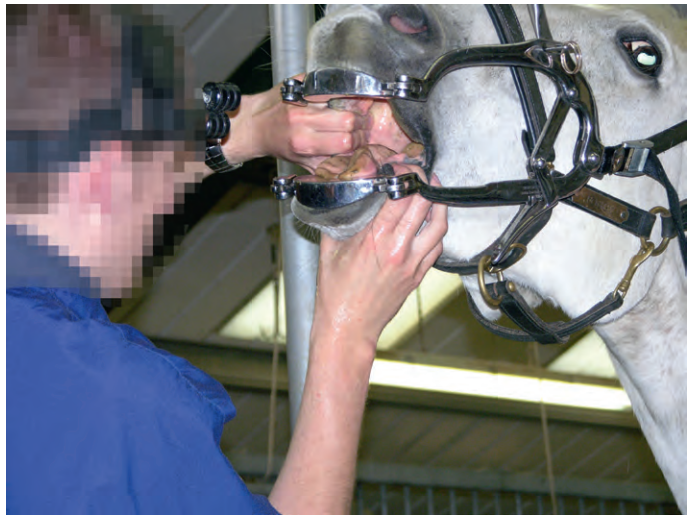


Figure 1.9 Visual oral examination using a Millenium oral speculum and bright headlight.

Figure 1.10 A dental mirror used to assess the occlusal surface of the maxillary cheek teeth; in this case it shows caries affecting the rostral infundibulum.

Figure 1.11 Acquisition of a dorsolateral–lateral 30° oblique radiograph in the standing sedated horse. This view allows assessment of the maxillary cheek tooth apices.

makes imaging easy to accomplish using portable X-ray generators. The use of computed or direct digital radiography has greatly enhanced the quality of image possible in practice and have reduced the need for repeat exposures; however, it is important that the practitioner is experienced in taking the views that are required to assess the teeth and surrounding structures. Due to the hidden nature of the dentition when viewing the skull externally, orientation can be difficult, resulting in poor quality or non-diagnostic images. Due to the relatively long exposures taken in radiography it is essential that movement of the subject is prevented. The use of sedatives, rope halters and, in some instances, head supports can greatly aid in steadying the head to achieve high-quality images.

Radiography of the incisors is best done using an intraoral view. The horse will need to be sedated heavily to prevent chewing whilst the cassette, protected in a plastic bag, is inserted diagonally between the incisor arcades and pushed caudally into the interdental space. Care should be taken not to allow the cassette to come between the cheek teeth. The angle of the X-ray beam will depend on the age of the horse, but rostral angulation of approximately 45 degrees to the cassette will achieve satisfactory images in most cases. The beam should be centred on the tooth of interest, although in cases where this is not clear, initial views centred on midline should be taken. These views, if the cassette is placed far enough caudally, can also be used to assess the canine teeth.

Latero-lateral radiography is useful for imaging the sinuses but seldom useful for imaging the teeth where superimposition obscures the apices and crowns. To image the sinuses, using a low kV and higher mAs provides reduced contrast necessary to assess pathology of soft-tissue density. The images are best taken with the head vertical and the beam centred midway between the rostral end of the facial crest and the eye, with collimation set to include the dorsal aspect of the frontal sinuses and the rostral extremity of the rostral maxillary sinuses. Fluid within the sinuses will appears as multiple straight soft tissue–air interfaces parallel to the ground.

To image the apices of the cheek teeth, laterodorsal 30° lateral obliqued angulation for the maxillary arcades and lateroventral 45° lateral obliqued angulation for the mandibular arcades are required. The cassette should be held parallel to the side of the horse's head being imaged (Figure 1.11). The image should be centred rostro-caudally on the rostral aspect of the facial crest and collimated to include all the cheek teeth in the arcade. Care should be taken to avoid excessive dorsal or ventral angulation as well as ensuring that the beam is directed in a truly lateral plane. The use of higher kV and lower mAs will improve the contrast and aid in assessing the teeth and surrounding boney structures.

Open-mouth oblique and intraoral radiography can be useful in assessing the clinical crown in cases with caries or periodontal disease. In open-mouth obliques, the mouth is held open using a Butler gag, and the beam directed lateroventral-10°-lateral for the maxillary arcades and laterodorsal-15°-lateral for the mandibular arcades, and centred rostro-caudally on the rostral aspect of the facial crest and between the cheek teeth arcades. Intraoral radiography avoids the complications of superimposition; however, the need for specialist cassettes and, in the case of computed radiography, specific equipment, has prevented its widespread use.

Other ancillary diagnostic techniques

Ultrasonography, due to the poor sound transmission of bone, is of limited direct use in equine dentistry. In some cases it may be useful to identify anatomical features, such as blood vessels, prior to surgery.

Endoscopy of the nasal cavities, pharynx and sinuses (through a small sinusotomy) can allow visual inspection of the associated structures of the head and can prove useful in identifying pathology related to dental disease or in ruling out non-dental diagnoses.

Computed tomography can generate extremely useful diagnostic images of the equine head. By producing a series of cross sectional images of the head, the superimposition problems associated with conventional radiography are eliminated. Recently, it has become possible to undertake computed tomography of the head in the standing horse, and this has the potential to revolutionize our understanding of dental disease.

Indications for dental extraction

Indications for dental extraction depend upon the type of tooth to be extracted.

Wolf teeth are the most commonly extracted teeth, and are often removed in young horses to prevent problems associated with bitting.

This has become so routine that consideration is seldom given to their need for extraction, despite the often flawed justifications used to support the procedure. Undoubtedly some wolf teeth, such as rostrally displaced, unerupted ('blind'), fractured or mandibular wolf teeth require extraction, but consideration should always be given as to whether the horse is currently or likely to exhibit bitting problems associated with the wolf teeth prior to extraction being undertaken. Extraction of wolf teeth, whilst usually straightforward, is not without complications. The close proximity of the palatine artery and also the large degree of variation in the apical and coronal anatomy of these teeth mean that careful pre-operative assessments should be made, and the horse should always be sedated. Regional analgesia should also be provided.

Indications for extraction of incisors, canines and cheek teeth are usually associated with pathology, either of the tooth itself, or occasionally due to surrounding disease. Reasons include apical infection, fracture, severe periodontal disease, tooth mobility and severe associated soft-tissue trauma. With the advances in treatment of periodontal disease and the improvement in dental instrumentation, some cases that previously required extractions, such as diastemata and moderate displacements, can be managed by more conservative means. It should be remembered that correct identification and diagnosis of the tooth to be extracted should be achieved before extraction is undertaken. In cases where dental fracture is associated with surrounding bone fracture, extraction at an early stage is contraindicated, as the tooth or teeth may be functioning to stabilize the jaw. In such cases, antibiosis, if the fracture is open or appears infected, followed by careful monitoring are required initially. Subsequent examination and diagnostic imaging once the fracture has stabilized with callus should be used to determine whether dental extraction is required.

Options for the extraction of incisors, canines and wolf teeth

Extraction of incisors and canines is less commonly practised than on wolf or cheek teeth. The principles are similar for both, and extraction can usually be accomplished in the standing horse using sedative infusions and local anaesthesia. Gingival and periodontal elevation may be sufficient in some cases; however, in many, the length and curvature of the reserve crowns of the incisors and canines respectively, necessitate removal of a proportion of the labial alveolar wall. This can be done using a bone chisel to remove the occlusal third of the alveolar wall following sharp dissection of the attached gingiva using a scalpel. After a portion of the alveolar wall is removed, the elevators can more easily be manipulated apically. Once loosened sufficiently, the tooth can be removed from the alveolus using forceps. If possible, the gingiva at the site of alveolar wall resection can be sutured, followed by the placement of a temporary gauze or wax plug to prevent food impaction.

Extraction of wolf teeth is routinely undertaken in the standing horse. Care should be taken not to confuse canines with wolf teeth. The use of sedation is essential to prevent inadvertent trauma to the horse, practitioner or handler from sudden movements. Local anaesthesia administered in the mucosa on either side of the tooth will help avoid the need for excessive sedation. The direction of the tooth should then be ascertained, remembering that many unerupted wolf teeth run in a horizontal direction along the maxillary bars. Using a long-handled dental luxator with the speculum in place, the periodontal ligaments of the tooth can be transected. The use of traditional cylindrical Burgess elevators should be avoided due to the high likelihood of crown fracture and excessive boney trauma caused by their use. Once all aspects have been cut using the luxator, the tooth can be removed from the alveolus using a small pair of forceps. Care should be taken to ensure adequate restraint to avoid trauma to the palatine artery which lies medial to wolf teeth in the maxillary arcades. Wolf teeth in the mandibular arcades are less common, and they present a greater challenge due to the anisognaethic conformation of the horse, though the same principles of removal apply.

Options for the extraction of cheek teeth

Extraction of equine cheek teeth is a major act of surgery in all cases and can carry significant risks of complication. In the last ten years, improvements in sedatives, analgesics and local anaesthetic techniques have led to standing oral extraction becoming the technique of choice.

Oral extraction relies on the presence of sufficient clinical crown to allow placement of molar forceps, and in those cases where this is compromised then alternative techniques may be required. Initial examination and diagnostic techniques should be used to identify the tooth requiring extraction. In cases where the diseased tooth cannot be easily identified, or if the signs of disease are not definitively dental in origin, extraction should not be undertaken. The patient should be sedated in a quiet environment; the authors' preference is to use a combination of the alpha2-agonist detomidine with morphine, prior to a short-stay intravenous catheter being placed. This allows maintenance of sedation using an infusion of detomidine in saline solution. Further analgesia can be provided using NSAIDs and local anaesthesia of the maxillary or mandibular branches of the trigeminal nerve. Administration of local anaesthetic into the gingiva of the tooth to be extracted using a butterfly catheter and crocodile forceps can provide further analgesia during initial elevation of the tooth. The correct tooth should be carefully identified in the mouth, and the gingival attachments elevated using curved right-angle periodontal elevators. By placing these into the periodontal space and then twisting them outwards, the gingiva can be loosened from the tooth. Following elevation, a molar spreader is placed into the interproximal space on each side of the tooth being extracted. Care should be taken when extracting 07 (second cheek) teeth not to place molar spreader rostral to the tooth which will only serve to loosen the 06 lying rostrally. The molar spreader need to be located precisely within the interproximal space, its position should be checked with a dental mirror prior to gradually closing the handles, which once fully closed should be left in place for 5 minutes to stretch the periodontal ligament. Molar spreaders of different sizes can be used sequentially to achieve further loosening, although the use of excessively large spreaders can result in damage to the crown of both teeth on either side of the interproximal space.

Once spreading of the teeth has been completed, a molar forceps is placed on to the clinical crown of the tooth (Figure 1.12). The style of forceps used depends on the nature of the tooth being extracted as well as practitioner preference, although for most situations a pair of universal serrated jaw molar forceps is suitable. Once around the tooth the jaws of the forceps can be slid apically before being secured on the tooth using a length of mountain bike inner tube wrapped tightly around the handles. The tooth can then be manipulated using the forceps to rotate the tooth within the alveolus. The oscillation of the handles will initially be small, and care should be taken not to exert excessive force which may result in crown fracture. As the tooth loosens blood released from the periodontium will froth around the tooth and can be used to confirm that movement of the forceps is from the tooth shifting and not from the jaws slipping on the crown. Gradually, the movement of the forceps will increase, resulting in a squelching noise from air sucked into and out of the alveolus, and the resistance felt to movement of the handles will decrease. At this stage, pressure, using a fulcrum placed on the tooth in front, can be used to ease the tooth out. If extraction is not easily achieved then

Figure 1.12 Molar forceps, in this case narrow jaw fragment forceps, secured around a dental fragment following elevation and spreading.

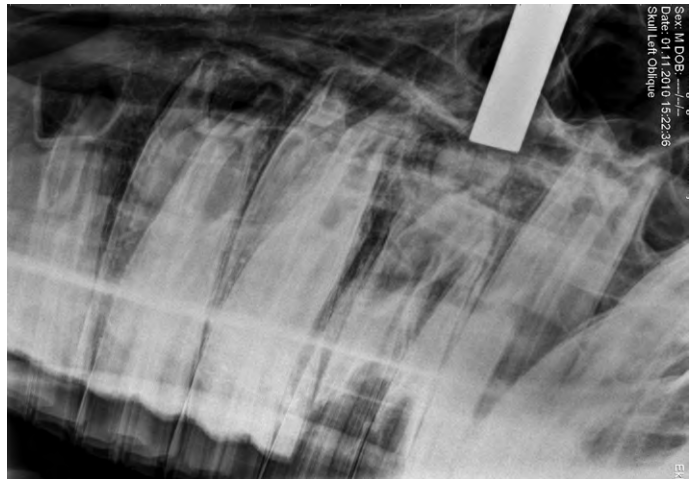


Figure 1.13 Intra-operative radiographs showing correct placement of the punch over the apex of 209 being extracted. In this case slight adjustment to the direction of the punch, to aim more rostrally, was required.

further manipulation is required before repeating use of the fulcrum. After the tooth has been removed, it should be examined, and the alveolus palpated to ensure that the extraction is complete. Repeating the radiographs is also useful to ensure that the extraction has been complete. The alveolus can then be plugged using a thermoplastic dental wax or two-part vinyl polysiloxane impression material, care being taken to ensure that only the occlusal half of the alveolus is plugged. This can remain in place for three weeks to ensure that a solid base of granulation tissue is formed within the alveolus.

Repulsion is indicated in cases where insufficient clinical crown is present to allow oral extraction. This procedure can be done with the horse anesthetised or sedated; however, it should be remembered that many complications arise from incorrect placement of the punch, and as such, sedation may not provide sufficient restraint to prevent these complications from arising. The exact site of the apical approach is defined using radiographs and skin staples. Following sterile preparation, an apical approach to the tooth is made using a Galt trephine. A punch is then placed on the apex of the tooth, and intra-operative radiographs taken to ensure that the placement of the punch is correct (Figure 1.13). If adjustments are necessary then these should be made before repeating the radiographs. Once correct placement is confirmed, an assistant places his or her hand in the mouth onto the crown of the tooth being extracted. The punch is then advanced by gentle tapping with a surgical mallet, with the assistant providing feedback by palpating the shocks transmitted through the tooth. Gradually the force applied to the punch can be increased, but it is important to ensure that this does not alter direction or placement of the punch. If insufficient progress is made or if the feedback from the assistant changes, then repeating radiographs to reassess the punch location is indicated. After the tooth begins to move then the force and frequency of striking the punch are reduced. The tooth is then removed from the oral cavity. The trephine site and alveolus should then be debrided using surgical swabs pushed through into the oral cavity. Repeat radiographs, alongside careful inspection of the tooth, should be used to check that the extraction is complete. As with oral extraction, the occlusal half of the alveolus is plugged using dental impression wax or an acrylic. Depending on practitioner preference, the trephine site may be closed surgically or left open to heal by secondary intention.

Buccotomy extraction comprises an incision through the cheek followed by removal of the lateral bony support of the alveolus and withdrawal of the tooth laterally. When the oral cavity is entered at the dorsal or ventral buccal cleft the tooth to be removed can be positively identified so that the possibility of extraction of the wrong tooth should not arise, and inadvertent damage to a neighbouring tooth is unlikely. The plate of bone lying lateral to the diseased tooth is resected to expose the reserve crown and roots of the tooth. Space for extraction is created by a longitudinal cut in the tooth using a burr. After removal of the tooth, the alveolus is packed with medicated dressing which is withdrawn over the following 14 days. The oral defect is plugged with a cap of dental impression compound, and the buccotomy incision is closed in four layers starting with the gingival mucosa. Structures to be avoided during the buccotomy approach include the parotid duct, the buccal venous plexus and the dorsal buccal branch of the facial nerve. Extraction by this route is restricted to maxillary 06–08s and mandibular 06–10s because of the position of the masseter muscle, paranasal sinuses and local vasculature.

Post-operative management immediately after extraction will largely be dictated by the procedure used. However, assessment of post-operative healing is necessary to achieve early resolution of post-operative complications such as sequestrum or fistula formation. The use of dental plugs varies between practitioners, but if they are temporary in nature, they should be removed in order that healing can be assessed and to avoid complications arising from their retention as a potential foreign body.

Longer term post-operative management should focus on the lack of occlusion with the opposing arcade created by removal of the tooth. Regular appointments for oral examination and rasping should be maintained every 6 months to prevent overgrowths of the opposing teeth, which, if left unmanaged, can result in significant soft-tissue trauma, as well as the problems which may arise from their attempted correction. Careful monitoring of the arcade which has lost a tooth is also required. The normal mesial drift which maintains tight interproximal occlusion will often result in the site of extraction becoming narrower. It has been reported in some cases that this drift alongside the lack of a tooth to oppose these forces results in the opening up of the interproximal spaces between the other teeth in the arcade, giving rise to diastemata and associated periodontal disease.

FURTHER READING

Easley J, Dixon PM, Schumacher J (eds) (2011) Equine dentistry, 3rd edn. Elsevier, Philadelphia

Johnson TJ (2003) Correction of common dental malocclusions with power instruments. In: Robinson NE (ed) Current therapy in equine medicine 5, section 3. Elsevier, Philadelphia

Johnson TJ (2009) Iatrogenic damage caused by modern dentistry procedures. In: Robinson NE, Sprayberry KA (eds) Current therapy in equine medicine 6, section 8. Elsevier, Philadelphia

Klugh DO (2009) Periodontal disease. In: Robinson NE, Sprayberry KA (eds) Current therapy in equine medicine 6, section 8. Elsevier, Philadelphia

Klugh DO (2010) Principles of equine dentistry. Manson Publishing, London

Porter C (2009) Dental nerve blocks. In: Robinson NE, Sprayberry KA (eds) Current therapy in equine medicine 6, section 8. Elsevier, Philadelphia

Tremaine WH (2009) Dental extraction. In: Robinson NE, Sprayberry KA (eds) Current therapy in equine medicine 6, section 8. Elsevier, Philadelphia

Chapter 2

Gastroenterology 1. Colic

G. Barrie Edwards

CONTENTS

2.1 Introduction 21
False colics 22
Alimentary (true) colic 22
Classification of colic 22
2.2 Pathophysiology of intestinal obstruction 22
Simple obstruction 22
Strangulating obstruction 24
2.3 Pharmacological management of colic 25
Aims of therapy 25
Types of drug 25
Fluids 27
2.4 Common types of colic 27
Spasmodic colic 27
Impaction colics 28
Flatulent / tympanitic colic 29
2.5 Grass sickness 30
2.6 Anterior enteritis (proximal enteritis, gastroduodenal jejunitis) 30
2.7 Approach to diagnosis in colic cases 32
Signalment 32
History 32
Clinical examination 33
Classification into categories 35
2.8 The decision for surgery 35
2.9 Preparation of the horse prior to transport to a surgical centre 37
2.10 The surgical management of the equine colic case – general points 37
Premedication and anaesthesia 37
Celiototomy (laparotomy) techniques 38
Examination of the small intestine 38
Examination of the large colon 38
Evaluation of gut viability 39
Resection of small intestine 40
Resection of large intestine 42
Closure of the midline incision 43
2.11 Postoperative management of the colic case 44
Monitoring protocol 44
2.12 Postoperative complications 45
Complications during recovery from anaesthetic 45
Complications during the hospitalization period 45
Further reading 47

2.1 INTRODUCTION

Acute abdominal pain (colic) in the horse is one of the most frequent emergency conditions encountered in practice. It is one which understandably causes owners considerable concern and apprehension, and about which few of them have any real understanding.

Colic is not a specific disease or even a diagnosis, it is only a symptom and represents a challenge of differential diagnosis. It simply indicates that the horse has pain, usually but not invariably in its abdomen and usually related to its gastrointestinal tract.

The horse with gastrointestinal pain can behave in a variety of ways:

- In response to mild pain it may occasionally paw the ground, turn its head to its flanks, stretch out or lie down for longer than normal.
- When moderate pain is present, the horse may show pawing, cramping with attempts to lie down, kicking at the abdomen, lying down and attempting to roll or rolling and turning its head to its flank. The horse also continues to move when not rolling.
- If the pain is severe, sweating, dropping to the ground, violent rolling and continuous movement or pawing are the signs usually displayed.

Such behaviour is not confined to horses with alimentary colic, and many of the signs described may be shown by horses which have one of a number of painful conditions unrelated to the gastrointestinal tract (i.e. 'false colic').

© 2013 Elsevier Ltd
DOI: 10.1016/B978-0-7020-2801-4.00004-3

False colics

- A mare with uterine torsion in the last trimester of pregnancy will exhibit signs of moderate colic due to tension on the uterine broad ligaments brought about by rotation of the uterus.
- A horse with acute exertional rhabdomyolysis may suddenly stop during exercise, drop to the ground, sweat and kick out.
- Severe impairment of blood supply to the hind limbs caused by aortoiliac thrombosis can similarly cause a horse to exhibit signs normally associated with severe colic due to intense muscle pain.
- Severe bladder distension due to urethral obstruction by a calculus, pleuritis, liver disease and laminitis can also simulate gastrointestinal colic.

An intelligent approach to the management of colic requires an understanding of the causes and mechanisms of gastrointestinal pain and the pathophysiological changes which occur as the result of intestinal obstruction.

Alimentary (true) colic

The majority of colic cases are associated with disruption of the normal gut motility brought about by a variety of factors such as diet, management and parasites. Certain anatomical features of the equine alimentary tract, particularly the large colon, also predispose to obstruction.

Several factors can contribute to the pain associated with alimentary (true) colic:

- An increase in intramural tension is probably the most common cause and may be brought about by distension associated with excessive fermentation or accumulations of fluid and/or gas or to obstructions caused by impaction, displacements or strangulation obstructions.
- Spasm of intestine associated with hypermotility and disruption of the normal coordinated contractions of bowel is another frequent cause.
- Pain due to tension on mesentery may well accompany intestinal displacements, torsion, hernias and intussusceptions.
- Ischaemia of gut brought about by vascular occlusion due to a large variety of strangulating obstructions to which the horse's intestine is prone results in the rapid onset of severe pain due to hypoxia which later abates when the gut becomes necrotic.
- Mucosal inflammation and irritation present in conditions such as acute salmonellosis and right dorsal colitis result in mild colic.
- Ulceration of the mucosa such as gastric ulcer syndrome.

The pain associated with these factors is designated *visceral pain* and is manifested by the clinical signs described earlier. The pain associated with widespread peritonitis is classed as *parietal pain*. In marked contrast to horses with visceral pain, those with severe parietal pain show great reluctance to move and obvious boarding of the abdominal wall.

Depending on the cause of the problem, one or more of these factors may be contributing to the pain in individual colic cases. For example, in horses with spasmodic colic, hypermotility and incoordinated contractions are the sole cause whereas in the early stages of a strangulation obstruction, ischaemia, hypermotility, distension and mesenteric tension may all be contributing to the pain exhibited.

Classfication of colic

Although acute abdominal pain is a frequent occurrence, the majority of cases are benign and respond readily to medical therapy. The remainder are potentially life-threatening and require surgical interference if the life of the horse is to be saved.

Colic can be conveniently classified into seven types:

1. Spasmodic.
2. Impactive.
3. Flatulent.
4. Obstructive.
5. Non-strangulating infarction.
6. Enteritis.
7. Idiopathic.

Colic cases with a mild or uncomplicated disease process fall predominantly into the first three categories while those with serious life-threatening conditions usually have obstructive lesions.

The earlier these various disorders are recognized and specific therapy instituted, the better is the prognosis for recovery. In the early stages pain is the common factor, and there may be little or nothing to differentiate the benign from the serious cases. A basic understanding of the pathophysiology of gastrointestinal obstruction is essential if the pathological effects of obstruction on the clinical values used to diagnose and evaluate colic cases are to be recognized and interpreted correctly.

2.2 PATHOPHYSIOLOGY OF INTESTINAL OBSTRUCTION

Any interference, mechanical or functional, with the progression of intestinal contents constitutes obstruction.

- The obstruction is said to be *simple* when the obstructive process is not complicated initially by vascular compromise of the bowel.
- In *strangulating obstruction* there is obstruction to both the blood supply and the lumen of the intestine.
- Obstructions due to intravascular occlusion of the blood supply, frequently associated with *Strongylus vulgaris* larvae in the cranial mesenteric artery or its branches are described as *non-strangulating infarction*.

The mechanisms of shock that may accompany some forms of intestinal obstruction are detailed in Chapter 26. Absorption of endotoxins (lipopolysaccharide component of the outer cell membrane of Gram-negative bacteria) into the circulation is termed endotoxaemia. Endotoxin activates inflammatory cells (including mononuclear cells, neutrophils and platelets) to release numerous inflammatory mediators resulting the systemic inflammatory response syndrome (SIRS) (see section 26.2).

Simple obstruction

Small intestine

1. Physical obstruction of the small intestine usually occurs by impacted food material, stricture, or foreign body (Box 2.1), thereby preventing the passage of the large volumes of fluid produced in the upper alimentary tract from reaching the absorptive surfaces of the lower intestine so that it becomes sequestered or maybe lost by nasogastric reflux.
2. During one day this volume almost equals the extracellular volume of the horse (approximately 125 L).
3. Systemically, the prime concern in simple obstruction is depletion of plasma volume and reduction in cardiac output together with acid–base disturbances.

Box 2.1 **Small intestinal obstructions**

I. Mechanical obstructions

Simple obstruction

A. Intraluminal.

1. Impacted food.
2. Ascarid impaction.
3. Foreign bodies.

B. Mural.

1. Ileal hypertrophy.
2. Idiopathic focal eosinophilic enteritis.
3. Neoplasia.
4. Proximal duodenal stricture (foals).

C. Extraluminal.

1. Congenital strictures.
   (a) Non-strangulating mesodiverticular bands.
   (b) Meckel's diverticulum.
   (c) Atresias.
2. Acquired strictures.
   (a) Inflammatory (adhesions and abscesses).
   (b) Trauma.
3. Compression.
   (a) Intra-abdominal abscesses.
   (b) Displaced large bowel.

Strangulating obstruction

A. Internal hernias.

1. Epiploic foramen.
2. Diaphragmatic.
3. Mesentery of small intestine.
4. Gastrosplenic mesentery.
5. Broad ligament of uterus.
6. Mesodiverticular bands.

B. External hernias.

1. Inguinal.
2. Umbilical.
3. Traumatic.

C. Pedunculated lipomas.

D. Intussusception.

E. Volvulus.

F. Fibrous bands and adhesions.

G. Arterial thrombosis.

II. Paralytic Ileus

A. Primary – grass sickness.

B. Secondary.

Postoperative.

Peritonitis.

4. Gas production by bacterial action continues and is even enhanced by the static medium. With continued secretion of fluids and build-up of gas, the intraluminal hydrostatic pressure (IHP) increases and distends the bowel.
5. As stretch receptors in the distended intestinal wall are activated, the pain increases and becomes continuous.
6. Peristaltic waves diminish and then cease altogether as the intestinal lumen is progressively filled, leaving an atonic rapidly distending tube.
7. Once IHP increases to above 15 cm H_2O, absorption of water by the mucosa stops and instead water begins to flow from the mucosa into the lumen.
8. The increasing pressure and expanding volume of fluid causes reflux into the stomach – how soon after the onset of the obstruction depends on where along the small intestine it is located.
9. Increased vascular hydrostatic pressure in the bowel promotes leakage of protein-rich plasma into the peritoneal fluid. Few leucocytes and no erythrocytes appear in the peritoneal fluid within the first 12–24 hours but may become more numerous with the progression of degenerative changes and vascular compromise of the intestinal wall.
10. Damage may be sufficient to allow absorption of endotoxins and cause production of prostaglandins and leukotrienes which may further compromise cardiovascular function. However, endotoxic shock plays only a very limited part in the fatal outcome of unrelieved simple obstruction. Hypovolaemia and altered blood electrolytes are the usual causes of cardiovascular collapse.

The severity of clinical signs associated with a simple obstruction of the small intestine depends on the degree of obstruction (partial or complete) and the level of obstruction (proximal or distal).

- In general, proximal obstructions have a more acute onset, produce greater pain, generate a greater volume of gastric fluid sequestration and have a more rapidly fatal course than distal obstructions. In proximal obstructions, large quantities of chloride are lost particularly if gastric reflux is removed by a nasogastric tube, resulting in metabolic alkalosis.
- Later the metabolic disturbance becomes complicated by acidosis secondary to hypoperfusion.
- Clinical signs resulting from distal small intestine obstructions develop more slowly and are generally less severe due to the compliance of the intestine and the ability to continue some fluid absorption until IHP initiates secretion.
- Established distal small intestine obstructions are characterized by metabolic acidosis with low serum concentrations of HCO_3.

Large intestine

1. Simple obstruction of the large intestine (Box 2.2) is usually due to impaction with food material, enteroliths or other intraluminal masses, or a change in position of the colon, e.g. nephrosplenic entrapment (left dorsal displacement of the large colon), and may be partial or complete.
2. In general the clinical signs or rate of systemic degeneration are much less dramatic in simple obstruction of the large intestine than in simple obstruction of the small intestine.
3. Incomplete obstruction allows the passage of small amounts of ingesta and gas.
4. Dehydration is mild at first because water still passes into the caecum where it is readily absorbed.
5. The production of volatile fatty acids and gas by bacterial fermentation is reduced due to decreased amounts of ingesta.
6. If the obstruction becomes complete, ingesta, and particularly gas, accumulate much more rapidly. Distension becomes marked and may become so great as to exert pressure on the diaphragm and vena cava, resulting in impaired pulmonary function and venous return to the heart.

Box 2.2 **Large intestine obstruction**

I. Simple obstruction

A. Congenital abnormalities.

Atresia of large or small colon.

B. Impaction.

C. Intraluminal concretions.

1. Enteroliths.
2. Trichobezoars.
3. Phytobezoars.

D. Foreign bodies.

1. Sand.
2. Nylon.

E. Displacement of large colon.

Nephrosplenic entrapment (left dorsal displacement).

Right dorsal displacement.

Retroflexion.

F. Strictures.

1. Adhesions.
2. Fibrous bands.
3. Idiopathic focal eosinophilic enteritis.
4. Neoplasia.

II. Strangulating obstruction

A. Volvulus.

Large colon.

Caecum.

B. Intussusception.

1. Caecocaecal.
2. Caecocolic.
3. Colonic.

C. Pedunculated lipoma (small colon).

D. Vascular disease.

Thromboembolic infarction.

Rupture of mesocolon.

Submucosal haematoma (small colon).

7. Prolonged and/or marked distension of the caecum and colon may cause interference with mucosal perfusion leading to devitalization and possibly fatal rupture.

Strangulating obstruction

Small intestine

1. Strangulating obstructions of the small intestine include incarcerations, intussusceptions and volvulus (Box 2.1) and represent a common cause of acute abdominal crisis.
2. The same fluid retention which occurs due to simple obstruction with eventual reflux into the stomach is present, but because vascular compromise of the intestine is present at the outset, the pathophysiological changes associated with strangulation obstruction are more acute and severe.
3. The incidence of mortality of surgical cases with strangulation obstruction can be high.
4. The vascular compromise may be venous, or venous and arterial, but typical lesions cause venous occlusion before arterial occlusion with consequent venous congestion.
5. Within minutes of strangulation occurring the involved segment of bowel and its mesentery become deep red as veins and venules are distended with blood. If there is immediate concurrent arterial occlusion the intestine becomes cyanotic.
6. More often, thicker walled arteries and arterioles resist compression and continue to pump blood into the distended veins and venules. As the involved intestine is engorged with blood, vascular stasis quickly develops, and the segment becomes red/black in colour. Almost immediately, the vascular endothelium becomes more permeable, and plasma diffuses into the tissue.
7. Within a few hours degeneration of vascular epithelium becomes so extensive that blood pours out of the distended vessel into the tissue (venous infarction stage) and eventually into the lumen.

The mucosal villi are extremely sensitive to hypoxia, and within minutes after oxygen deprivation, ultrastructural morphological changes are evident.

- The epithelial cells slough in sheets starting at the tip of the villus and working towards the crypts.
- Within 4–5 hours the mucosal epithelium is completely necrotic.
- By 6–7 hours the degenerative effects of hypoxia have extended through the external muscle layer.

As soon as the mucosal barrier is damaged Gram-negative bacteria and endotoxins permeate the lamina propria and submucosa.

- Early in the development of the ischaemic lesions, the bacteria and endotoxins readily gain entry to the circulation via viable tissue adjacent to the lesion.
- By 6 hours or possibly earlier, as the muscularis degenerates, bacteria and toxins leak through the serosa into the peritoneal cavity from which they are readily absorbed.
- Release of endotoxins into the general circulation results in damage to endothelial cells and platelets. Platelets are immediately stimulated and release the potent vasoconstrictor substances, thromboxane and serotonin. Damage to the endothelium increases vascular permeability, prostacyclin is released, and neutrophils are stimulated, especially in the lungs and site of intestinal injury.
- The endotoxic shock is dose-related and is more severe the greater the length of bowel involved.

The clinical picture is acute with severe pain which is continuous and shows no, or only temporary, response to analgesics.

- The heart rate increases progressively, and pulse quality deteriorates.
- Mucous membranes become congested, and the capillary refill time increases.
- The PCV and total protein also rise progressively, and the respiratory rate increases in response to developing metabolic acidosis.
- At first the peritoneal fluid is slightly serosanguinous with a mild increase in protein and leucocytes. As the strangulation process continues, all these substances increase dramatically, and the fluid becomes flocculent and turbid. Toxic neutrophils indicate leakage of toxins and bacteria.
- The clinical course is rapid, and most horses with an untreated strangulating obstruction of the small intestine die within 24–30 hours of the onset of disease from irreversible endotoxaemic shock / systemic inflammatory response syndrome and marked vascular collapse. However, the deterioration in the animal's condition is such that for surgical correction to be successful, it must be carried out within a few

hours of the obstruction occurring. Eighty per cent or more of affected horses may recover if operated upon within 8 hours.

Large intestine

1. Strangulating obstructions of the large intestine (Box 2.2) include intussusception of the caecum, torsion and volvulus of the large colon and incarceration of the small colon.
2. The pathophysiology is similar to that previously described for the small intestine, but there are points of variance.
3. The rate of systemic deterioration can vary markedly between caecocaecal intussusception in which it is slow, and 360° torsion of the large colon which is the most rapidly fatal of all the intestinal obstructions of the horse.
4. Such is the size of the submucosal space in the large colon that venous occlusion can result in the horse losing half its circulating blood volume into the wall of the gut within 4 hours of a 360° torsion occurring.
5. Hypovolaemia is rapidly profound, and the mucous membranes become pale and cyanotic.
6. The degeneration of the large surface area of bowel wall allows massive leakage of endotoxin and bacteria into the peritoneal cavity, and the effects of endotoxaemia are added to those of the hypovolaemia.
7. Because of the short clinical course prior to death, rupture is not normally seen.

2.3 PHARMACOLOGICAL MANAGEMENT OF COLIC

Aims of therapy

The aims of therapy are:

- To relieve pain.
- To restore normal propulsive motility of gut without masking the clinical signs that must be monitored for proper assessment of the horse's condition and progress.
- Correction and maintenance of hydration and electrolyte/acid–base balance.
- Treatment for endotoxaemia.

Relief of pain

- Relief of visceral pain in horses with severe colic is essential on humane grounds and to minimize injury to the horse and attending personnel during evaluation and therapy.
- The most satisfactory method of pain relief is the correction of the cause of increased intramural tension resulting from distension or spasm. However, this may take time, and it is frequently necessary to achieve temporary relief of severe pain chemotherapeutically to allow a thorough clinical examination without risk of injury to the horse and attending personnel.
- It is important to select a drug which will accomplish the desired effect without creating complications such as depressing gut activity, predisposing to hypovolaemic shock or, most important, masking the signs of developing endotoxaemia.

Types of drug

A wide variety of therapeutic agents are used to treat equine colic. They include:

- analgesics to control visceral pain.
- agents to normalize intestinal contractions during adynamic ileus.
- anti-inflammatory drugs to reduce the adverse effects of endotoxin.
- agents to soften and facilitate the passage of ingesta.
- drugs to improve cardiovascular function during endotoxic and hypovolaemic shock.

Non-steroidal anti-inflammatory drugs (NSAIDs)

Amongst the most useful analgesics for both surgical and non-surgical disease are the non-steroidal anti-inflammatory drugs. The therapeutic and adverse effects of these drugs result from inhibition of cyclooxygenase enzyme-mediated biosynthesis of prostaglandins.

The NSAIDs commonly employed – dipyrone, phenylbutazone, ketoprofen, meloxicam, firocoxib and flunixin meflumine – differ greatly in efficacy in the treatment of visceral pain in horses.

Dipyrone

Dipyrone is a very weak analgesic drug that provides only short-term relief in a few cases of very mild abdominal pain. Combined with hyoscine N-butylbromide it is effective in relieving intestinal spasm. Its failure to help reduce or stop pain in individual cases should signal that a condition exists which is more serious than a simple intestinal spasm or tympanic colic.

Phenylbutazone

Phenylbutazone provides no greater relief from visceral pain than does dipyrone. However, the toxic side-effects of phenylbutazone are numerous and include gastrointestinal ulceration and nephrotoxicity. For this reason the dosage should not exceed 4.4 mg/kg every 12 hours.

Meloxicam

Meloxicam is available in Europe for the alleviation and relief of pain associated with musculoskeletal disorders and colic. Meloxicam selectively inhibits cyclooxygenase-2 (COX-2) over COX-1, and therefore has a lower risk of side-effects compared to phenylbutazone or flunixin meglumine. The analgesic effects are equivalent to phenylbutazone.

Firocoxib

Firocoxib is a selective COX-2 inhibitor available in north America for control of pain associated with musculoskeletal disorders. There is currently no official approval for its use for control of pain associated with colic.

Flunixin meglumine

- Flunixin meglumine is effective in the control of visceral pain in horses and has been shown to block the production of prostaglandins, specifically thromboxane and prostacyclin, for 8–12 hours after a single dose.
- The duration of analgesia produced varies from 1 hour to more than 24 hours depending on the cause and severity of the pain.
- Although this drug has basic side-effects similar to phenylbutazone, the greater risk associated with its use devolves from its ability to mask clinical signs of intestinal strangulation or obstruction by reducing heart rate, relieving pain and improving mucous membrane colour.
- If administered to horses in which the precise cause of colic has not been ascertained, it is essential to closely monitor rectal examination findings, nasogastric reflux, peritoneal fluid, heart rate and respiratory rate over the next few hours.
- It should be administered to control severe pain and diminish the effects of endotoxins in horses needing transport to a referral centre for surgery.

Sedatives

Xylazine

- Xylazine produces both sedation and visceral analgesia by stimulating alpha$_2$ adrenoceptors in the central nervous system (CNS), thereby decreasing neurotransmission. At a dose rate of 1.1 mg/kg IV, the visceral analgesia it provides is similar to that of flunixin and the narcotics.
- The duration of effect of xylazine is much shorter (usually 10–30 minutes) than that of flunixin, making xylazine more useful for controlling pain during evaluation of the cause of colic and of the need for specific therapy.
- Potentially detrimental side-effects of xylazine include bradycardia, decreased cardiac output, transient hypertension followed by hypotension, ileus and decreased intestinal blood flow, and may affect its use in horses in shock.
- In contrast to the bradycardia, hypertension and reduced intestinal blood flow, which last only a few minutes, the ileus and hypotension can be prolonged.
- A reduced dosage of 0.2–0.4 mg/kg IV can be administered in an attempt to reduce the severity and duration of the side-effects. Alternatively it can be used at the lower dosage in combination with a narcotic agonist such as butorphanol.

Detomidine

- Detomidine, another alpha$_2$ adrenoceptor agonist, is a more potent sedative and analgesic than xylazine.
- The same complicating effects are likely to be present for detomidine as for xylazine.
- Detomidine reduces intestinal motility similarly to xylazine and can mask many of the signs which assist the clinician to diagnose the cause of the colic.
- Because it is such a potent drug, any signs of colic observed within an hour of administration are an indication that a severe disease is present, one which may require surgery.
- Therefore it is a useful drug when used with caution, and preferably at the low dosage of 10 µg/kg IV.

Romifidine

Romifidine has a similar action to xylazine and detomidine. At a dose of 40–80 µg/kg IV it provides potent analgesia lasting 1–3 hours.

Acepromazine

Phenothiazine tranquillizers have a peripheral vasodilatory effect and so are contraindicated in horses with reduced circulatory volume because they block the life-saving vasoconstriction which maintains arterial blood pressure and assures, within limits, perfusion of vital organs.

Narcotic analgesics

The analgesic and sedative effects of these drugs result from interaction with central and/or peripheral opioid receptors.

Morphine

- Morphine and pethidine are opioid receptor agonists.
- They are potent analgesics but can cause excitement in horses unless used in combination with drugs like xylazine.
- Morphine is known to reduce progressive motility of the small intestine and colon while potentially increasing mixing movements and increasing sphincter tone.
- The disadvantages of morphine are sufficient to discourage its use in horses with abdominal disease.

Pethidine

Pethidine is a narcotic agonist with few side-effects and provides slight to moderate analgesia of relatively short duration in horses with abdominal pain. Used repeatedly it can potentiate obstructions due to impactions by reducing colonic activity.

Butorphanol

- Butorphanol is a partial agonist and antagonist which gives the best pain relief of the drugs in this group, with least side-effects.
- It can be used in combination with xylazine in horses with moderate to severe abdominal pain.
- The dose can vary from 0.05 to 0.075 mg/kg. Doses exceeding 0.2 mg/kg can cause excitement.
- Butorphanol reduces small intestinal motility but has minimal effect on pelvic flexure activity.
- It is potent enough to stop colic for short periods of time when colic is due to severe intestinal disease, but the pain from large colon torsion or small intestinal strangulation may not be altered.

Pentazocine

Pentazocine is a partial agonist which is more effective than dipyrone but less effective than xylazine and flunixin in relieving visceral pain.

Spasmolytics

Increased frequency of intestinal contractions as in spasmodic colic can be relieved by spasmolytics. Spasmolytic drugs include cholinergic blockers such as atropine and hyoscine N-butylbromide.

Atropine

Atropine is not recommended for use in horses with colic because its effect in relaxing the intestinal wall and preventing contractions can last for several hours or even days creating tympany and complicating the initial problem with ileus.

Hyoscine

- Hyoscine has a shorter muscarinic cholinergic blocking effect compared to atropine and is effective in relaxing the bowel wall.
- It is available in Europe combined with dipyrone.

Laxatives

Laxatives are commonly used on horses with colic to increase the water content and softness of ingesta thereby facilitating intestinal transit. The most common indication for their use is the treatment of large colon impactions.

Mineral oil

Mineral oil (liquid paraffin) is the most frequently used laxative in equine practice and is administered at a dose rate of 10 mL/kg by nasogastric tube. Its effects are considered mild, and it is safe for prolonged use. Vegetable oils can be used in the same way.

Psyllium hydrophilic mucilloid

- Psyllium hydrophilic mucilloid is a bulk-forming laxative which causes the fluid and ion content of faeces to increase by absorbing water.
- It is particularly useful for treating impactions caused by ingested sand.

Osmotic laxatives

Magnesium sulphate and common salt can be used as an osmotic laxative in horses, but because, undiluted, they will cause enteritis by

osmotic damage to the mucosal cells, each dosage of 0.5–1.0 g/kg should be diluted in 4 litres of warm water and administered by nasogastric tube.

Dioctyl sodium succinate (DSS)

- DSS is an amonic, surface-active agent with wetting and emulsifying properties.
- It reduces surface tension and allows water and fat to penetrate the ingesta.
- DSS can cause damage to the mucosa and increases fluid permeability of colon cells.

Intravenous and enteral fluids

Balanced electrolyte solution administered intravenously will sometimes provide a stimulus for intestinal motility. This treatment works particularly well for colon impaction and appears to stimulate motility in cases of ileus of the caecum and large colon. 'Overhydration' with Hartmann's solution at 40–80 L every 24 hours helps to provide secretion of fluid to soften hardened impactions, however when fluid therapy is stopped, the horse may suffer a rebound dehydration. Continuous or repeated administration of enteral fluid by nasogastric tube can also be used in the treatment of impactions.

Drugs that alter intestinal motility

Postoperative ileus is the most common indication for pharmacological manipulation of intestinal contractile activity.

Neostigmine methyl sulphate

- Neostigmine directly stimulates intestinal contractions.
- The duration of effect is very short (15–30 minutes), and the drug may decrease propulsive motility of the jejunum and delay gastric emptying in horses. It can cause abdominal pain by stimulating spasmodic regional contractions.

The preferred method of correcting postoperative ileus is to specifically antagonize the inhibiting neurogenic or hormonal processes.

Metoclopramide

Metoclopramide appears to have a beneficial effect on stomach emptying and small intestinal motility when used as a constant drip infusion at 0.1 mg/kg/hour over several hours or constantly until some response is seen. Higher doses up to 0.5 mg/kg can cause untoward nervous signs.

Domperidone

Domperidone, a newer dopaminergic antagonist does not cross the blood–brain barrier and at a dose rate of 0.2 mg/kg IV has been shown to block dopaminergic receptors and prevent postoperative ileus induced experimentally. It has potential for use in clinical cases.

Cisapride

- Cisapride is a substituted benzamide with gastrointestinal prokinetic properties.
- The mode of action is believed to be enhancement of release of acetylcholine from intramural interneurons leading to increased calcium flux.
- Tablets suitable for the treatment of motility disorders in humans can be administered either orally or per rectum in horses.

Erythromicin

- Erythromicin stimulates enteric motilin receptors.
- Its action is independent of antimicrobial activity.

Lidocaine

- Lidocaine has analgesic, anti-inflammatory and pro-kinetic properties.
- It is commonly used in the management of post-operative ileus.
- An initial intravenous bolus (1.3 mg/kg administered slowly over 5 minutes) is followed by a continuous rate infusion (0.05 mg/kg/min) (diluted in a crystalloid solution).
- Signs of toxicity include muscle fasciculations, ataxia and possible seizures (most likely if the initial bolus is administered too quickly).

Fluids

While universally employed to support horses with severe intestinal obstructions requiring surgery and/or in horses receiving intensive medical therapy, the value of this simple medical therapy for colic in a field situation has not been fully appreciated. The type of fluid and rate of administration will change from the initial therapy which is designed to replace the deficits, to maintenance therapy which is designed to keep pace with ongoing requirements. A third category of fluid therapy is overhydration which is most commonly attempted in horses with large colon impaction, however the efficacy of this therapy is uncertain.

2.4 COMMON TYPES OF COLIC

Spasmodic colic

Aetiology and pathogenesis

- Spasmodic colic is the most common form of colic in horses and accounts for some 40% of all cases.
- It is a functional intestinal disorder that is rarely associated with histological changes of the mucosa. It is attributed to an increase in vagal tone causing increased peristalsis and a propensity to spasm.
- Bouts of spasmodic colic can be precipitated by weather changes, overexertion, chilling, feeding errors, feeding technique, and feed quality and quantity. An individual predisposition to this type of colic is not uncommon.

Clinical signs

- The disease is characterized by severe paroxysmal attacks of colic lasting from 5 to 10 minutes and separated by pain-free intervals during which the horse's appearance and behaviour are normal.
- Initially systemic effects are very mild.
- The respiratory and pulse rates increase little during bouts of pain and return to normal when the horse is quiet.
- Often the hyperperistaltic activity is audible at some distance from the horse and frequently has a metallic sound.
- Faeces may be passed frequently and in small amounts and may have a soft to semi-liquid consistency.
- Rectal findings are seldom remarkable, but one or more spastically constricted loops of small intestine may be palpable which may then relax. In other cases small amounts of gas may be evident in the jejunum or caecum.
- Barring possible complications such as volvulus or intussusception, the colic symptoms will pass in 4 to 6 hours.

Treatment

- The administration of a spasmolytic/analgesic drug combination such as hyoscine/dipyrone will quickly abolish the spasm and

Box 2.3 **Anatomical sites and common causes of impactions**

Stomach

Corn
Shavings bedding in neonates
Food – secondary impactions in horses with liver failure

Duodenum/proximal jejunum

Coarse ingesta, or secondary to stricture caused by idiopathic focal eosinophilic enteritis

Ileum

Ingesta – primary impaction or secondary to ileal hypertrophy, tapeworm infestation, and grass sickness

Caecum

Coarse roughage
Often underlying motility problem

Large colon

Sand
Enteroliths
Dehydration (e.g. ileal impaction or secondary to anterior enteritis)
Grass sickness

Pelvic flexure

Coarse roughage
Can be concurrent with right dorsal colon impaction
Sand
Grass sickness

Transverse colon

Enteroliths
Foreign bodies
Sand

Small colon

Enteroliths
Foreign bodies
Meconium in foals
Faeces – may be associated with *Salmonella* infection

Rectum

Cauda equina neuritis
Perirectal abscesses

thereby relieve the pain. It is therefore both diagnostic and therapeutic.
- The treatment may be repeated after several hours if necessary, but most cases show no recurrence of colic when the effects of the initial injection wear off.

Differential diagnosis

- Attacks of colic associated with physical obstructions of intestine also commence with loud hyperperistalsis (resistance peristalsis) but do not respond promptly to spasmolytic therapy.
- Colic attributed to reduced blood flow due to compromise of the cranial mesenteric artery caused by migrating strongyle larvae may have a similar clinical presentation.

Impaction colics

Impactions are among the more common medical colics encountered by equine practitioners (Box 2.3). Diagnosis is seldom difficult, and impaction colics, particularly those of the large colon, can usually be resolved with conventional therapy.

The primary objectives of therapy are:

- to maintain hydration.
- to provide gastrointestinal lubrication.
- to stimulate gastrointestinal motility.
- to control pain.

Although the majority of impactions are treated successfully without recourse to surgical interference, prolonged obduration and complete obstipation (e.g. due to enteroliths) must be recognized and dealt with before bowel necrosis and rupture occur.

In the majority of cases of impaction the obstructing material comprises ingesta of a drier than normal consistency, but extraneous materials such as sand or foreign objects, like nylon hay nets, may be the cause.

Prevention

Optimal management practices aimed at preventing impaction colic include:

- avoiding excessively fibrous feeds (especially horses which eat straw bedding).
- avoiding abrupt change to diet, e.g. sudden change to high fibre diet because of box rest imposed by lameness or injury.
- providing adequate water supply.
- maintaining adequate parasite control and dental care.
- removing foreign objects such as baling twine from food sources.

Clinical signs

- Rapid consumption of excessive amounts of corn can result in gastric and duodenal impactions.
- Pain is usually moderate and frequently intermittent.
- Signs include pawing, lying down and flank watching.
- Horses with an impacted pelvic flexure located within the pelvis adopt a stance for urination frequently and appear to find relief by lying in dorsal recumbency.
- The pulse may be slightly raised (40–50 per minute) with increases related to hypovolaemia and pain.
- The packed cell volume (PCV) and plasma protein are often normal but can be slightly increased if the impaction involves the small intestine or if it has been present for more than 24 hours in the large colon or caecum.
- Auscultation of the abdomen usually reveals a decrease in borborygmi, but in horses with large colon impactions, bouts of pain are often concurrent with bowel activity.

Diagnosis

Rectal examination is the most important diagnostic procedure enabling firm masses which can be gently indentated with the fingers to be identified within the bowel lumen.

- Impacted ileum may be recognized as a firm tubular structure the diameter of one's forearm to the left of the caecum early in the course of the condition before it is masked by numerous loops of distended jejunum.
- Primary impaction of the pelvic flexure is characterized by a firm evenly-filled viscus which is often located on the pelvic floor.
- The firmness and extent of the impaction should be noted so that the response to treatment can be evaluated at subsequent visits.
- The thickness of the wall of the colon should also be checked. Oedema indicates a degree of vascular occlusion usually due to torsion.

- Transverse colon impactions may be out of the examiner's reach and may be large, thus requiring a more prolonged course of treatment with a guarded prognosis.
- Sand impactions may be suspected whenever faeces retrieved during rectal examination contain sand or grit. If water is added to faecal material in a rectal sleeve and massaged, the sand will settle into the fingers of the glove.

Secondary impaction of the colon is not uncommon in conditions causing dehydration such as ileal impaction, grass sickness and anterior enteritis. This can be recognized by the fact that the colon is contracted down on the firm ingesta, and the constrictions and sacculations of the ventral part are very distinct.

Caecal impactions are particularly difficult to assess since digesta may bypass the caecum while the impaction is present:

- Physical signs are similar to large colon impaction except that pain may be continuous and severe when distension is marked.
- The peritoneal fluid may demonstrate increased protein and cellular changes indicating compromised bowel.
- Impaction of the overhanging part of the base of the caecum can be palpated per rectum if the horse is not too large.

Small colon impactions present as a long tube evenly filled with faeces extending forward from the rectum. Generalized tympany of the large colon and caecum is a feature of such impactions.

In neonates meconium retention can be felt *per rectum* with a finger of the large colon and caecum at the pelvic inlet.

Treatment

Medical management of **colonic** impactions is relatively simple and involves the use of nasogastric intubation for lubrication and hydration, parenteral fluid therapy and the control of pain.

1. Lubrication of the intestinal tract is achieved using mineral or vegetable oil (4 to 8 L) either on its own or mixed with electrolyte supplements.
   - This should be administered via a nasogastric tube by gravity or by carefully using a stomach pump.
   - If necessary this is repeated at 12 and 24 hours.
2. Since softening of the obstruction requires gut motility to mix the oil with the mass of ingesta, it is important to use an analgesic which does not depress colonic contractions. Flunixin meglumine 0.5–1.0 mg/kg at 8- to 12-hour intervals is the analgesic of choice.
3. While not necessary in horses with mild impactions of short duration, the intravenous administration of balanced electrolyte solution may be of value in horses with large or firm impactions. The goal of IV fluid administration is to increase plasma volume and decrease osmotic pressure of plasma thereby allowing fluid to move into the extracellular space with subsequent secretion into the bowel lumen. However the efficacy of this treatment is uncertain.

   The patient usually tolerates fluid administration at 2 to 3 times maintenance with rates of as much as 5 L per hour. The average time for resolution is approximately 2 days with total fluid volumes ranging from 54 to 350 L per treatment. Total plasma protein and plasma electrolytes should be monitored.
4. More recently, administration of large volumes of water through a nasogastric tube (as much as 10 L q 30 minutes) have been used to successfully treat large colon impactions. The average time for resolution is 2.5 days and volumes ranging from 85 to 200 L may be required. The nasogastric tube is connected to a fluid reservoir. The stomach is checked for absence of reflux before commencing administration of fluid.
5. Due to the risk of rupture, horses with **caecal** impaction should always be considered candidates for surgery and may require an ileocolostomy to prevent recurrence of the problem.
6. Horses with impactions should be bedded on shavings or paper or muzzled if on straw. They should be allowed water ad libitum, but all food should be withheld until the impaction has cleared and then reintroduced slowly over 48 hours.
7. Horses with impactions generally benefit from a little gentle exercise.
8. The use of gastrointestinal stimulants such as neostigmine is not necessary, and in severe cases bowel contraction, rather than propulsion, around the impaction causes pain.
9. Excessive tympany together with increasing episodes and intensity of pain requiring frequent administration of analgesics indicate the need for surgical intervention. It should be remembered that large colon impactions may be accompanied by torsion or displacements.
10. Surgical removal of impactions is indicated whenever a definitive diagnosis confirms that the obstruction cannot be cleared medically as with enteroliths or faecoliths or when sustained medical treatment has become unsuccessful and the horse becomes subject to bowel necrosis identified by changes in peritoneal fluid. This stage is reached much sooner in the case of caecal impaction than with large colon impaction. Early surgical intervention is also required in extensive small colon impactions due to the severe tympany and a rapid progression towards necrosis.
11. Meconium retention in neonates rarely requires surgical interference. Enemas are a mainstay of treatment for small colon meconium impactions. Warm water liquid detergent enemas (half a teaspoonful of liquid detergent in 500 mL of water) are gentle to the mucosa and effective. Acetylcysteine retention enemas are now widely used and have proved highly successful.

    A 4% solution of acetylcysteine, pH 7.6, is made by adding 20 g of baking soda and 8 g of acetylcysteine to 200 mL of water. A 30-French Foley catheter with a 30-mL bulb is inserted approximately 2.5 to 5 cm into the rectum and the bulb slowly inflated to occlude the rectum. One hundred to 200 mL of the solution is administered by gravity flow and retained by clamping the catheter for 30 to 45 minutes, at which point the cuff is deflated and the catheter removed. The enema may need to be repeated once or twice at intervals of 12 hours in about 40% of cases.
12. Ileal impaction is the most common cause of simple obstruction of the small intestine.
    - It may be primary or secondary to thickening of the wall of the distal ileum or the caecal mucosa at the ileocaecal orifice associated with a heavy tapeworm burden. It is also common in grass sickness cases.
    - Although impactions involving only a short length of ileum may clear spontaneously or in response to a spasmolytic drug such as hyoscine, more extensive impactions require surgical intervention. Delay in carrying out surgery can result in postoperative ileus due to the great increase in intraluminal pressure brought about by sequestration of large volumes of fluid and gas in the jejunum.

Flatulent / tympanitic colic

Aetiology and pathogenesis

- Flatulent colic results from accumulation of excessive volumes of gas in the gastrointestinal tract. The overdistension of the viscera stimulates pain and pressure receptors causing mild to severe colic.

- The condition is usually due to increases in fermentation or ineffectual gastrointestinal motility or may be secondary to partial luminal obstruction.
- Distension inhibits vagal motility while fermentation continues.
- Since the release of gas is normally dependent on escape through the gastrointestinal tract, gas accumulates in the stomach, caecum and large colon.
- The source of the gas includes endogenous production of volatile fatty acids and other byproducts of fermentation of feedstuffs.

The causes of flatulent colic include:

- interruptions in gastrointestinal motility from stress, excitement or pain.
- impactions.
- displacements.
- late pregnancy.
- ileus secondary to anaesthesia, surgical manipulation of intestines, vascular compromise (thromboembolic colic) and liver disease.

Contributory factors related to management include:

- feeding highly fermentable substrate (grain overload).
- feeding horses when exhausted or overheated.
- cold water engorgement and poor feed quality.

Behaviour-associated contributory factors include:

- inadequate mastication.
- rapid feed engorgement.

Clinical signs

- The clinical signs of flatulent colic depend on the rate of gas accumulation and the part of the gastrointestinal tract involved. They vary from the horse merely being off feed to signs of acute distress which accompanies gastric distension.
- The temperature, pulse and respiratory rate will usually be elevated in proportion to the clinical signs.
- The mucous membranes are pale, and the respirations become more rapid and shallow as the distended viscera occupy a greater proportion of the abdominal cavity and place pressure on the diaphragm.
- Because the stomach is situated immediately adjacent to the diaphragm, shallow respirations occur more consistently with gastric tympany.
- Rectal examination reveals gas-filled sections of intestine. Although the stomach itself is infrequently felt, the spleen may be felt extending more caudally than usual. The caecum is fixed at its base and therefore can always be located in the upper right caudal quadrant of the abdomen. Distended large colon can readily be identified from its location, size and the presence of longitudinal bands (absent at the pelvic flexure).
- The shape of the abdomen changes in response to the intra-abdominal distension. Large colon tympany tends to result in bilateral abdominal distension, while caecal tympany often flattens and elevates the region of the right paralumbar fossa.
- Auscultation of the abdomen is valuable in determining the presence or absence of intestinal sounds.
- Percussion will aid in identifying more accurately the region of gaseous distension.
- The rate and location of gas accumulation tend to govern the intensity of pain. Gastric distension results in severe signs of pain, while with distension of the caecum and colon the pain tends to be dull and intermittent.

Therapy

The primary objective of therapy is to evacuate the gases from the region of distension and to prevent its formation.

1. Stomach tubing is always indicated as a diagnostic procedure in colic, and whenever gastric tympany is present, the elimination of gases through the tube will provide immediate, although possibly temporary, relief of clinical signs.
2. In very severe cases trocharization of the caecum can be employed to bring about a similar, rapid decompression. However, in the majority of cases the tympany is not immediately life-threatening, and these horses frequently benefit from 15 to 20 minutes of trotting on a lunge rein.
3. Medical treatment should include supportive therapy to relieve pain, (α2 agonist ± butorphanol and intravenous fluids).
   - Mineral oil is often used to coat fermentable substrate and to lubricate the food material within the gastrointestinal tract for easier passage.
   - Metronidazole and/or neomycin by mouth, twice daily, can also be of value.
4. Decompression by trocharization should be used only when the sublumbar fossae are very distended, to prevent rupture. It should be taken into consideration that the character of the abdominal fluid will be changed shortly after trocharization so that the value of cytological examination of peritoneal fluid obtained by abdominal paracentesis for diagnosis is decreased. Trocharization of the caecum is carried out via the right sublumbar fossa. The site is clipped and surgically prepared. A local anaesthetic skin bleb and a small stab incision with a number 11 scalpel blade facilitate the insertion of a 10-cm, 14- or 16-gauge needle through the abdominal wall into the caecum. A rush of gas from the needle will occur immediately, and the needle should be left in place until the flow ceases. A broad spectrum antibiotic (10 to 20 mL) is injected through the needle as it is withdrawn as an aid to the prevention of septic peritonitis and localized infection in the abdominal wall. Systemically administered antibiotics may further suppress diffuse septic peritonitis.

Prognosis

The prognosis for uncomplicated cases of flatulent colic is usually good. However, prolonged or recurrent cases require further investigation for an underlying disorder affecting gastrointestinal motility or lumen patency.

2.5 GRASS SICKNESS

See Chapter 3.

2.6 ANTERIOR ENTERITIS (PROXIMAL ENTERITIS, GASTRODUODENAL JEJUNITIS)

The term anterior enteritis is used to describe an acute clinical syndrome characterized by abdominal pain, ileus, gastric distension and hypovolaemic shock often complicated by endotoxic shock.

Clinical signs and diagnosis

- The disease can be diagnosed for certain only at surgery or autopsy, but in most cases the signs are said to be specific enough to allow a clinical diagnosis to be made.

- Horses present with moderate to severe colic frequently accompanied by marked sweating.
- Depression may replace the colic at 12–24 hours, although most horses continue to show intermittent pain.
- IIeus is a consistent physical finding.
- Gastric distension occurs secondary to the ileus. Hyper secretion and decreased absorption of fluid occur with a few hours of the onset of the condition and in extreme cases may result in stomach contents being expelled via the nose.
- The volume of gastroenteric fluid obtained on nasogastric intubation varies from 5 to 30 L. The fluid is usually foetid, alkaline and brown-tinged. It may be positive for occult blood.
- Most horses have fever and an accompanying leucocytosis.
- Varying degrees of dehydration and endotoxic shock are seen. Heart rate (70–100 beats per minute) and respiratory rate (30–60 breaths per minute) are elevated, and capillary refill time is extended to 4 to 6 seconds. Mucous membranes are congested and injected and in cases presenting in severe shock, are cyanotic.
- Usually there are no intestinal borborygmi evident on abdominal auscultation.
- Rectal examination reveals a relatively empty intestinal tract except for a thickened or slightly distended segment of the distal duodenum as it courses over the base of the caecum. Distended loops of proximal jejunum may be palpated to the upper right quadrant. The large colon is reduced in size and contracted down onto the contents which are dry and firm secondary to the breakdown of intestinal fluid transport.

Laboratory data

- The packed cell volume and total plasma protein values reflect severe dehydration and are often in excess of 0.55 L/L and 85 g/L respectively.
- Serum electrolytes are usually normal. However, mild to moderate hypochloraemia is present in 50% of cases.
- There may be acidosis or alkalosis depending on the amount of gastric reflux and the severity of shock.
- Abdominocentesis findings tend to be unremarkable unless mural necrosis is advanced. The peritoneal fluid is usually straw coloured and either clear or slightly clouded. Microscopically some red blood cells are present with normal numbers of leucocytes. The total protein in the fluid is elevated to more than 30 g/L. Although sometimes, with anterior enteritis, the peritoneal fluid is red-tinged, the absence of blood staining of the fluid can be of value in differentiating this syndrome from strangulation obstruction of the small intestine.

Gross intestinal lesions

- At celiotomy or autopsy, varying lengths of the proximal small intestine are found to be slightly distended. Rarely is its diameter greater than 7 cm, and the intraluminal pressure is usually less than 10 cm H_2O.
- The duodenum and proximal jejunum have red and yellow streaks. The serosal surface is mottled with petechial and ecchymotic haemorrhages.
- The wall of the affected gut is slightly thickened but is not as oedematous as infarcted or obstructed intestine.
- In some cases the lesions may progress to focal necrosis of the intestinal wall.
- Haemorrhages may also be seen in the mesoduodenum and mesojejunum together with subserosal oedema at the junction of the mesentery and intestine.
- The rest of the small intestine is contracted.
- No external signs are visible on the stomach.

Histopathology

- The histological lesions are confined to the duodenum and proximal jejunum in most cases but can extend from stomach to colon.
- There is submucosal and mucosal oedema and hyperaemia with sloughing of the villous epithelium.
- In severe lesions there is neutrophilic infiltration and degeneration in the submucosa and haemorrhages in the muscularis and submucosa.
- The severity of the lesions varies between cases.
- The disease seen in Germany also has an associated haemorrhagic gastritis which is not commonly seen in cases in the UK or the USA.

Aetiology

- The aetiology of anterior enteritis is still uncertain.
- *Clostridium* spp. and *Salmonella* spp. have been suggested as possible causes. The histological lesion is similar to clostridial enteritis seen in young pigs but is unlike the recognized clostridial enteritis in the horse which usually produces lesions in the caecum and colon. *Clostridium perfringens* has been cultured from over 70% of affected horses examined by the author. Isolation of *Clostridium difficile* has also been reported.

Treatment

In the absence of an identified cause for this condition, the management of anterior enteritis has been directed primarily at supportive therapy rather than at eliminating a specific aetiological agent. Various methods of treatment have been reported, both conservative and surgical.

The major *medical* therapeutic objectives are:

1. Maintaining adequate decompression of the stomach and intestine by nasogastric intubation as needed.
2. Controlling the effects of endotoxaemia.
3. Replenishing lost fluids and electrolytes.
4. Encouraging resumption of intestinal motility.

The use of antibiotics has been a matter of debate.

Maintenance of fluid and electrolyte balance in many cases of anterior enteritis requires a major effort and financial commitment; the need for infusion of up to 70–80 L per day is not particularly unusual.

The paralytic ileus associated with this syndrome is the basis of the clinical problem and the most difficult thing to deal with therapeutically. Cisapride, which has prokinetic activity in relation to the intestines as well as the stomach, has not been as clinically effective as might have been expected from experimental studies.

Surgery is an option that some find unacceptable because the stress involved might make the prognosis worse. In the experience of some authors who favour medical therapy a high percentage of horses with anterior enteritis undergoing general anaesthesia and ventral midline celiotomy succumbed to circulatory shock and/or laminitis. However, if laparotomy and decompression of the intestine are completed rapidly and if adequate blood pressure is maintained under anaesthesia, there is no greater risk of postoperative shock or laminitis.

Following the isolation of *Clostridium perfringens* from one of the first horses referred to the author, the following treatment regimen was developed and employed for all subsequent cases:

- Horses suspected of having anterior enteritis are anaesthetized, and via a short midline celiotomy the characteristic serosal discoloration of the proximal small intestine is identified and other conditions eliminated by systematic examination of the alimentary tract.

- The intestine is decompressed by gently massaging its contents into the caecum.
- When large volumes of fluid contents are present, the caecum is evacuated via a small enterotomy incision near its apex.
- Metronidazole is administered IV as soon as the diagnosis is confirmed.
- Hartmann's solution is administered throughout the operation.
- Every effort is made to keep the procedure as short as possible. The average operation time is 30–40 minutes.
- Nasogastric intubation and gastric reflux are performed on recovery from anaesthesia and at 3-hourly intervals until no fluid is obtained on two successive occasions.
- Low dose flunixin (0.25 mg/kg three times daily) and metronidazole therapy is carried out for 2 to 3 days.
- Most cases show a steady fall in heart rate and packed cell volume which takes 24–72 hours to return to normal. Very few horses have any significant gastric reflux and this ceases 12 to 24 hours after surgery. Intravenous fluid therapy is continued during this period. Horses which have no postoperative gastric reflux and show a progressive return of normal gut activity are allowed small amounts of water to drink at hourly intervals until normal hydration is achieved and then slowly returned to normal rations.

The recovery rate with this treatment regimen has been extremely good (95%).

The advantages of surgery in the treatment of this condition can be summarized as follows:

- It overcomes the difficulty in differentiating anterior enteritis from a physical obstruction and from acute grass sickness.
- By resorting to surgery without delay, complete decompression of the distended proximal small intestine can be achieved, thus encouraging the return of normal circulation and peristalsis.
- It also ensures that should the horse in fact have a physical obstruction, its correction is not delayed while conservative therapy of suspected anterior enteritis is instituted and its effects evaluated.
- As a result of the rapid return of normal gut activity and cardiovascular status, the horses require very little postoperative care and are discharged within 8 days of admission. This helps to keep down the cost to the client.
- The prolonged period of inactivity necessary after surgery is a disadvantage. However, when horses with anterior enteritis have been treated medically it is advisable to allow adequate time for them to recover fully because of the possibility of recurrence.

2.7 APPROACH TO DIAGNOSIS IN COLIC CASES

The majority of cases of colic, probably in excess of 90%, respond to medical therapy.

The primary aim of the initial examination is to distinguish horses with a mild or uncomplicated disease process from those with a potentially life-threatening disorder requiring further monitoring, surgery or intensive care. The earlier these serious disorders are recognized and specific therapy instituted the better is the prognosis for recovery.

In cases of a serious nature, any attempt at a diagnosis is often made under stress and with less than favourable conditions, but in mild cases there will be adequate time to examine and initiate therapy without placing the patient at risk. However, even when a horse has mild colic, the veterinarian should complete a thorough examination and not be caught in a trap of letting the response to treatment guide the decision-making.

The ideal goal of the systematic examination is an aetiological diagnosis, and it should include the following:

- Signalment.
- Observation.
- Clinical examinations.
- Laboratory investigations.

The cause of the colic and the length of time it has been in existence will largely determine whether a diagnosis can be made at the initial examination or requires the procedure to be repeated at 2-hourly intervals. It is important that the results are carefully documented. By comparing findings recorded at different times, the veterinarian is able to discern important trends in the course of the illness. This is particularly important if a subsequent examination is carried out by another veterinarian in the practice. A printed colic sheet listing the various procedures and providing spaces for recording the findings at each examination is of considerable value.

Signalment

1. The age of the horse should signal the veterinarian to consider specific conditions – for example, meconium impaction in a foal 1 to 2 days old; ileal intussusception in yearlings or strangulating lipomas in horses older than 15 years.
2. Some conditions have a sex predisposition. Inguinal herniation affects stallions while large colon volvulus and uterine torsion typically affect mares.
3. The breed of horse may occasionally suggest certain disorders. Large-bodied horses appear to have a high prevalence of left dorsal displacement of the large colon and Standarbreds have a predisposition to inguinal hernia.

History

1. The most important factor of the history is the time which has elapsed since the onset of clinical signs. This may be known precisely but often can only be estimated, for example, in the case of horses which are found in colic at the owner's first inspection of the day, having been seen normal for the last time the previous evening. It is essential that a reasonably accurate assessment of the likely duration is made in order that the significance of the clinical findings can be evaluated.
2. The history should include the general history relating to husbandry and management and the recent history and management and more specific details related to the present colic episode (Box 2.4).
3. The general history may not help to identify the specific cause of the colic episode under investigation, but information such as a history of inadequate worming may be significant. Recent changes in housing, bedding material, and quantitative or qualitative changes in feed may be directly involved, e.g. horses which are brought in from pasture and bedded on straw may eat the bedding in preference to hay and develop large colon impaction as a consequence.
4. In foals with heavy ascarid burdens, treatment with anthelmintics can lead to obstruction of the small intestine by dead worms. Caecocaecal intussusception following administration of an anthelmintic is another example where the recent worming history may be significant.
5. A history of previous illness may be important, e.g. in a horse with chronic colic that had an episode of strangles several weeks earlier may suggest a mesenteric abscess.

Questions relating to the present colic episode should specifically address the following:

Box 2.4 History of the colic case

General history

1. When last seen normal.
2. Housed or at grass.
3. Feed.
4. Use.
5. Daily routine.
6. Parasite control.
7. Past medical history.

Recent history

1. When last fed.
2. Consumption of feed and water.
3. Any recent changes in feeding, bedding, housing, exercise or routine.
4. Recent worming.
5. Pregnancy.
6. Recent exercise.

Details relating to present colic episode

1. Degree and any changes in pain and how manifested.
2. When last defecated.
3. Sweating.
4. Any treatment given and response.
5. Previous episodes of colic or abdominal surgery.

Box 2.5 Clinical examination

1. Cardiovascular system

(a) Rate and quality of pulse.
(b) Appearance of mucous membranes.

2. Examination of abdomen

(a) Abdominal distension.
(b) Auscultation and palpation.
(c) Rectal examination.
(d) Abdominal paracentesis.
(e) Nasogastric intubation.
(f) Abdominal ultrasonography.

3. State of peripheral perfusion and hydration

(a) Capillary refill time.
(b) Packed cell volume.
(c) Total protein.

- How severe has the pain been and has it altered during the time it has been observed?
- When did the horse last defecate and what was the character of the faeces?
- Has the horse shown specific behaviour such as playing with water or adopted any unusual postures?
- Could it have gained accidental access to too much highly fermentable feed?

Clinical examination

Observation

- Whilst the history is being taken, the horse may be inspected in its stall and its behaviour observed.
- The nature and the degree of colicky signs currently being shown are noted.
- Skin abrasions about the eyes and over the tuber coxae are indicative of rolling and other violent behaviour prompted by severe pain.
- Marks on the wall of the box caused by the horse's kicking and excessive disturbance of the bedding, or flattening of an area of grass in horses at pasture are further evidence of severe pain consistent with a severe intestinal obstruction.
- Horses in which a strangulating obstruction has occurred only within the past 3 or 4 hours or so may still be exhibiting signs of severe pain, whereas one in which the obstruction is of much longer duration and the segment of gut involved has undergone advanced necrosis will show no or very few signs of overt pain. However, although apparently calm it will be showing signs of intense depression, e.g. standing with its head low and taking no interest in its surrounding. This 'stage of indolence' is associated with severe endotoxaemia and is often mistaken by the owner of the horse as an indication that the horse is improving.
- Such an evaluation of the circumstantial evidence and presenting signs at the time of examination allows a reasonably accurate assessment of the duration of the problem when the time of onset is unknown.
- Other signs to note are evidence of abdominal distension, sweating, or muscular tremors.
- In stallions the scrotum should be observed for unilateral enlargement, but palpation of the vaginal rings per rectum will probably be necessary later in the examination to confirm or eliminate the presence of inguinal herniation.

The veterinarian should now proceed to a systematic clinical examination of the horse to include the cardiovascular system, abdomen and state of peripheral perfusion and hydration (Box 2.5). If the colic is so severe that an orderly examination is impossible without risk to the horse or veterinarian, heavy sedation, preferably with xylazine, is necessary. In the majority of cases the pain is sufficiently mild to allow most of the examination to be performed without recourse to the use of drugs which might modify the heart rate or gut activity. However, on completion of examination of the cardiovascular system and auscultation of the abdomen, the administration of a sedative will greatly facilitate rectal examination, which is such a vital part of evaluation of a colic case, and other procedures such as nasogastric intubation and abdominal paracentesis which may be performed.

Examination of the cardiovascular system

Heart rate and pulse character

- The heart rate and character of the pulse are important criteria in assessing the colic patient.
- Pain, and the activity as a consequence of pain, have only a relatively minor effect on the heart rate which is influenced much more by haemoconcentration and diminished venous return, and by toxins absorbed from the intestine.
- There is a close relationship between the pulse and the nature and duration of the colic.
- Infarctive disease is usually accompanied by a non-fluctuating elevation in heart rate which increases progressively as toxic shock develops.
- In contrast, the heart rate will be only slightly raised after 48 hours in horses with pelvic flexure impaction.

Mucous membranes

- The colour of the mucous membranes is of value in assessing the severity of the disturbance and the prognosis.
- The oral mucous membranes, provided they are not pigmented, provide the best surface because the conjunctivae are often congested due to trauma.
- Reddening of the membranes reflects developing haemoconcentration, and at a later stage when the patient is in shock, vasodilatation adds to the reddening process.
- Patients with cyanotic mucous membranes invariably die.

Capillary refill time

- In conjunction with palpation of the extremities, this measurement provides a direct means of assessing the state of peripheral tissue perfusion while providing indirect information on the degree of hydration and vascular tone.
- The application of digital pressure on the oral mucous membrane just above the incisor teeth is a simple way of assessing capillary refill time.
- Adequate peripheral perfusion, hydration and vascular tone are indicated by a normal refill of 1–2 seconds and warm extremities.
- An increase to 3 seconds or more and cool extremities indicate inadequate peripheral perfusion and excessive vascular tone or vasoconstriction.

Examination of the abdomen

Abdominal distension

- Observation of the degree of abdominal distension is helpful in making the distinction between involvement of the stomach and small intestine on the one hand and the caecum and large intestine on the other.
- The large intestines when distended are the only viscera large enough to cause noticeable enlargement of the abdomen in the adult horse (in the foal, small intestinal distension can cause abdominal enlargement).
- The pattern of distension will vary, e.g. distension of the right paralumbar fossa accompanies caecal dilatation, while general abdominal distension is seen in horses with small colon obstructions or as the result of flatulent colic.

Auscultation and palpation

- The abdomen should be thoroughly auscultated for several minutes.
- The best sites are along the caudal edge of the rib cage from paralumbar fossa to ventral abdomen on both right and left sides.
- The sounds normally heard are fluid gurgling mixed with gas.
- In almost all horses with abdominal pain, the propulsive sounds will be reduced.
- In cases of severe intestinal disease such as strangulation, all sounds will be absent within a very few hours of the obstruction occurring.
- Sounds will be reduced or even absent after the administration of drugs such as xylazine or detomidine.
- Increased sounds are heard in horses with spasmodic colic and during resolution of ileus in tympanic or simple cases of colic.
- The primary importance of auscultation is in monitoring the progress of the disease rather than arriving at a diagnosis.
- Palpation of the abdominal wall in the adult horse has little value other than in identifying 'boarding' in response to generalized peritonitis due to rupture of stomach or intestine.

Rectal examination

- Rectal examination is the single most important part of the clinical examination of the horse with colic and should be carried out in all cases whenever possible but must be approached with respect for both its value and the risk involved.
- It should be carried out after reviewing the pertinent aspects of the history and the remainder of the physical examination. In this way an attempt can be made to predict what should be felt and to compare the findings with those preconceived ideas.
- Adequate restraint is essential to prevent damage to the horse or examiner. Sedation with xylazine will aid a thorough and systematic examination of the caudal 40% of the abdominal cavity which is within reach.
- Several large intestinal obstructions can be diagnosed by this means including pelvic flexure impaction, left dorsal displacement of the left colon, large colon torsion, caecal impaction and intussusception.
- Although rectal examination enables a specific diagnosis of very few obstructions of the small intestine such as inguinal herniation and ileal impaction, seldom does it provide no useful findings. Small intestine obstructions or adynamic ileus produce distended loops of bowel lying side by side. In the early stages of obstructions, careful, patient palpation over a period of several minutes may be necessary before a distended loop is recognized. Later multiple tightly distended loops pushing back into the pelvic inlet make examination difficult. The presence of distended small intestine almost always indicates a problem requiring surgical correction. Early identification greatly enhances the chances of recovery.
- The normal structures that are palpable include, in the left dorsal quadrant, the spleen, the caudal pole of the left kidney and, linking the two, the nephrosplenic ligament. Moving to the right and extending forward below the spine, the root of the mesentery can be palpated. In the right dorsal quadrant the base of the caecum is identified by the caudal and medial bands which normally are relaxed and run downwards, forwards and medially. Moving ventral to the pelvic brim the pelvic flexure of the large colon can usually be detected and, extending cranially from it, the large diameter left ventral colon with its clearly recognizable longitudinal bands and the narrow, smooth left dorsal colon can be palpated. The space above this and to the left of the caecum is usually occupied by small intestine and small colon. The normal small intestine is usually not palpable unless it happens to contract when touched, but the small colon is easily recognized by the formed faecal balls it usually contains.

Abdominal paracentesis (abdominocentesis)

- Analysis of the peritoneal fluid reflects the changes that occur in the tissues and organs within the abdominal cavity and on the peritoneal surface. In colic cases it assists in determining the type of disease and the severity of the lesion.
- The simplest method is to insert a 21-gauge, 2.0-inch in needle through a prepared site on the ventral midline of the abdomen at its most dependent point. Entry of the needle into the peritoneal cavity is indicated by the flow of varying amounts of fluid which is collected in a tube with or without anticoagulant depending on the information required. In obese horses a longer needle may be required to penetrate the thick layer of retroperitoneal fat.
- The fluid obtained is evaluated by gross visual examination, total protein determination and, if necessary, microscopic evaluation.

- Normal fluid is pale yellow and clear.
- As the fluid changes with specific diseases, the fluid can become more turbid due to increases in protein, red blood cells (RBC) and white blood cells (WBC). Normal fluid will be present in horses with non-strangulating obstructions or strangulating obstructions within an hour or two of the obstruction occurring or when the infarcted bowel is not in direct contact with the peritoneal cavity as in diaphragmatic herniation.
- Abdominal paracentesis is particularly valuable in the early stages of severe colic, when serosanguineous fluid, which may require centrifugation to show the presence of red blood cells, helps to confirm the presence of infarcted bowel in advance of diagnostic rectal findings. With the availability of portable lactate analysers it is possible to measure lactate concentrations in peritoneal fluid during ambulatory assessment. A peritoneal fluid lactate concentration above 2.0 mmol/L will successfully predict the presence of a strangulating lesion approximately 4 times out of 5.
- Late in the course of strangulating obstruction, when distended loops of intestine and gastric reflux have been confirmed, paracentesis may be difficult to perform without accidental penetration of bowel and is not necessary for the decision to operate. However, if gastrointestinal rupture or very advanced gut necrosis are suspected, their confirmation by paracentesis indicates the need for immediate euthanasia.

Nasogastric intubation

- In addition to being of diagnostic value, decompression of the stomach via a nasogastric tube produces immediate alleviation of pain and reduces the risk of rupture.
- Gastric reflux of more than 2 L is considered to be significant and usually indicates a primary disorder located in the small intestine or stomach.
- However, in some large colon obstructions, e.g. left dorsal displacement of the large colon, the proximal small intestine is compressed preventing normal gastric emptying.
- Acute grass sickness and anterior enteritis are other possible causes of gastric distension.
- Normal gastric fluid has a pH of 3–6, but following intestinal obstruction, pH will be 6–8 due to the buffering effect of fluid from the small intestine.
- In horses with a history of recent access to excessive amounts of concentrated feed or those showing dyspnoea and spontaneous regurgitation of gastric contents down the nose, nasogastric intubation should be performed at the start of the examination to reduce pressure and prevent rupture.
- It is important in horses suspected of having gastric dilatation that continued efforts at decompression are made even though the initial attempt(s) at starting a siphon are not successful.

State of peripheral perfusion and hydration

Packed cell volume (PCV) and total protein

- Packed cell volume on its own is an unreliable indicator of the patient's intravascular hydration because of the wide range of normality.
- Progressively increasing PCV on repeated examinations, and values over 50%, are considered significant.
- In the absence of a means of spinning down blood samples, samples can be left to settle out.
- Since it is changes in PCV rather than a single percentage which are of assistance in evaluating colic cases, simple visual comparison of sequential samples is sufficient.
- When PCV is considered along with total protein (TP normal 65 to 75 g/L), it provides a very useful means of assessing intravascular hydration and acts as a guideline for fluid therapy. Raised levels of PCV and TP represent intravascular dehydration.

Imaging techniques

Although not routine clinical practice, there are indications for employing various imaging modalities in selected cases of equine colic.

1. *Endoscopy*: gastric ulcers.
2. *Ultrasonography*: intussusceptions, inguinal herniation, strangulating small intestine obstructions, colonic displacements, right dorsal colitis.
3. *Radiology*: intestinal obstruction in foals, diaphragmatic herniation, enteroliths and sand impaction.
4. *Laparoscopy*: Only feasible in horses with low-grade or chronic colic unassociated with intestinal distension. Conditions that can be diagnosed include intra-abdominal masses and adhesions.

Classification into categories

Although at first sight this may appear a formidable list of procedures, with experience the clinical examinations can be carried out in 15 to 20 minutes. At the initial examination it should be possible to classify the problem into one of the following three categories:

1. A benign problem requiring medical therapy, e.g. pelvic flexure impaction diagnosed by rectal palpation.
   Action: Administer the appropriate treatment and make arrangements to monitor progress.
2. A problem requiring surgical correction.
   Action: Arrange for immediate admission to a surgical facility.
3. A problem which may require surgery but for which the evidence at that point is not conclusive.
   Action:
   - Administer an analgesic.
   - Ensure the horse is in a 'safe' environment.
   - Arrange to re-examine it in 2 hours.

Note. Because of its ability to mask several of the clinical signs on which the decision to operate is based, flunixin should not be used at this stage.

At the second visit the same examination procedures are repeated and the results compared with those obtained 2 hours earlier.

The horse may be found:

- to have improved.
- to have developed signs which indicate the need for surgery.
- to still have inconclusive findings.

If results are still inconclusive the arrangement may be made to re-examine the horse in 2 hours, but if there is the least cause for concern, consideration should be given to discussing the problem on the telephone with a recognised specialist at a referral centre, when the decision may be taken to refer the horse for further investigation and, if necessary, surgery.

2.8 THE DECISION FOR SURGERY

Surgical intervention is indicated:

1. When the exact cause of the colic can be diagnosed and the obstructing lesion requires surgery for its correction.

Box 2.6 **Recurrent/chronic colic – possible causes**

1. Partial occlusion of the gut lumen.

A. Small intestine.

Muscular hypertrophy of the ileum.
Hypertrophy of caecal mucosa at ileocaecal junction.
Intramural neoplasia.
Ileocaecal intussusception.
Adhesions.
Mesenteric abscess.

B. Large intestine.

Caecocaecal or caecocolic intussusception.
Recurrent displacements or mild torsions of the large colon.
Enteroliths.
Neoplasia.

2. Gastroduodenal ulceration in foals and gastric ulceration in adults (see Chapter 3).

3. Cranial mesenteric arteritis.

4. Miscellaneous causes.

Oestrus.
Granulosa cell tumour of ovary.
Crib biting.
Renal calculi.
Chronic inflammatory bowel disease.
Chronic grass sickness.

2. When there is no exact diagnosis but there is sufficient evidence to indicate that surgery is the only means of saving the horse's life.
3. When horses with recurrent colic over a period of days or weeks are suspected of having partial obstruction due to adhesions, neoplasia etc. (Box 2.6).

Most cases fall into the second category.

Despite tremendous advances in anaesthetic and surgical techniques and in postoperative management, the survival rate in horses undergoing colic surgery is still only around 70%. Most horses which die as a direct result of colic succumb to acute circulatory failure (shock) secondary to intestinal ischaemia/infarction. Early recognition holds the key to incidence of survival so that surgical intervention occurs before the horse's condition begins to deteriorate.

In deciding the need for surgery, there is no single criterion that can be relied upon. All the information that the circumstances permit must be gathered, and the facts must be weighed against each other so that a prognosis as well as a preliminary diagnosis can be rendered. The criteria most useful in determining whether a horse suffering from colic requires surgery are set out below in order of importance:

1. Degree of pain.
   - In some cases of strangulating obstruction, particularly 360° torsion of the large colon, the pain from the outset is so severe and unresponsive to analgesics that this alone is sufficient indication for resorting to surgery as soon as possible.
   - Large colon displacements, ileal impaction and partial obstructions such as ileal intussusceptions are usually associated with moderately severe intermittent pain.
2. Rectal findings.
   - Positive rectal findings of obstructions such as large colon torsion or displacement, caecocaecal intussusception, enterolith obstruction of small colon, ileal impaction and ileocaecal intussusception are indications for immediate surgery regardless of the other clinical findings.
   - In horses with other types of small intestinal obstruction, distended, taut loops of intestine are highly significant. It may be 8–10 hours before multiple loops are palpable in the caudal abdomen. Prior to this, careful, patient palpation over a period of several minutes may be necessary before a distended loop is recognized. Early identification greatly enhances the chances of recovery.
3. Heart rate and pulse quality.
   - Infarctive disease is usually accompanied by a non-fluctuating elevation in heart rate which increases progressively as toxic shock develops.
   - A similar, but slower, increase occurs in horses with simple obstruction of the small intestine.
   - In general, a pulse rate which has risen progressively to 60–70 per minute 6 hours after the onset of colic gives rise for concern, particularly if it remains high during quiet interludes and in the face of adequate analgesia.
4. Cardiovascular deterioration.
   - Progressive deterioration evidenced by a PCV of >55, congestion of mucous membranes despite fluid therapy.
5. Absence of gut sounds.
6. Nasogastric intubation.
   - Gastric reflux in excess of 2–3 L is considered significant.
   - If primary gastric dilation can be ruled out, such reflux is indicative of small intestinal obstruction located anywhere between pylorus and ileocaecal junction, or ileus due to peritonitis, grass sickness or anterior enteritis. There is usually an accompanying progressive reduction in intestinal motility.
7. Abdominal paracentesis.
   - Changes in peritoneal fluid, viz an increase in RBC, WBC and protein, indicate morphological changes of the bowel.
   - Sanguinous fluid is usually associated with infarction and can indicate the need for surgery, although it may sometimes occur in cases of anterior enteritis.
   - Reddish brown fluid containing a large number of WBC as well as RBC indicates more advanced changes and consequently a poorer prognosis.

Changes seen in the peritoneal fluid depend on a number of factors including the time which has elapsed since the obstruction occurred, the size and length of gut involved and whether or not it is in direct contact with the peritoneal cavity. From the point of view of the attending veterinary surgeon, referral of horses with colic of unknown cause should not be delayed until changes have occurred in the peritoneal fluid.

8. Abdominal distension.
   - Progressive abdominal distension which is becoming life threatening.
9. Abdominal ultrasound findings.

These criteria are only guidelines. Such is the great variety in the type of obstruction, the part and length of gut involved, the degree of obstruction etc., it is inevitable that some cases will present difficulties in diagnosis. Where evidence for and against surgery is evenly balanced, intuition based on clinical experience is often the deciding factor.

If the horse is to be referred to a surgical centre, it is preferable that this is done when the referring veterinary surgeon is less than certain of the need for surgery rather than wait until he or she has no doubt, under which circumstances the additional delay in arranging admission and transporting the horse may make the difference between success and failure. It can be useful to contact staff at the surgical

centre to which they refer cases to discuss problem cases. The advice given may be to continue treatment at the premises or to refer the horse for further investigation and, if necessary, surgery. A significant number of these horses will recover without surgery and will be discharged within a day or two. Owners are appraised of the difficulties in identifying the cases requiring surgery and the advantages of hospitalization for further evaluation. Rarely if ever do they resent having transported the horse and are often delighted not to have to meet the considerable cost of surgery. It is important that horses in which the primary problem has progressed to severe irreversible cardiovascular compromise or gastric rupture are euthanased immediately and not subjected to transport.

2.9 PREPARATION OF THE HORSE PRIOR TO TRANSPORT TO A SURGICAL CENTRE

If a horse is to be referred to a surgical centre, and this involves a journey of any significant length, the following measures should be taken.

1. Decompress the stomach and, if necessary, leave the nasogastric tube in place and tape it to the head collar.
2. Administer an analgesic. If it is clear that surgery is necessary and a strangulation obstruction is suspected, flunixin is ideal because in addition to relieving the pain it counters the effects of endotoxaemia.
3. If the horse is in shock, plasma expanders may be given to improve the circulating blood volume. Crystalloids may be administered while awaiting the arrival of a horsebox, but departure to the surgical centre should not be delayed for the purpose of administering the large volumes necessary to make any significant impression on the PCV. On arrival at the surgical centre a rapid improvement in the cardiovascular status of severely shocked horses can be achieved by the administration of 2 L of hypertonic saline solution. This is not an option prior to referral because a large volume of isotonic saline solution is necessary within 2 hours of the hypertonic saline solution being given. The synthetic colloid hydroxymethyl starch (hetastarch) is a commonly employed alternative to hypertonic saline solution. Side-effects, which are dose dependent, include effects on coagulation. Hetastarch should not be used in conjunction with metronidazole.
4. Rug the horse up and bandage its limbs.
5. Provide a detailed report of the treatment given to the horse prior to referral.
6. Provide the owner with accurate directions how to get to the hospital.
7. Owners should be strongly discouraged from travelling in the back of the horsebox or in the trailer with the horse.

2.10 THE SURGICAL MANAGEMENT OF THE EQUINE COLIC CASE – GENERAL POINTS

Colic surgery is a team effort, and the chances of success are greatest when the optimum number of people and back-up facilities are available.

As stated previously, the patient in abdominal crisis is either in shock or predisposed to it and ideally should not undergo celiotomy until all its physiological values are within normal limits and the gastrointestinal tract has been decompressed. However, this is rarely a practical proposition. A balance between restoration of blood volume and complete rehydration must be found because if the whole of the calculated fluid deficit is given before anaesthesia, a substantial proportion will diffuse into the lumen of the intestine before the obstruction can be relieved. This will not only be lost from the circulation but it will also make the surgery more difficult. It is therefore advisable to administer as much balanced electrolyte solution as possible during preparation for surgery, and then to restore the remainder during the operation once the obstruction is relieved. This approach is satisfactory for horses with relatively mild deficiencies in circulating blood volume. However, acute restoration of the circulation in horses in severe shock is best achieved using hypertonic saline solution; 4 mL/kg of 7.5% saline solution given rapidly before induction of anaesthesia draws fluid into the circulation and increases cardiac contractility and output. The effect can be dramatic and lasts well into the surgical period during which isotonic saline solutions are administered as usual.

It is generally agreed that the prognosis depends to a large extent on the duration of the operation which should be performed as expeditiously as possible without sacrificing good surgical technique. Whenever possible, the ventral abdomen should be clipped beforehand to reduce to a minimum the interval between induction and the incision. This is particularly important in those horses with severe abdominal distension, where rapid decompression of the large intestine is essential if the patient is not to succumb to respiratory embarrassment caused by pressure on the diaphragm. The passage of a nasogastric tube and evacuation of fluid stomach contents prior to induction reduces the risk of gastric rupture, regurgitation and aspiration of fluid.

Premedication and anaesthesia

The anaesthetist frequently has to contend with the residual effects of a variety of drugs (e.g. sedatives, narcotics and spasmolytics) which have been administered between the onset of colic and surgery. Colic patients are often quiet and depressed, but when sedation is required, the alpha-2 agents are the drugs of choice. Xylazine may be preferable to detomidine as it is shorter acting, but small doses of detomidine (5–10 μg/kg) may also be used.

Induction and maintenance

The objective should be to achieve:

- smooth induction so that the horse sinks quietly to the ground.
- a similarly smooth recovery a relatively short time after completion of surgery.

A variety of drugs and dose rates may be used depending on the preference and experience of the anaesthetist and the state of the patient. Whichever induction technique is chosen, it is important it is one with which the anaesthetist is fully familiar, and it must be remembered that in toxic patients the doses required of any depressant drug will be considerably reduced.

The main requirements of anaesthetic maintenance are similar to any procedure except that there is much greater emphasis on fluid replacement.

- Progress should be assessed by regular monitoring of PCV and plasma protein.
- Arterial blood pressure, mucous membrane colour and capillary refill time (CRT) provide valuable information as to the efficiency of the circulation.
- Cardiac support with the inotropes dopamine or dobutamine is widely accepted as effective therapy whenever hypotension is evident.

Respiratory depression may be a problem in the horse with colic, but the relative merits of positive pressure ventilation and spontaneous respiration must be balanced for each individual concerned.

Blood gas analysis, if available, will enable the existence and degree of acidosis or alkalosis present to be determined. In the absence of means of blood gas measurement, it is reassuring that after fluid therapy is initiated and kidney function and hepatic circulation are restored, the body rapidly adjusts its acid/base deficit.

Recovery from anaesthesia

If colic surgery has been successful in resolving the problem the horse is usually remarkably calm in the recovery period.

Celiototomy (laparotomy) techniques

The ventral midline approach is used now almost universally for colic surgery.

- The incision allows easy, rapid access to the abdominal cavity with the minimum of haemorrhage.
- Great latitude of access and exposure of the viscera is possible, an important factor when we consider the frequency with which celiotomies are performed without the exact site of the obstruction being known.
- Adequate draping is necessary to protect the surgical field.
- The abdomen and limbs of the patient are covered with a large cotton or paper drape, and the area adjacent to the incision is further protected with waterproof, plastic sheeting.

The initial incision is made accurately in the midline extending cranially from the umbilicus. A 16-cm long incision is adequate for most small intestinal surgery, but a significantly longer incision is required to investigate and correct large colon problems.

In order that a lesion does not go undetected in this, the largest and most complex of the body compartments, it is imperative that a routine, systematic exploration be employed.

- Much of the search is conducted virtually at arm's length, correlating palpable findings with the surgeon's knowledge of anatomical relationships. An accurate command of both normal and pathological anatomy is a considerable advantage.
- On incising the peritoneum the colour and character of any effusions, the distribution pattern of distended bowel and any discernible discoloration of visceral or peritoneal surfaces should be noted.
- If gas- or fluid-filled bowel is ballooning out of the abdomen, some decompression of the alimentary tract will is necessary before exploration can commence. In addition to facilitating exploration, decompression reduces intra-abdominal pressure, encourages peristalsis and simplifies closure of the abdomen.
- Decompression of the caecum, which can be partially exteriorized, can be quickly achieved using a 16-gauge needle attached to a suction line.
- Removal of gas from the large intestine is frequently necessary in situ by needle suction before any attempt can be made to exteriorize it.
- Lifting distended, sometimes friable, large colon must be done very carefully to avoid rupture.
- Evacuation of its contents is carried out by placing the pelvic flexure between the horse's hind limbs, preferably on a colon tray, and performing an enterotomy in the terminal part of the left ventral colon. Dry contents are flushed from the lumen using a hose introduced up the left dorsal and ventral parts in turn via the enterotomy incision.
- Decompression of the small intestine can be achieved by stripping its contents into the caecum in the case of simple obstructions, or via the open end of transected intestine into a receptacle, in horses with strangulating obstruction requiring resection.
- When most of the small intestine is severely distended, decompression must be carried out in sections commencing distally. This means that the distal jejunum and ileum will are handled several times.

Gentle handling and ample lubrication with warm Hartmann's solution minimizes trauma and the risk of ileus. The caecum can be evacuated of the large volume of fluid it contains via an enterotomy at its apex.

- The initial gentle sweep of the abdominal cavity serves to check the sites of commonly occurring obstructions.
- Palpation is aimed at detecting firm masses, fluid distension and impaction or tympany of the large intestine. Abrupt changes in direction of intestine, tight mesenteric bands around or across intestine, or immobility of normally mobile portions of intestine indicate an area of involvement.
- If palpation fails to disclose the problem, the bowel must be exteriorized to facilitate visual examination.
- Normal small intestine can easily be exteriorized except for the proximal 1 metre and the terminal 15–20 cm.
- The apex and part of the body of the caecum can be lifted out, as can the left ventral and dorsal large colon and parts of the right dorsal and ventral colon.
- The proximal 60–70 cm of the small colon and the rectum are inaccessible.
- Location of the obstruction can be achieved quickly and with less trauma to the intestine if the manipulation is commenced at the distal empty portion of the bowel and continued proximally. The terminal reference point of the segment of bowel which appears to be involved should be located, namely the ileum for the small intestine and the pelvic flexure or proximal small colon for the large intestine.

Examination of the small intestine

- The ileum can be located by exteriorizing the caecum and turning it backwards to reveal the dorsal band which bears the ileocaecal fold. If this fold is traced downwards to the lesser curvature of the caecum, it will lead to the ileum which is instantly recognized by the ileocaecal fold on its mesenteric border.
- If it cannot be exteriorized, the ileum must be involved in the obstruction.
- Compared to the remainder of the small intestine, the ileum is involved in a wide variety of obstructive conditions to a disproportionate degree, and in these patients the cause of the obstruction will be quickly identified.
- If the ileum is not involved, the empty flaccid intestine is traced proximally until the obstruction and distended bowel proximal to it are reached.
- Each length of bowel examined is returned to the abdomen as the adjacent bowel is lifted out, and a rough check is kept of the length examined.
- Eventually, in the absence of obstruction, the duodenum, which cannot be exteriorized, is encountered.

Examination of the large colon

- Obstruction of the large intestine can usually be anticipated prior to celiotomy on the basis of the physical examination.

- Segments of large intestine are more easily differentiated on the basis of their appearance and position than are those of the small intestine.
- By taking note of the pattern and extent of the distension of the various parts, and taking into account the common causes and sites of obstruction, the location of the problem can be deduced without too much expenditure of time or energy.
- The caecum again serves as a landmark for the initial exploration. If its base and ventral longitudinal band cannot be palpated, a caecocaecal or caecocolic intussusception should be suspected.
- If the caecum can be exteriorized, the caecocolic fold is found and followed to the right ventral colon. This can then be traced cranially to the pelvic flexure, which in turn can be traced caudally to the pelvic flexure which is another easily recognizable part of the large colon. From there, the pelvic flexure can be followed back to the diaphragmatic flexure and on to the transverse colon cranial to the root of the mesentery. The transverse colon which is short in length and fixed in position constitutes an abrupt constriction of the lumen and change in direction.

Evaluation of gut viability

The surgery necessary to relieve obstruction may vary from simply incising a constricting band of tissue to resecting several metres of intestine. The decision as to the necessity for resection and the length of bowel which needs to be removed often depends on a subjective assessment of bowel function.

- Any intestine of abnormal colour, decreased motility or thickened appearance must be considered suspect.
- After correction of obstructions causing secondary vascular embarrassment, observation of the bowel for 5–10 minutes for the return of colour, motility (spontaneous or evoked) and pulsation of vessels will aid in deciding for or against resection.
- The junction between viable and compromised bowel may be difficult to discern especially after displacement has been corrected and a significant improvement in the appearance of the bowel has taken place. Making a small incision in questionable bowel to determine how easily it bleeds and the appearance of the mucosa is fully justified.
- Such clinical assessment of viability is not uniformly reliable, and its accuracy has been put as low as 36%. The use of these criteria becomes more beneficial with experience. It has been shown experimentally that following release of jejunal ligatures applied 50 minutes earlier, progressive degeneration continued even though there was gross evidence of perfusion and muscular activity soon after release. A number of alternative techniques have been investigated.
- The most popular clinically applicable methods of evaluating intestinal viability measure tissue perfusion by some direct or indirect method. Unfortunately, ischaemic damage can occur at the cellular level in spite of apparently adequate perfusion and can be impossible to discern intraoperatively.
- Intravenous fluorescein and Doppler ultrasound have been the most reported methods of detecting local perfusion of segments of bowel. Experience is needed to use either method.
- Fluorescein dye injected IV (6.6–11 mg/kg) is rapidly distributed to all perfused tissues. The serosal surfaces become yellow/green within 60 seconds and emit a yellow fluorescence when illuminated with long wave ultraviolet light. It is important to compare the segment being evaluated with unaffected small intestine or colon. When reduced fluorescence is generalized, little credence can be placed upon the procedure.
- The Doppler transducer senses the passage of blood through vessels within the wall of the intestine. Signals from arterial flow in any form are considered to indicate viable bowel, with clinical and experimental evidence indicating a high degree of accuracy. One limitation is that the probe has to be placed in the exact area to be evaluated. When compared to sodium fluorescein fluorescence, the Doppler is generally no better in detecting nonviable segments.
- Surface oximetry measures the oxygen tension (mmHg) where the oxygen probe contacts the tissue surface. The PO_2 on the surface of the bowel (PsO_2) depends upon diffusion distance from the nearest vessel, local O_2 consumption, and arterial (PaO_2) and local blood flow. Measurement of PsO_2 gives a qualitative estimate of tissue oxygenation beneath the 3-mm electrode. Applied to ischaemic bowel, values are compared to normal bowel and to PaO_2.
- Luminal pressure provides an indirect method of determining bowel viability. Clinical studies in equine small intestine and large colon have shown that horses with strangulating obstruction have greater luminal pressures than those with simple obstructions and that survivors have significantly lower luminal pressures than non-survivors. The distribution of horses with large colon volvulus into survivor and non-survivor categories can be predicted using a luminal pressure of 38 cm H_2O as the dividing line.
- Histopathological examination of intestinal wall provides an objective method for evaluating bowel viability but is applicable only to large colon. Interpretation of colonic viability is based on the magnitude of haemorrhage and oedema in the mucosa and submucosa, the percentage of superficial epithelial cell loss and crypt cell damage and the interstitial-to-crypt ratio (normally less than 1). The I:C also indirectly reflects the extent of crypt loss because the crypts collapse with increased oedema in the lamina propria. Non-viable colon has more than 97% superficial cell loss, more than 50% crypt cell loss and an I:C of more than 3. While the facility to obtain histopathological results intra-operatively is rarely available to the surgeon, if the biopsy is processed within the next 24 hours the patient can be euthanased if the results show the colon to have undergone irreversible damage thus saving unnecessary suffering and expense.

In summary, evaluation of bowel viability remains a perplexing surgical dilemma and at present there is no totally reliable method available. Viability refers to the ability of the intestine to survive without developing adhesions, or much more seriously, without the horse experiencing shock, ileus, peritonitis and death. Clinical judgement tends to be pessimistic. Although the colour may improve once the obstruction is relieved, serosal petechiation and mural oedema will not resolve within the time available to make the assessment. Spontaneous or evoked motility will appear sluggish in viable strangulated bowel because of 'splinting' of the muscle wall by oedema and haemorrhage. Intestine showing advanced infarction leaves the surgeon no option but to resect. However, bowel showing equivocal changes is more problematical. Whereas in the past it has been customary to advise resection of suspect bowel, the risk of adhesions with resection and anastomosis, together with increased anaesthetic time, must be balanced against the risk of complications arising from leaving compromised bowel *in situ*. Experience has shown that leaving *in situ* suspect bowel which previously would have been resected, in selected cases proves to be the right decision.

Resection of small intestine

A physiological limit of small intestine resection has been identified in the horse. Ponies in which 50% of the small intestine had been resected maintained their pre-surgical weight. These findings correlate well with clinical experience which suggests that, in all but exceptional cases, 8 metres is the maximum length which should be removed.

Resection and anastomosis

- Resection and anastomosis of the jejunum in the horse are simplified by the long mesentery, the clearly visible blood vessels for ligation and the intestinal calibre which allows ample inversion.
- It is widely accepted that, when technically feasible, an end-to-end anastomosis is the simplest and most physiologically compatible method.
- Whenever possible, the fluid-filled ischaemic bowel should be isolated with intestinal clamps before manipulation is begun. If not contained in this way, toxic fluids will reflux into normal intestine and be absorbed, resulting in rapid physiological deterioration of the patient. Failing that, the gut should be clamped as soon as it is freed.
- A suitable site for resection can subsequently be chosen proximal and distal to the strangulated bowel.
- The mesenteric vessels to the compromised bowel are double ligated, the ends of the proximal ligature being left long.
- The distal end of the gut is transected and the mesentery incised between the ligatures.
- As each vessel is cut, the long ends of the individual proximal ligatures are tied together. This greatly reduces the length of the gap in the mesentery to be closed when anastomosis has been completed. This is particularly helpful when a long length of intestine has been resected.
- With the infarcted gut now mobilized in this way, the distal end can be carefully lifted beyond the abdomen and drained into a suitable receptacle by removing the distal clamp.
- Removing the proximal clamp allows decompression of much of the distended bowel proximal to the ischaemic portion.

Open end-to-end anastomosis using one of a number of inversion suture patterns is still the most commonly employed method of intestinal anastomosis in the horse, but other methods have been evaluated experimentally and clinically. The author prefers a two-layer closure comprising a simple continuous suture for the mucosa and a Cushing seromuscular suture (Figure 2.1), or alternatively a single-layer closure using interrupted Lembert sutures. Synthetic absorbable suture material (e.g. polyglactin or polyglycolic acid) is used in both types of anastomosis.

Closure of the mesentery

- After the anastomosis has been checked for patency and leakage, the remaining gap from the stump of the mesentery to the mesenteric border of the intestine is closed with a continuous suture. Care is taken not to create a kink in the intestine by pulling the suture too tightly.
- To prevent adhesion formation to the extensive stump of the mesentery in which some ischaemic tissue remains distal to the ligature after resection of a long length of bowel, it can be enveloped in two folds of adjacent mesentery, one drawn from each side.
- The intestinal segment and the mesentery are rinsed clean of blood using warm polyionic solution before being carefully replaced itno the abdomen.

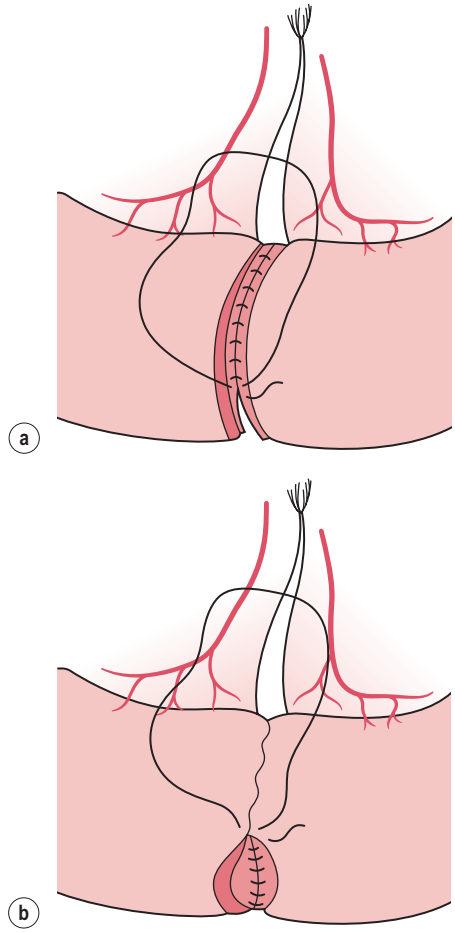


Figure 2.1 Two-layer end-to-end anastomosis. (a) Mucosa is closed with a simple continuous suture. (b) Anastomosis is completed with a Cushing suture.

Anastomosis of intestinal segments of different diameters

- Dilatation of the proximal segment necessitates joining intestine of unequal diameters. While this can be overcome by cutting the smaller diameter segment obliquely, a side-to-side anastomosis is preferable and is essential when chronic obstruction of small intestine due to progressive constriction of its lumen by neoplasia or ileoileal intussusception results in gross hypertrophy of the proximal bowel.
- Side-to-side anastomosis is used routinely by some for all jejunal anastomoses.
- It is also used for jejuno- and ileo-caecostomy, for bypassing obstructions and anastomosing adjoining segments of large colon.

Resection of the ileum

- The ileum is involved in obstruction almost as frequently as the all of the entire 21 meters or so of the rest of the small intestine. Many of these obstructions result in strangulation of bowel necessitating resection and anastomosis which, due to inaccessibility of the terminal ileum can be technically difficult if the infarction extends below the abdominal incision.

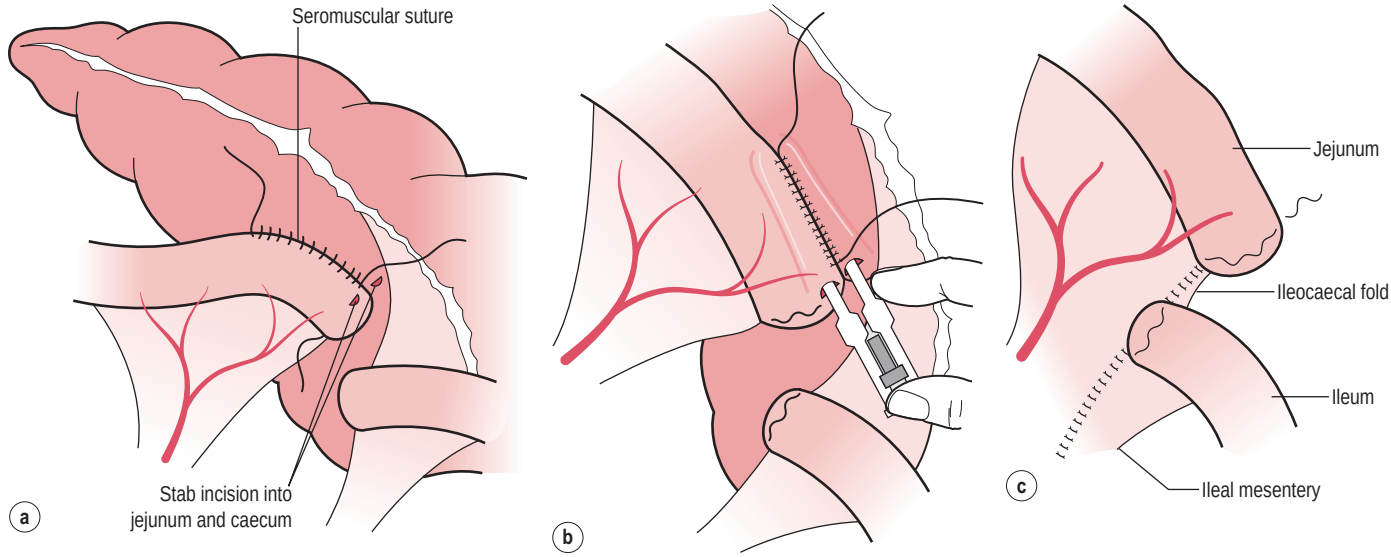


Figure 2.2 Side-to-side jejunocaecal anastomosis using a Gastrointestinal Anastomosis stapler. (a) Jejunum is anchored to the caecum with a continuous seromuscular suture and a small stab incision is made into each. (b) The forks of the stapling instrument are inserted and locked together prior to pushing the firing knob. (c) The mesenteric defect is closed by suturing the cut edge of the jejunal mesentery to that of the ileum and to the ileocaecal fold.

- Side-to-side ileocaecal and jejunocaecal anastomoses whereby the natural ileocaecal orifice is bypassed have been employed in countless thousands of horses since first described by Donawick in 1971.
- A right-angled rectal clamp is applied as low down the ileum as possible. After the contents have been stripped proximally for 20 cm an intestinal crushing clamp is applied and the ileum is transected just distal to this clamp.
- Oedema and friability frequently makes closure of the ileal stump difficult. Compressing the end of the ileum between dry swabs will reduce the thickness of its wall by expressing submucosal oedema fluid thereby facilitating inversion.
- To avoid leaving potentially necrotic ileum in situ it is necessary to progressively invaginate it into itself until only viable ileum is in direct contact with the peritoneal cavity. Alternatively, the ileum may be occluded using an automated stapler as close as possible to the caecum and then transected.
- The jejunum is clamped and transected at a suitable site proximal to the ischaemic bowel.
- After closure of the jejunal end with a double layer inversion suture, the jejunum is placed between the dorsal and medial bands of the caecum as far dorsally on the body with its closed end directed towards the caecal base.
- A side-to-side anastomosis is now performed creating an opening 10–12 cm in length using a conventional suture pattern or alternatively a 10-cm linear stapler may be used (Figure 2.2).
- On completion of the anastomosis the mesenteric defect is closed by suturing the cut edge of the jejunal mesentery to that of the ileum and the ileocaecal fold. This is not possible when the ileal stump is very short.
- A modification of this technique in which the ileum is anastomosed side-to-side to the caecum without resection can be used to bypass nonstrangulating obstructions caused by hypertrophy or ileo-ileal intussusceptions.

Survival studies of large numbers of cases have shown that horses which have undergone side-to-side jejunocaecostomy have a poorer prognosis and a greater risk of complications than those undergoing end-to-end jejuno-jejunal anastomosis.explained by:

- Creation of a sharp transition between intestinal segments of dissimilar function.
- Jejunum must overcome intra-caecal pressure to empty, without the coordinating mechanism of the ileum and the ileocaecal valve.

Consequently end-to-end jejunoileal anastomosis should be considered as an alternative to jejunocaecostomy provided healthy, accessible ileum is available. Obstruction can occur at the anastomosis due to residual oedema of the ileum or delay in a return to normal motility.

Automated mechanical stapling techniques

- The use of stapling equipment in equine intestinal surgery is now becoming widespread. The various instruments have been developed for intestinal anastomosis to reduce surgery time and thereby increase the chances of survival of critically ill patients.
- The very fine stainless steel staples, which close to form a perfect B, preserve the blood supply to the tissues involved in the anastomosis and therefore avoid ischaemia.
- The linear staplers, which have 30-mm, 60-mm, and 90-mm cartridges, insert staples in a double staggered row. The 4.8-mm size of staple is recommended for equine use. The application of these linear staplers include closure of intestine during resection of small or large intestine. As the instrument does not cut automatically, resection must be must subsequently be performed with a scalpel.
- The gastrointestinal anastomosis instrument (GIA) inserts staples arranged in two double-staggered rows 3.5 mm apart. A knife-blade cuts between them creating a side-to-side anastomosis. It is used for jejuno- and ileo-caecostomy, gastrojejunostomy and large colon anastomoses.
- Care should always be taken not to use stapling equipment if tissues are oedematous or thickened because of the risk of disruption and leakage.

Resection of large intestine

Resection of large intestine in the horse is performed very much less frequently than resection of the small intestine for two reasons:

- Many of the obstruction are caused by displacement with no, or only minimal, interference with the blood supply, or simple obstruction of the lumen, for example by enteroliths, which can usually be relieved by simple enterotomy.
- When a strangulating obstruction is present, the length of bowel involved may be so great that resection and anastomosis is not considered feasible because of technical difficulties or because of likely postoperative nutritional or metabolic problems.

Caecum

Partial resection of the caecum is indicated when it shows infarctive changes due to intussusceptions or thromboembolism. A small amount of healthy caecum beyond the infarcted zone is included in the resection which is usually performed at the attachment of the caecocolic fold. The medial and lateral caecal arteries and veins are double ligated in their course along the medial and lateral bands. The diseased portion is excised, and closure carried out in two layers using a Cushing suture or, alternatively, with staples.

Total resection of the caecum, together with an ileocolostomy requires an approach through a lateral right flank laparotomy in which the 17th and 18th ribs are resected.

Large colon

Clinical and experimental investigations have shown that horses can tolerate resection of up to 90% of the large colon remarkably well. Many of the technical difficulties can be overcome by the use of automated stapling equipment, but if the compromised bowel is restricted to the left colon, easy access allows resection and anastomosis to be carried out successfully using conventional techniques.

Indications for resection at the level of left colon include:

- Idiopathic focal eosinophilic enteritis (IFEE) lesion.
- Non-strangulating infarction.

Short focal lesions such as an IFEE lesion at the typical site in the left dorsal colon can be removed by wedge resection and continuity restored by end-to-end anastomosis (Figure 2.3a).

More extensive lesions require resection of pelvic flexure together with a length of left dorsal and left ventral colon. A side-to-side anastomosis is required because of the disparity in diameters (Figure 2.3b).

Resection of the bulk of the large colon 25–35 cm distal to the caecocolic fold is greatly facilitated by the use of automated stapling equipment, but mural oedema can be a limiting factor. Resection at this level has been used predominantly in horses with large colon volvulus. Patients with advanced ischaemic compromise and resulting endotoxaemia are not good candidates for resection. Additional surgery time worsens the prognosis, and many patients are euthanased on the operating table or succumb during anaesthetic recovery.

In horses with large colon volvulus of >270 degrees with less severe mural compromise, and those with non-strangulating lesions, the prognosis is more favourable. However, long-term complications, particularly repeated bouts of colic and weight loss, have been reported in up to 50% of cases.

Small colon

Following resection of infarcted small colon, continuity is restored by end-to-end anastomosis using interrupted Lembert sutures.

The large amount of fat in the mesocolon makes identification of the vessels more difficult than identification of the jejunal mesenteric

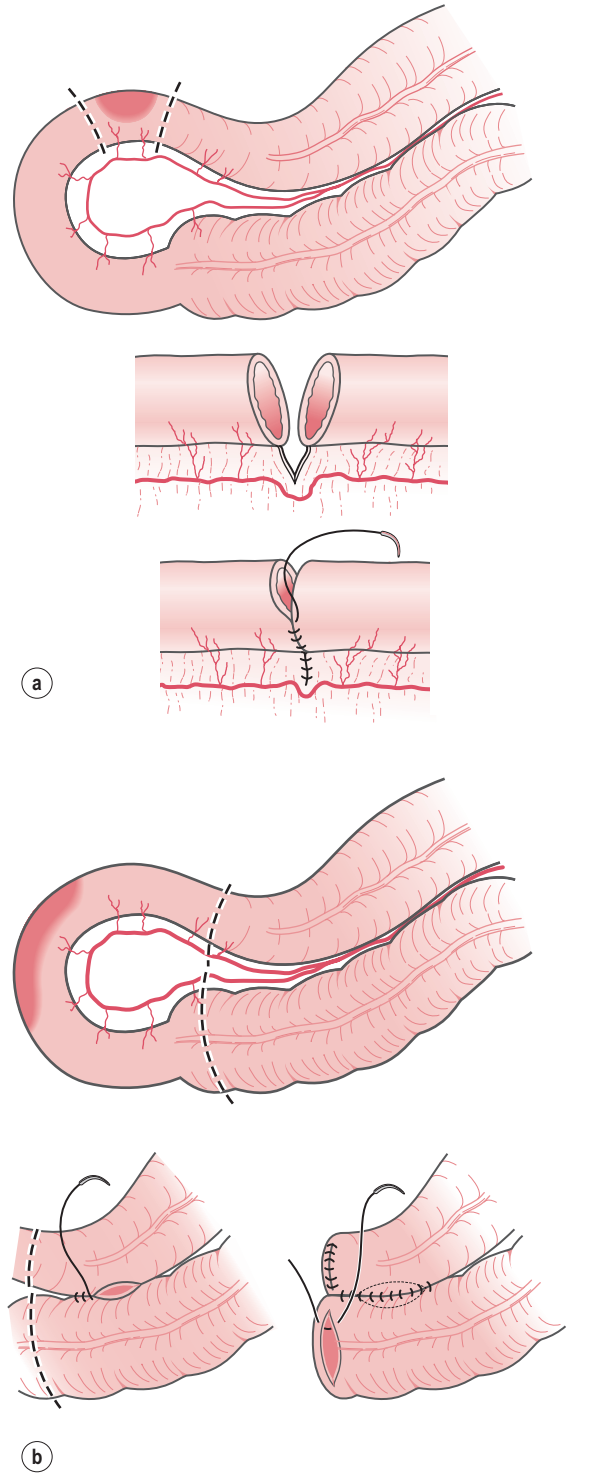


Figure 2.3 (a) Wedge resection of an IFEE lesion at the typical site in the left dorsal colon. End-to-end anastomosis is performed using interrupted Lembert sutures. (b) More extensive lesion requiring resection of approximately 45 cm of left ventral and left dorsal colon. Continuity is restored using a side-to-side anastomosis.

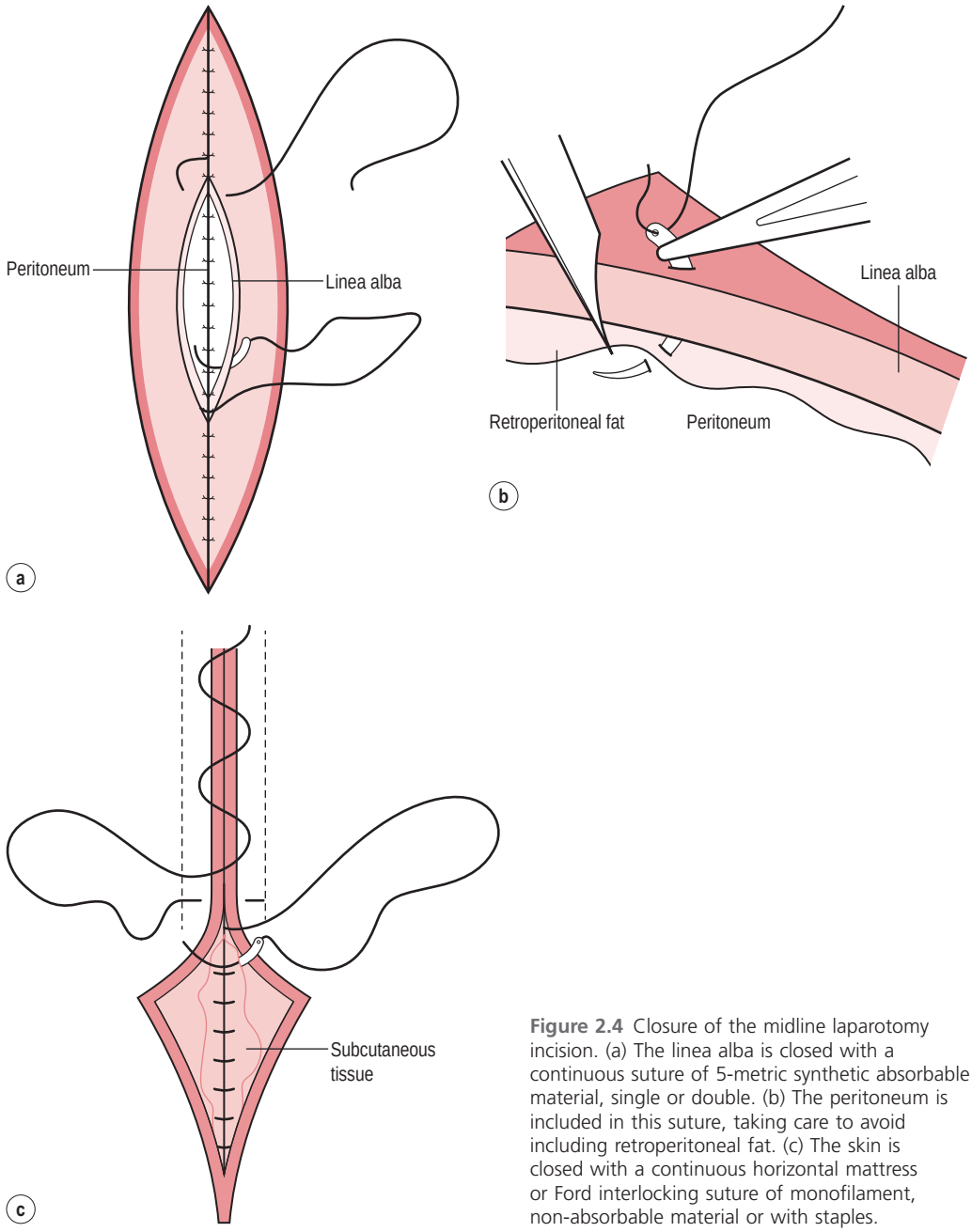


Figure 2.4 Closure of the midline laparotomy incision. (a) The linea alba is closed with a continuous suture of 5-metric synthetic absorbable material, single or double. (b) The peritoneum is included in this suture, taking care to avoid including retroperitoneal fat. (c) The skin is closed with a continuous horizontal mattress or Ford interlocking suture of monofilament, non-absorbable material or with staples.

vessels. Particular care must also be taken when placing sutures at the mesenteric border to ensure a water-tight seal.

Closure of the midline incision (Figure 2.4)

Midline incisions heal more slowly than paramedian or flank incisions because of the relative lack of vasculature of the linea alba, but reconstruction can be accomplished quickly and easily with only minimal risk of herniation and evisceration. Although a wide variety of suture techniques and materials have been described, closure using a continuous suture of synthetic absorbable material has been the standard method for more than twenty years.

- The peritoneum is thin and often tears if an attempt is made to close it on its own. This can be avoided by including it in the continuous suture used to close the linea alba. If the layer of retroperitoneal fat is very thick the peritoneum is left unsutured. Experimental and clinical experience has shown that this has no detrimental effects.
- Closure of the linea alba with a continuous suture of 5-metric, synthetic absorbable material such as polyglactin or lactomer 9-1 has proved reliable and effective in a very large number of horses. Lactomer 9-1 is available as a 120-cm long loop allowing it to be used double, which is particularly useful in very large horses.
- The suture bites should be 12 to 15 mm from the margins of the incision and a similar distance apart and the edges approximated without undue tension.
- Thorough decompression of the intestine and good abdominal relaxation greatly facilitate abdominal closure.

- Experience has shown that closure of the subcutaneous tissue is not necessary and that its omission reduces the risk of postoperative swelling and discharge.
- The skin is closed with a Ford interlocking suture of prolene or staples.

2.11 POSTOPERATIVE MANAGEMENT OF THE COLIC CASE

Adequate postoperative management of the equine abdominal patient is just as important to patient survival as the most heroic corrective surgery. Its success depends on adequate intensive care based on close monitoring of physiological values, including acid–base, electrolyte and fluid balance. Routine management of the uncomplicated surgical case is simple and places little strain on resources in terms of personnel or laboratory facilities, but patients with postoperative complications, particularly ileus, demand a great deal of supervision which may last several days.

Postoperative management of horses undergoing colic surgery depends upon:

- The nature of the initial lesion.
- The physiological condition of the horse at the time of surgery.
- The anticipated complications.

There are no fixed rules or formulae that apply to postoperative management. Each case should be managed individually and plans adapted according to the horse's changing condition.

Monitoring protocol

The adoption of a protocol for monitoring the postoperative colic patient, especially when carried out by a number of people over some time, should aid accurate assessment. A flow sheet is needed to systematically record all the required physical and laboratory data so that the previous examinations can be compared and progress, or lack of it, can be assessed.

The two major factors which affect cases adversely are loss of propulsive gut activity (ileus) and cardiovascular insufficiency (shock). The following values have proved valuable for early diagnosis of these and other complications:

- Heart rate and pulse quality.
- Mucous membrane colour and capillary refill time.
- Packed cell volume.
- Pain.
- Rectal temperature.
- General demeanour.
- Urination and defecation.
- Gut sounds.
- Nasogastric intubation.
- Rectal examination.
- Wound integrity.
- Abdominal wall tension.
- Willingness to move.
- Digital pulses.

Fluids, analgesics and other treatments administered are recorded.

Frequent monitoring of the horse's clinical condition and hydration status is very important in the first 24 hours. Examination every 4 hours is recommended – more frequently if the individual case merits it. Foals should be monitored every 2 hours because their physiological condition changes more rapidly.

Once heart rate, PCV and total protein have returned to normal or near normal, faeces have been passed and gastric reflux has been absent on 2 or 3 successive nasogastric intubations, the examinations can be carried out at intervals of 6–8 hours.

Food and water

- Prolonged fasting (>3 days in adults, less in foals and neonates) results in atrophy of intestinal mucosa, impaired wound healing and increased susceptibility to infection.
- The primary source of energy for enterocytes is glutamine which is obtained from the gut lumen not the blood.

Following enterotomy or resection oral fluids are introduced 24–36 hours postoperatively provided there is evidence of effective gut activity.

Small amounts of food are introduced after 48 hours and amounts slowly increased. Gentle walking exercise may aid recovery by decreasing wound oedema, reducing boredom and possibly encouraging defecation.

Fluid therapy

- Maintaining fluid balance in the postoperative horse, or in the horse in shock, is critical as it affects internal function and the normal body defence systems.
- Isotonic fluids for maintenance requirements (50 mL/kg day = 24 L for a 500 kg horse) plus ongoing losses (diarrhoea, nasogastric reflux, sequestration).
- Requirements are guided by the PCV and TP.
- Electrolyte status should be monitored and the fluids supplemented if necessary.

Catheter care

- Monitoring catheter patency and vein reaction to the catheter is important for proper fluid and drug administration as well as for prevention of thrombophlebitis, which in its most severe form can be life threatening.
- The catheter site should be part of the daily monitoring, and the findings recorded on the flow chart.
- If there is any evidence of exudate at the site of catheter entry, or perivascular thickening, heat or pain, the catheter should be removed immediately.
- Horses suffering from severe endotoxaemia at the time of referral are often in a highly 'coagulable' state. Postoperative therapy in these cases may continue for several days so vein patency is essential.
- If the jugular vein used shows signs of thrombophlebitis it is preferable to use the externjal thoracic vein than to risk similar obstruction developing in the other jugular vein.

Analgesia and anti-inflammatory therapy

- If the primary problem has been corrected and normal gut activity has returned, little analgesia should be required. Some horses will show signs of mild discomfort for the first 12–24 hours after surgery: pain beyond this period may indicate ileus, continued bowel ischaemia or adhesion formation.
- Most horses will receive flunixin to reduce the effects of any residual endotoxaemia.
- 1.1 mg/kg IV BID for the first 24 hours.
- 0.5 mg/kg IV BID for the second 24 hours.
- 0.25 mg/kg IV TID for the third 24 hours.

Antibiotics

- Antibiotics are commonly used in the prevention and treatment of shock in the acute abdominal patient.
- A 3 to 5-day course of a combination of penicillin and an aminoglycoside antibiotic is usually sufficient. May be administered for longer if wound suppuration occurs.

2.12 POSTOPERATIVE COMPLICATIONS

Several studies documenting the incidence of postoperative complications have been reported. Some complications may occur as early as the period of anaesthetic recovery, while others may not occur until weeks or months after discharge from the hospital. However the majority occur during the first week after surgery. Some are life-threatening but others are more benign.

Complications during recovery from anaesthetic

Myopathy

Postoperative myopathy is a possible complication of any extended surgical procedure requiring maintenance of the horse under general anaesthesia. With horses in dorsal recumbency, the gluteal and longissimus dorsi muscles are most likely to be affected because of direct pressure combined with inadequate circulation in the hind limbs due to pressure on abdominal vessels by distended viscera. An intraoperative fall in arterial blood pressure is another important contributory factor. Careful positioning, adequate padding and a short operating time are important, but the ability of anaesthetists to maintain adequate arterial blood pressure during surgery has been critical in reducing the incidence of this problem.

Affected horses are reluctant or unable to rise or may show slight hind limb stiffness. Euthanasia is necessary if no improvement is made, the horse becomes distressed, or fractures a limb during attempts to stand.

Long-bone fractures

The incidence of long-bone fracture is higher following colic than other types of surgery. This may be due to the debilitated state of the horse, muscle and nerve damage, electrolyte imbalances and/or major central hypoxia, all of which may contribute to incoordination.

Complications during the hospitalization period

Shock

Shock is a constant hazard of colic and may be fatal especially during the first few hours after surgery. It is hypovolaemic and/or endotoxic in origin. If intraoperative fluid therapy has been sufficient, plasma volume may have been restored, but this may be a temporary improvement. Endotoxaemia is associated with altered haemostasis including disseminated intravascular coagulation (DIC).

Much has been written about the treatment of shock. No single case is identical to another, but some guidelines do exist. During the immediate postoperative period while the horse is still recumbent, enriching the inspired air with oxygen to increase arterial oxygen tension is of benefit to the patient.

A rising packed cell volume indicates the need for for fluid therapy, but plasma protein concentrations should be measured to avoid over use of crystalloid solutions which may produce a marked hypoproteinaemia. The possibility of postoperative ileus should be considered in these cases. Hartmann's solution is the fluid of choice, but the constituents of the replacement therapy should be related to laboratory results of plasma electrolyte and protein status. Acidosis is probable but not invariable.

Endotoxamis and SIRS

Endotoxaemia and SIRS are comon in horses with colic and contributes to the morbidity and mortality. Treatment of endotoxaemia and SIRS and its related effects (SIRS) is more complex, although fluid therapy and electrolytes are important. Pretreatment using pharmacological agents and prevention of the effect of endotoxin on the inflammatory cascade would be ideal. However at the time the colic case is evaluated endotoxin has usually already been released and has produced clinical signs of shock. Nevertheless some pharmacological agents can, to some degree, block the effects of endotoxin after the cascade has been initiated and block further release of endotoxin.

The cyclo-oxygenase inhibitor flunixin is used routinely. It interferes with the metabolism of arachidonic acid to arrest the production of vasoactive metabolites mediating arterial hypoxia, platelet adhesion and lactic acidosis. The drug should be given during surgery (1.1 mg/kg IV) followed by 0.25 mg/kg at 8-hourly intervals.

Polymixin B is an antimicrobial drug that is effective against Gram-negative bacteria. However, at antibacterial doses it has severe nephrotoxicity and neurotoxicity – limiting side-effects.

At lower doses, polymixin B acts as a chelating agent by binding to the lipid-A moiety of lipopolysaccharide (LPS), which removes endotoxin from the vascular system, and by that action, prevents the development of the pro-inflammatory cascade.

In horses with clinical evidence of endotoxaemia, the use of aggressive IV fluid therapy is indicated before polymixin B is used. Consequently it is convenient to commence treatment towards the end of the surgical procedure by which time haemodynamic status has been achieved and azotaemia is resolved. 6000 units/kg IV is given in 1 L of isotonic saline solution over 15–30 minutes and repeated every 8 hours for 5 doses.

Postoperative ileus (POI)

Postoperative ileus has a reported prevalence of 10–47% and results in an incidence of mortality of 13-86% in the horse and is particularly prevalent in those horses that have undergone small intestinal manipulation and/or resection. Unlike man, where the clinical signs are related to large intestine dysmotility with bloating due to failure to pass accumulated colonic gas, in horses the clinical signs are more related to small intestine dysmotility with reflux of large volumes of small intestine contents into the stomach which requires frequent removal by nasogastric tube.

Frequently the horse makes a good anaesthetic recovery and may appear bright during the first part of the postoperative period. There is the loss of bowel sounds after a few hours, but in a small number of cases this may be delayed for 2–3 days or longer. The PCV and TPP rise steadily in the absence of effective transport of gut contents, any fluid therapy producing only temporary improvement. Deterioration in mucous membrane colour and capillary refill time, accompanied by a corresponding rise in heart rate, are evidence of circulatory collapse. The horse shows mild to moderate abdominal discomfort. Accumulation of fluid in the stomach and small intestine is confirmed by nasogastric intubation, ultrasonography and rectal examination.

POI falls into one of two categories:

- Ileus which is associated with a demonstrable cause such as a mechanical obstruction or continuing presence of devitalized bowel.
- A functional ileus, sometimes designated idiopathic postoperative ileus (IPOI), the causes of which are not fully understood. Endotoxaemia and intestinal distension resulting from sequestration of fluid, together with inflammation in the intestinal muscle layers and myenteric neural plexus sufficient to disrupt motility, brought about about by intraoperative manipulation, are believed to be important factors. PCV at admission is an important risk factor.

It is important to attempt to determine into which category the patient falls.

For more than 30 years considerable time and effort has been directed at finding a pharmacological panacea to restore normal gut motility as quickly as possible, but without much success.

This focus on drugs, combined with diagnosis of POI based on insufficiently rigorous criteria, has resulted in cases being treated which would have benefited more from undergoing a second celiotomy without delay to resolve an iatrogenic obstruction.

The following criteria have been suggested to assist the veterinarian in identifying and resolving ileus.

1. Clinical findings which are indicative of abnormal transit due either to functional ileus or simple obstruction.
   - Volume of gastric reflux >4 L at any intubation or >2 L/hour on repeated intubation.
   - Heart rate persisting >40/min.
   - Mild to severe signs of abdominal discomfort.
   - Rectal or ultrasound evidence of distended, fluid-filled small intestine.
2. The odds are in favour of functional ileus if:
   - The heart rate and signs of abdominal discomfort decrease following gastric decompression.
3. The odds are in favour of intestinal obstruction if:
   - A jejunocaecostomy was performed.
   - An anastomosis involving any part of the small intestine was performed using staples.
   - Cardiovascular parameters continue to deteriorate even after gastric decompression.
   - Severe signs of abdominal discomfort continue or progress after gastric decompression.
   - Rectal or ultrasound examination indicates something more complex than simple small intestinal distension.

The equine intestinal tract appears to be sensitive to minor mechanical obstructions imposed by different types and methods of anastomosis. Narrowing of the lumen at the anastomosis, intestinal rotation at or near the anastomosis, and mesenteric shortening or rotation are sufficient to cause intestinal dysfunction and many of horses with these complications might benefit from repeat celiotomy.

Correct management of IPOI cases is more controversial. Conventional management aims to maintain gastric decompression by nasogastric suction (10–15 L may be removed at 3–4 hour intervals), hydration and electrolyte and acid–base balance by IV fluid therapy, and to restart gut activity.

There is no generally accepted pharmacological treatment for IPOI, and there is even debate as to whether it has a part to play in the management of the condition. Altering intestinal motility may not be possible if there has been severe ischaemia or distension. Any drug given for this purpose is best administered as a prophylactic measure at the end of surgery when the gut has been decompressed.

Lidocaine administered as a bolus of 1.3 mg/kg IV over 5 minutes followed by infusion of 0.05 mg/kg/min in isotonic saline solution over 24 hours is currently the most commonly used drug used to treat POI and other motility disorders. The mechanism of lidocaine is presumed to be inhibition of sensory nerve action within the wall of the intestine, thereby reducing reflex sympathetic inhibitory activity. In addition, IV administered lidocaine appears to be an effective analgesic. Thus an important feature of IV lidocaine therapy may be to control postoperative pain-induced reduction of gastrointestinal activity.

Nursing care is very important. Frequent short walks and occasional mouthfuls of grass are useful in stimulating gut activity.

Repeat celiotomy to decompress distended small intestine should be considered sooner rather than later if ileus persists and can be a lifesaving procedure. The disadvantages of repeat celiotomy are expense and risk of incisional complications – infection and sometimes hernia formation. Survival following repeat celiotomy ranges from 43% to 56% but can be as low as 20% long-term.

Colic

A significant number of horses suffer from one or more episodes of colic after undergoing colic surgery. However, less than 5% of horses suffer three or more episodes, Most episodes occur during the first year after surgery. Postoperative colic was the major reason for euthanasia following discharge from the hospital in one study. Horses that had suffered strangulating torsion of the large colon were three times more likely to develop colic as were horses which had undergone repeat celiotomy. The cause of colic in these cases is not clear, but it is likely that adhesions play an important role.

Wound complications

The most common wound problem is **superficial infection** of the celiotomy, with a incidence of up to 37%. Variables significantly associated with wound suppuration have not been identified. Specifically, the choice of postoperative antibiotic, enterectomy/enterotomy and repeat celiotomy were not found to be significant risk factors in one study.

Affected horses show moderately severe oedema along the length of the incision. One or more foci of exudates appear 5–7 days postoperatively. The oedema then subsides. Systemically administered antibiotics and frequent wiping clean of the incision aid resolution. Most of these infections will continue to discharge for 1–4 weeks and then resolve.

Incisional herniation may be a delayed complication of low-grade wound infection. Exercise too soon after surgery and weight >600 kg are further significant risk factors (see Chapter 4).

Wound dehiscence is life-threatening but fortunately very rare.

Recent studies have shown that applying an abdominal bandage immediately after recovery from abdominal surgery and maintaining it for 1–3 weeks significantly reduces incisional complications.

Thrombophlebitis

See Chapter 7.

Laminitis

Laminitis is an ever present threat to horses with an acute abdomen. The cause and pathology are still not fully understood, and the ability to totally prevent the problem is still elusive. It appears to be due to the systemic absorption of endotoxin. All horses with intestinal disease, particularly those with strangulating obstructions or grain overload, are susceptible.

Low-dose flunixin therapy helps to minimize the risk of laminitis. The feet of colic cases should be monitored frequently for increased digital pulsation and heat, and the horse's willingness to move freely noted. The onset of laminitis as determined by clinical signs is often on the second, third or, occasionally, the fourth day after surgery. Foot pads should be applied to provide frog support.

Adhesions

Studies in laboratory animals and humans suggest that a large percentage of horses undergoing exploratory celiotomy will develop adhesions. The incidence increases with enterotomy, small intestinal surgery, repeat celiotomy and postoperative ileus.

Precisely how often they become problematical is unknown. However, they were shown in one series to be the cause of colic in 18% of horses with chronic colic, and the most common pathological finding at repeat celiotomy in another.

Since surgery is one of the main initiators of adhesions, surgery to correct them usually results in the formation of further adhesions. Therefore the goal should be to prevent adhesions. Good surgical technique aimed at reducing peritoneal and serosal inflammation by avoiding leaving ischaemic tissue, gentle handling of intestine, precise haemostasis, minimizing contamination, and ensuring that mucosa is inverted is the best advice available

Many chemicals and drugs have been evaluated as potential agents in the prevention of adhesions including NSAIDs, antimicrobials, peritoneal lavage, heparin and carboxymethylcellulose. While none has proven unequivocally to be of benefit, some appear to be more effective than others.

1% carboxymethylcellulose (CBCM) applied to the surface of intestine at surgery protects it during handling and prevents ingesta and bacteria from adhering to it. A recent study has shown that application of CBMC to small intestine and surrounding viscera during surgery to correct an obstruction improves post operative survival. Horses in which CBMC was not used were twice as likely to die as those in which it was not employed.

Postoperative diarrhoea

Horses which have undergone surgery to correct strangulating volvulus of the large colon, resection of small colon or resection or bypass of the caecum, frequently have loose faeces early in the postoperative period. However, normal faecal consistency is quickly restored when damaged mucosa is repaired (large colon volvulus), or the water absorbing function is taken over by the remaining gut (small colon or caecal resection).

It is important to differentiate this type of diarrhoea from more serious causes such as *salmonellosis* or *clostridial* overgrowth. A precipitous drop in leucocyte count, pyrexia and depression may herald a *Salmonella* infection in advance of frank diarrhoea. All cases of postoperative diarrhoea should be placed in strict isolation until the cause has been identified.

Septic Peritonitis

Peritonitis may result from significant contamination of the peritoneal cavity with ingesta during surgery or from leakage of endotoxin and bacteria due to necrosis of the entire bowel wall or from a mucosal injury only.

Recognition of septic peritonitis during the postoperative period is based on the presence of one or more signs including ileus, fever, colic, and abdominal guarding. The diagnosis is confirmed by analysis of peritoneal fluid.

Broad spectrum antibiotics, NSAIDs and maintenance of hydration are the main elements of treatment. Peritoneal lavage may also be helpful.

FURTHER READING

Mair TS, Proudman CJ (guest editors) (2011) Supplement 39. Equine Veterinary Journal 43:1–155

Merritt AL, Blikslager AT, Pinchbeck GL (guest editors) (2008) Colic special issue. Equine Veterinary Journal 40(4):293–428

White NA, Moore JM, Mair TS (eds) (2008) The equine acute abdomen, 2nd edn. Teton New Media, Jackson WY

Chapter 3

Gastroenterology 2. Hepatic and intestinal disorders

Tim S. Mair, Sandy Love

CONTENTS

Liver disease 49
3.1 Diagnostic approach to liver disease 50
3.2 Pyrrolizidine toxicity 51
3.3 Theiler's disease (acute hepatic necrosis, serum hepatitis, serum sickness) 52
3.4 Cirrhosis 52
3.5 Liver fluke 52
3.6 Cholangiohepatitis and choledocholithiasis (biliary calculi) 52
3.7 Chronic active hepatitis 53
3.8 Iron overload (haemochromatosis) 53
3.9 Hydatid cysts 54
3.10 Other hepatic diseases 54
3.11 Treatment for liver failure 54
Pancreatic diseases 54
3.12 Pancreatic diseases 54
Equine grass sickness 54
3.13 Equine grass sickness (equine dysautonomia) 54
Diseases of the stomach (equine dysautonomia) 56
3.14 Gastric ulceration (equine gastric ulcer syndrome) 56
3.15 Gastric impaction 57
3.16 Gastric neoplasia 58
Intestinal disorders 58
3.17 Diagnostic approach to intestinal disease 59
3.18 Chronic inflammatory bowel disease 59
3.19 Alimentary lymphoma 61
3.20 Sand enteropathy 62
3.21 Idiopathic chronic diarrhoea 62
3.22 Acute enterocolitis 63
3.23 Non-steroidal anti-inflammatory drug toxicity 64
3.24 Other gastrointestinal disorders 64
Further reading 65
Appendix 65

Liver disease

Liver disease is relatively common in the horse, and there are a large number of disorders that can cause hepatic disease (i.e. pathological damage to the liver) (Table 3.1) in both adult horses and foals. However, liver failure is much rarer because the liver has a large reserve capacity and good capacity to regenerate. Liver failure will not occur until 70% or more of the organ has been damaged. The distinction between liver disease and liver failure is important clinically. In view of the large reserve capacity, many horses with mild hepatic disease will make a full recovery given time and removal of the inciting cause. However, if the damage progresses to cause liver failure, then the prognosis for survival is much reduced.

The liver has a limited number of ways to respond to various different insults, and, therefore, the clinical and laboratory changes tend to be similar regardless of the underlying cause.

Chronic weight loss is the most common presenting sign of liver failure, but other presentations include dullness, anorexia, behavioural abnormalities, skin lesions, pruritus, bleeding disorders, diarrhoea and colic.

Weight loss is a common, but non-specific, finding in chronic liver disease. It is related to failure of metabolic functions of the liver and reduced food intake.

Hyperammonaemia results when ammonia from portal circulation is not metabolized to urea within the liver and enters the systemic circulation. Raised plasma ammonia affects gluconeogenesis causing utilization of branched-chain amino acids. The resultant decrease in ratio of branched-chain to aromatic amino acids affects neutrotransmission and may give rise to *hepatic encephalopathy* (see Chapter 11) or, very rarely, *peripheral neuropathy* which can lead to unusual complications such as gastric impaction (and colic) or laryngeal paralysis. Signs of hepatoencephalopathy may vary from depression to bizarre maniacal behaviour. Common signs include:

© 2013 Elsevier Ltd
DOI: 10.1016/B978-0-7020-2801-4.00006-7

Table 3.1 Important causes of liver disease in the horse

Toxic causes:
- Pyrrolizidine alkaloid toxicosis.
- Alsike clover.
- Panicum grasses.
- Iron.
- Mycotoxins.

Infectious causes:
- Cholangiohepatitis.
- Tyzzer's disease.
- Hepatic abscess.
- Equine herpesvirus (EHV-1).
- Liver fluke.

Inflammatory, non-infectious causes:
- Chronic active hepatitis.
- Neoplasia.
- Granulomatous disease.

Metabolic causes:
- Hepatic lipidosis.
- Hyperammonaemia in horses with gastrointestinal disease (no liver disease present).
- Hyperammonaemia in Morgan foals.

Obstructive causes:
- Biliary stones.
- Right dorsal colon displacement.
- Papillary stricture – foals with duodenal ulcers.
- Neoplasia.
- Hepatic torsion.
- Portal vein thrombosis.

Unknown / miscellaneous causes:
- Theiler's disease.
- Neonatal isoerthrolysis.
- Senile cirrhosis.

Congenital:
- Portosystemic shunt.

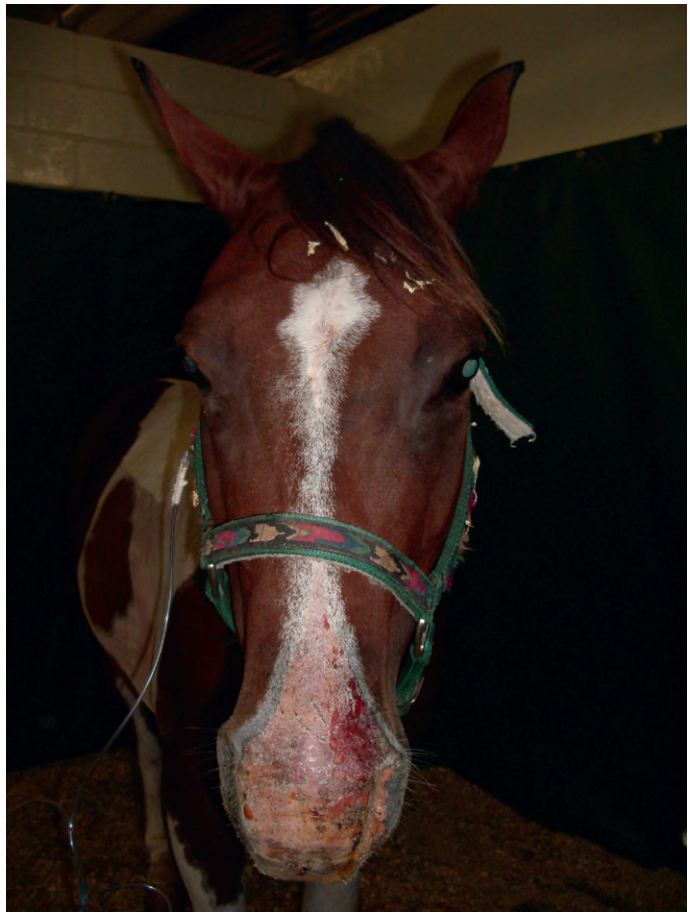


Figure 3.1 Photosensitization of the unpigmented areas on the face.

- apparent blindness.
- ataxia.
- head pressing.
- compulsive circling.
- frequent yawning.

Accumulation of bilirubin in plasma may result in jaundice. There may be decreased hepatic metabolism of photodynamic phylloerythrin which, when exposed to ultraviolet light within superficial dermal circulation, results in necrotic skin lesions in white areas referred to as *photosensitization* (Figure 3.1) (see Chapter 13). Pruritus occasionally occurs due to bile salt accumulation in the skin.

Altered faecal consistency is not uncommon in equine liver failure, and some affected horses may develop diarrhoea, probably caused by portal hypertension or thrombosis. Some affected horses develop abnormally firm faeces. Colic may arise due to hepatocellular swelling and obstruction of the biliary tract. A reduction in intestinal motility is a fairly frequent finding in cases of equine liver failure.

Failure of the liver to synthesize clotting factors may result in an increased *bleeding tendency*, especially after trauma. Spontaneous haemorrhage into the lungs or alimentary tract is rarely seen. Such haemorrhage usually occurs as a terminal event.

The underlying hepatic pathology is generally chronic, but clinical signs are often relatively sudden in onset when the degree of organ failure exceeds the considerable functional reserve capacities of liver. Hepatic failure occurs in a wide variety of conditions which can be difficult to differentiate, but clinical management of affected cases is often similar and directed at ameliorating hyperammonaemia and providing nutritional support.

The main differential diagnoses of equine liver failure are chronic enteropathies and chronic grass sickness.

3.1 DIAGNOSTIC APPROACH TO LIVER DISEASE

History

- Age – in foals, liver failure is associated with rare disorders such as Tyzzer's disease or portosystemic shunt. Senile cirrhosis occurs in elderly horses.
- Multiple cases affected implies ingested hepatotoxin (e.g. pyrrolizidine alkaloid) or, very rarely, liver fluke infection. Also, hyperlipaemia with associated hepatic dysfunction may occur in groups of horses under the same management, and Theiler's disease is seen as a group disease in North Western USA.
- Duration/progression of signs – insidious weight loss is common and non-specific for chronic hepatopathies. Neurological signs associated with hepatic encephalopathy can develop fairly suddenly in chronic advanced liver failure (e.g. pyrrolizidine toxicosis) or, more likely will be the presenting complaint in acute liver failure (e.g. Theiler's disease).

Physical examination

Few physical findings are diagnostic of equine liver disease. Generally the approach is to rule out other possible causes of weight loss, behavioural changes or skin lesions, e.g. signs of enteropathy, primary central nervous system (CNS) disease (Chapter 11) or primary skin disease (Chapter 13).

- Jaundice occurs relatively infrequently in equine liver failure but is more likely in acute and/or cholestatic diseases. Horses commonly become mildly jaundiced from anorexia, even when the liver is not diseased.
- Mucosal petechial/ecchymotic haemorrhages or bleeding due to clotting abnormalities occur uncommonly.
- Peripheral oedema or ascites is rare in equine liver failure.
- Photosensitization lesions occur in unpigmented skin, especially the skin of the head (Figure 3.1).

Laboratory investigation of liver disease

In the mature horse, leakage of hepatic and biliary enzymes into the circulation, failure to convert ammonia to urea and failure to conjugate bilirubin are generally recognized before failure to produce clotting factors or albumin is recognized.

1. Plasma/serum liver enzymes.
   - *Hepatocellular damage:*
     glutamate dehydrogenase (GLDH) – liver specific.
     sorbitol dehydrogenase (SDH) – specific, unstable if stored (e.g. postal).
     lactate dehydrogenase (LDH) – isoenzyme 5 non-specific.
     aspartate transferase (AST) – non-specific.
     ornithine carbamoyltransferase (OCT) – liver specific.
   - *Biliary tract damage or obstruction:*
     gammaglutamyl transferase (γGT) – specific.
     alkaline phosphatase (AP) – non-specific.
2. Liver function.
   Liver function tests only become abnormal when approximately 60-70% of liver function is lost, and these tests include elevations of bile acids, direct bilirubin, blood ammonia, prothrombin and partial thromboplastin time, serum iron, and gamma globulins (with chronic disease).
   - An increase in direct bilirubin is a highly sensitive and specific marker of liver failure due to either hepatocellular or hepatobiliary disease. However, mild hyperbilirubinaemia may occur in horses that are anorexic, regardless of the cause. An increase in direct bilirubin of 25% or more of the total bilirubin is suggestive of a predominant biliary disease. Clinically evident jaundice associated with marked unconjugated hyperbilirubinaemia but in the absence of other biochemical evidence of liver disease is suggestive of haemolytic anaemia. Septic foals with intestinal ileus sometimes have elevations in direct bilirubin with minimal evidence of hepatocellular dysfunction; treatment should focus on the sepsis and intestinal ileus.
   - There may be a decrease in blood urea nitrogen and albumin with chronic liver diseases.
   - Serum or plasma bile acids are elevated in horses with both hepatocellular and hepatobiliary disorders, and elevations can be an early predictor of liver failure when values rise above 30 µmol/L. Unlike for other species, fasting samples are not required in horses to interpret bile acid results, although mild elevations of bile acids (up to 20 µmol/L) may occur as a result of anorexia.
   - Blood ammonia can also be used as an assessment of liver function. However, rapid and careful sample handling is required. Ideally a control sample should be obtained from a healthy horse and measured simultaneously for comparative purposes.
   - Dye excretion tests (such as bromosulphophthalein and indocyanine green) are now very rarely used to assess liver function.
3. Other analytes may be altered in hepatic disease, e.g. hypoglycaemia, hypoalbuminaemia, decreased albumin: globulin ratio, and raised concentrations of triglycerides and cholesterol.
4. Clotting function – prolonged prothrombin time. Many of the standard biochemical indices of liver function/damage possess significantly different reference ranges than adult horses. GGT, bile acids and AP, for example, are normally higher in healthy foals than adults.
5. Reference ranges for many of the standard biochemical indices of liver function differ between foals and adult horses.

Liver biopsy

In many cases biopsy can provide a definitive diagnosis (which laboratory tests cannot). Liver biopsy is best performed after the liver has been visualized by ultrasonography on either the right or left side. Liver biopsies can be useful to determine the amount of fibrosis, inflammation, and predominant location of disease and for culture purposes. Pre-biopsy evaluation of extrinsic, intrinsic and common clotting function, by measurement of prothrombin time (PT) and activated partial thromboplastin time (APTT) is often recommended. Although prolonged clotting times are rare even with severe liver disease, the risk of haemorrhage is higher in adults with Theiler's disease and foals with Tyzzer's disease. Using a 14-cm needle, percutaneous biopsy can also be performed 'blind' in the standing horse at the 12th, 13th or 14th right-sided intercostal spaces between the level of lines drawn from the tuber coxa to the point of the olecranon and to the point of the shoulder.

Diagnostic ultrasound in liver disease

Transabdominal ultrasonography is best performed with either a 2.5-MHz or 5-MHz transducer. In neonatal foals, 7.5-MHz or 10-MHz transducers are effective. The liver is best imaged from the right, immediately caudal and ventral to the lung. Typical landmarks for imaging the liver are the 6th to 15th intercostal spaces on the right, and the 6th to 9th intercostal spaces on the ventral aspect of the abdomen. In neonatal foals, the liver can also be imaged from the ventral aspect of the abdomen. In adults, the image quality is variable, depending on such factors as the underlying disease, normal age changes (right lobe atrophy in old horses), extent of the lung fields, degree of gas distension of the colon, amount of subcutaneous fat, etc. Healthy liver tissue is less echogenic than the spleen, and has a more prominent vascular pattern. The portal veins can be distinguished from the hepatic veins by the greater amount of fibrous tissue in the walls of the portal vessels. Bile ducts are not visible in the normal liver. Discrete lesions such as abscesses/masses, choleliths, biliary sludge and dilated bile ducts can be visualized, and chronic fibrosis or hepatomegaly can be appreciated.

3.2 PYRROLIZIDINE TOXICITY

Ingestion of plants containing pyrrolizidine alkaloids is a common cause of equine liver disease and failure. The plant species most frequently include *Senecio jacobea* (ragwort), *Crotalaria* spp. (rattlebox), *Senecio* spp. (groundsel), *Amsinckia* spp. (riddleneck) and *Echium plantagineum* (Patterson's Curse). These plants are generally unpalatable to horses unless withered, e.g. following pasture-topping or within hay, or if grazing is severely restricted. The intoxication typically results

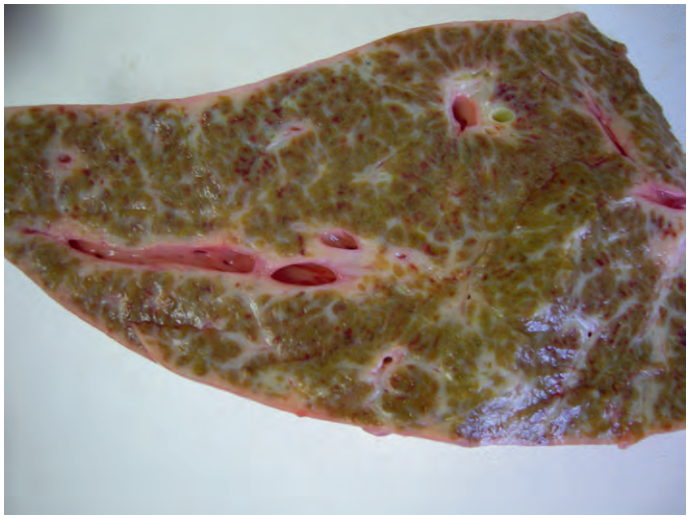


Figure 3.2 Post-mortem appearance of fibrosed, end-stage liver due to pyrrolizidine toxicity.

in the delayed onset of chronic, progressive liver failure. The onset of clinical signs is usually delayed weeks to months following ingestion of pyrrolizidine alkaloid-containing plants. Not all horses that consume the plants develop clinical signs. Horses generally present with variable periods of:

- depression.
- anorexia.
- weight loss.

Horses with areas of unpigmented skin may develop photosensitivity. The clinical course may vary from several days to several months, but when sufficient liver damage has occurred to produce functional failure, there may be an abrupt onset of profound clinical signs of hepatic encephalopathy, and in many cases death. The apparent acute onset of clinical illness generally represents the end stage of a chronic, progressive disease process (Figure 3.2). Clinical signs and death may occur up to a year after the contaminated feed was eaten.

- Elevation of liver-derived serum enzyme activities (SDH and AST) is associated with active liver damage, but activities may decrease toward normal until the later stages of the disease process when marked elevation may again be noted.
- Elevation of GGT and AP activities reflects the focus of the pathological process in the periportal regions and the biliary system.
- Serum bile acid concentration is generally increased.
- Serum bilirubin concentrations may remain within normal limits until the horse reaches a state of functional failure.
- The blood urea nitrogen concentration is generally below normal in horses with functional failure.

Definitive diagnosis can be made by percutaneous biopsy. Typical lesions include megalocytosis, periportal fibrosis, biliary hyperplasia, and occlusion of the central veins. Treatment (see 3.11 below) can be attempted; although the prognosis is poor, some cases survive.

3.3 THEILER'S DISEASE (ACUTE HEPATIC NECROSIS, SERUM HEPATITIS, SERUM SICKNESS)

The aetiology is unknown but often there is a history of the affected horses receiving an equine-origin biological product (now most commonly tetanus antitoxin) 4–10 weeks prior to the onset of clinical signs. In some cases, the affected horses may not have received an antitoxin, but may have been in contact with another horse that had received tetanus antitoxin. It may occur as a group outbreak during autumn in North Western USA but is only rarely recognized as an entity elsewhere. Generally, affected cases develop peracute liver failure (signs of hepatic encephalopathy, jaundice, red discoloured urine, etc) but individuals may have mild or subclinical signs with raised serum liver enzyme concentrations. The history, onset, clinical signs, and histopathological findings of Theiler's disease are similar to hepatitis B virus in humans. However, no viral aetiology has ever been proven. The diagnosis is based on:

- history.
- clinical findings.
- laboratory confirmation of hepatic disease and hepatic failure.
- liver biopsy.

3.4 CIRRHOSIS

This is not well documented but is not uncommon. End-stage pathology is the same as for other specific liver condition or for senile change. The most likely clinical presentation is chronic, progressive weight loss. Diagnosis is confirmed by biopsy. Supportive treatment may prolong life by weeks or months, but the prognosis is very poor.

3.5 LIVER FLUKE

Fasciola hepatica infection is rare in the horse, but donkeys may be more susceptible. Clinical signs of liver failure can occur, and it may be a group disease. Diagnosis can be confirmed by finding fluke eggs in the faeces, but immature, migrating parasites could cause signs prior to patency. Treatment is with triclabendazole, 15 mg/kg body weight *per os* (not licensed in the horse).

3.6 CHOLANGIOHEPATITIS AND CHOLEDOCHOLITHIASIS (BILIARY CALCULI)

Cholangiohepatitis is the most commonly encountered, clinically significant form of biliary tract disease in horses. The condition probably begins as a cholangitis, but extension into the periportal region of the liver normally follows (hence the term 'cholangiohepatitis' is usually used). It is probable that many mild cases of cholangitis/cholangiohepatitis are asymptomatic, but the condition predisposes horses to chronic, active, inflammatory hepatobiliary disease and the formation of biliary calculi (Figure 3.3). Chronic cholangiohepatitis may frequently be associated with significant intrahepatic or extrahepatic biliary calculus formation. Discrete calculi can often be visualized ultrasonographically or at post mortem examination, but some horses with cholangiohepatitis develop a sonolucent 'sludge-like' material within the biliary tract. With severe suppurative cholangiohepatitis significant periportal and bridging fibrosis can occur. Clinically significant hepatobiliary disease appears to be more common in middle-aged to old horses.

Clinical signs are non-specific, and include:

- fever.
- icterus.

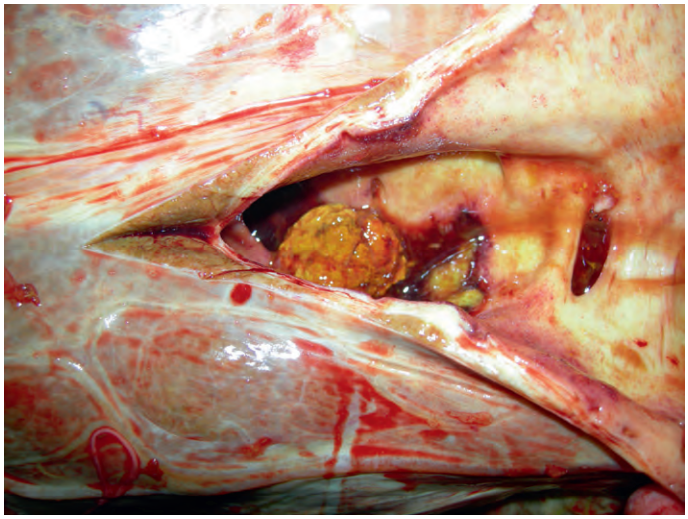


Figure 3.3 Large biliary calculus at post-mortem examination of a horse affected by cholangiohepatitis.

- colic.
- weight loss.
- encephalopathy.

Recurrent bouts of mild-to-moderate colic coincident with fever may occur. Significant weight loss will commonly accompany more chronic cases. Occasionally signs of hyperammonaemic hepatic encephalopathy can be seen when complete calculus obstruction to biliary outflow occurs or the disease process has progressed to hepatic failure. Serum biochemical abnormalities include:

- increases in the hepatobiliary enzymes GGT and AP, with moderate increases in the hepatocellular enzymes (AST and SDH).
- total serum bilirubin is elevated with the conjugated fraction representing more than 25 per cent of the total.
- bilirubinuria may also be observed.
- serum bile acids are usually elevated, and blood ammonia concentration may be raised in horses with complete calculus obstruction.

Typically, haematological changes are consistent with chronic, active inflammation and include neutrophilia and hyperfibrinogenaemia. If the condition is more than 2–3 weeks in duration, hyperglobulinaemia may also be documented. A definitive diagnosis of cholangiohepatitis requires liver biopsy.

Long-term (weeks to months) antimicrobial therapy is essential in the successful treatment of cholangiohepatitis and choledocholithiasis/hepatolithiasis in adult horses. In certain situations where biliary obstruction is complete, or the horse is in uncontrollable abdominal pain, surgery may be considered. The choice of specific antibiotics should be based upon both aerobic and anaerobic cultures of liver biopsy material. If biopsy culture results are either unavailable or negative, then broad spectrum antibiotics such as potentiated sulphonamides, cephalosporins, or fluoroquinolones would be appropriate choices.

3.7 CHRONIC ACTIVE HEPATITIS

Chronic active hepatitis is a descriptive term for a group of conditions characterized by active, progressive, inflammatory liver disease of long duration. The history is often one of depression, weight loss, and variable icterus. Signs are often intermittent and may be associated with fever. Some horses have a history of previous or active intra-abdominal disease. The disease can progress to the point of liver failure with major central nervous system involvement and death. Unusual cutaneous manifestations such as moist lesions at the coronary bands may be present. Liver lesions tend to be located in the periportal region, and the histopathological diagnosis is often cholangiohepatitis. The cause of chronic active hepatitis has not been determined, although autoimmune disease is possible. Alternatively, some cases may be a manifestation of chronic cholangitis.

Laboratory evaluation provides evidence of liver damage:

- elevation of liver-derived serum enzyme activities and marked elevation GGT and AP.
- serum bilirubin may be elevated with direct-reacting bilirubin comprising up to 40% of the total.
- the urine is positive for bilirubin.
- serum bile acids are elevated.

Haematological examination may show leukocytosis, left shift, and monocytosis. Total plasma protein concentration is generally elevated. Culture of liver biopsy specimens may be rewarding because bacterial agents may contribute to hepatitis or cholangitis.

Treatment involves general supportive care until horses regain their appetite. Corticocosteroids are often helpful; initial treatment should consist of 20–40 mg of dexamethasone given by injection. This dose rate is maintained for 3–5 days (depending on the response) and is then gradually decreased over the next 7–10 days. At this time the horse may be placed on oral prednisone at 1 mg/kg/day. Treatment may be necessary for 4–6 weeks or longer with careful monitoring of clinical signs and biochemical values. Bacterial infection may play a role, especially in horses with fever and a neutrophilic inflammatory infiltrate present on liver biopsy, and long-term (4–6 weeks) systemic administration of antibiotics is indicated.

3.8 IRON OVERLOAD (HAEMOCHROMATOSIS)

Newborn foals given an oral intestinal inoculum containing ferrous fumarate during the first day or two of life can develop acute liver failure due to iron overload. This is probably associated with an inability of the newborn foal to effectively regulate intestinal absorption of iron. Clinical signs develop within a few days with rapid progression of anorexia, depression, icterus, collapse, and death. Acute iron overload with liver damage has also been reported in a few adult horses given iron supplements orally.

Iron overload or haemochromatosis associated with chronic hepatic cirrhosis has been reported in adult horses. Clinical signs include depression, anorexia, weight loss, icterus, ventral oedema, and terminal hepatic encephalopathy. Liver-derived enzyme activities and serum bilirubin are increased. Histological lesions include disruption of hepatic architecture, bridging fibrosis, and bile duct hyperplasia. Iron accumulation is noted within hepatocytes, macrophages, and Kupffer's cells as indicated by Prussian blue staining. Unlike the condition in human beings, there is no evidence that the disorder is inherited. Since excessive dietary iron has not been a consistent feature in these horses, it has been suggested that for unknown reasons excessive intestinal iron absorption occurs with resultant accumulation of iron in the liver. It is possible that the accumulation of iron in the liver is the result of liver damage, and may not be the cause of liver failure.

3.9 HYDATID CYSTS

Cysts of the horse-dog tapeworm *Echinococcus granulosus equinus* are common in equine livers, but they are virtually never associated with clinical disease.

3.10 OTHER HEPATIC DISEASES

Various other liver diseases have been documented in the horse but should be regarded as uncommon. Other diseases may affect liver function, and some of these are described elsewhere in the text.

- Portosystemic shunts (Chapter 20).
- Liver abscess.
- Equine herpesvirus (Chapter 19).
- Biliary atresia.
- Mycotoxicosis.
- Hyperlipaemia (Chapter 9).
- Hepatic carcinoma/neoplasia.
- Lymphoma (see 3.16).
- Tyzzer's disease (Chapter 20).
- Right hepatic lobe atrophy.

3.11 TREATMENT FOR LIVER FAILURE

Treatment of liver failure is basically supportive. Treatment is most likely to be successful in acute hepatic failure and is not indicated in horses with severely cirrhotic livers. The prognosis is poor for severe hepatoencephalopathy.

- Sedation may be necessary in horses with signs of hepatoencephalopathy. Xylazine or detomidine administered in small doses is usually effective. Doses of sedatives that cause lowering of the head should be avoided if possible as low-head position and hypoventilation may worsen cerebral oedema. Phenobarbital can be used, but diazepam should be avoided since it may worsen hepatoencephalopathy.
- Intravenous fluids are probably the most important component of treatment for acute liver disease and hepatic encephalopathy. The intravenous fluids should consist of a balanced electrolyte solution, preferably without lactate, and should be supplemented with potassium 20–40 mEq/L, and 5–10 grams of dextrose per 100 mL. Sodium bicarbonate should be given only if blood pH is less than 7.1 and/or bicarbonate is less than 14 mEq/L. Additional potassium may be given as potassium chloride mixed in molasses and administered per os via a dose syringe. Fresh frozen plasma may be used, but hetastarch or stored whole blood should be avoided.
- Attempts should be made to decrease blood ammonia concentration.
  - *Mineral oil* is administered by nasogastric tube.
  - *Neomycin* can be administered at 5.0 mg/kg every 8 hours orally by dose syringe for 2 days.
  - *Lactulose* 0.2–0.5 mL/kg every 8–12 hours may also decrease ammonia production in the bowel and can be used concurrently with neomycin. Both lactulose and neomycin may cause diarrhoea if given in excessive dosages or for prolonged periods.
  - *Vinegar* (acetic acid) may also be effective in decreasing blood ammonia when it is administered per os at 240 mL/450 kg horse.
- Affected horses should be fed high-carbohydrate, high-branch-chain amino-acid (BCAA) feeds, with moderate to low total protein content. Sorghum and/or cracked corn mixed with molasses or commercially prepared BCAA paste are ideal. Carbohydrates should be fed frequently in small amounts. A grass hay with a moderate amount of protein, rather than alfalfa hay or spring-cut grass, should be fed.
- Affected horses should be protected from sunlight to prevent photosensitization.
- *Vitamin* B_1, *folic acid* and *vitamin* K_1 should be administered weekly.
- *Anti-oxidant, anti-inflammatory and anti-oedema therapy* is indicated in acute hepatic failure. The anti-oxidant, anti-oedema treatments include dimethylsulphoxide, acetylcysteine and mannitol given intravenously and vitamin E given intramuscularly. Anti-inflammatory therapy should include flunixin meglumine and pentoxifylline.
- *Antibiotics* are indicated in cases of bacterial cholangitis/cholangiotepatitis.
- Hyperlipaemia may be treated by administration of insulin and heparin (see Chapter 9).

Pancreatic diseases

3.12 PANCREATIC DISEASES

Clinical disease due to pancreatic disorders is rare in the horse. Non-specific signs of weight loss, intermittent colic, jaundice and fever can arise from either chronic pancreatitis or pancreatic adenocarcinoma. Both disorders may give rise to increased concentrations of serum amylase and lipase as well as hypocalcaemia. Pancreatic diseases may also be associated with raised liver enzymes and hyperbilirubinaemia due to either biliary obstruction or pancreatic production of gamma glutamyl transferase (γGT) which is regarded as a 'liver-specific' enzyme. Confirmation of chronic pancreatitis or pancreatic carcinoma is by either exploratory celiotomy or post-mortem examination. Acute pancreatitis is a very rare cause of severe abdominal pain and is characterized by nasogastric reflux and circulatory failure such that it must be differentiated from strangulating intestinal obstruction and anterior enteritis.

Equine grass sickness

3.13 EQUINE GRASS SICKNESS (EQUINE DYSAUTONOMIA)

Grass sickness is a neurological disease associated primarily with degeneration of neurons of the autonomic nervous system (in particular the enteric nervous system) (dysautonomia) which is typically manifest by gastrointestinal signs. It is hypothesized but not yet proven that the aetiology of grass sickness (*equine dysautonomia*) is a toxicity; the current leading hypothesis is that the disease results from a toxicoinfection with *Clostridium botulinum* types C and D (i.e. the toxin is produced locally within the intestinal tract by resident bacteria). The disease is fairly common in regions of UK (the North East region of Scotland has the highest prevalence of the disease) and parts of northern continental Europe. A virtually identical condition (known as *mal seco*) occurs in South America.

Epidemiology

All equidae appear to be susceptible to grass sickness. There is no breed predisposition. Horses in good to fat body condition may be particularly at risk. The disease affects grazing horses – it is extremely rare in horses that have no access to grass.

Regional prevalence occurs with certain localities having higher risk for the condition, and the incidence within regions varies from year to year. Horses in large number on a premise and young horses are at increased risk. Horses pastured on sandy and loam soil are at increased risk.

Age prevalence peaks between 2 and 7 years old, but the disease can occur at any age.

Seasonal prevalence exists with increased occurrence in April–July which probably relates to periods of dry, cool weather (7–11 °C, 45–62 °F).

Grass sickness is more prevalent in horses introduced to premises within the last 2 months and/or which are outdoors all the time and/or which are not receiving supplementary feeding. Frequently more than one horse is affected on same premises, and the risk of grass sickness is much greater if horses graze pasture on which affected horses have grazed.

Other risk factors for grass sickness include recent disturbance of the pasture (e.g. construction or mole activity), mechanical removal of faeces from the pasture, pasture cutting, and high frequency use of anthelmintics (especially use of ivermectin for the most recent treatment).

Pathogenesis

Widespread and generally severe damage to neurons of the enteric autonomic nervous system and certain somatic ganglia and nuclei of the central nervous system with resultant disruption of gastrointestinal motility.

Clinical signs

The disease is arbitrarily divided into three forms depending on the severity of the disease and the time period until death. Acute (death in <4 days), sub-acute (4–10 days) or chronic (week to months) forms occur.

Acute form

The acute form is characterized by rapid onset and progression of clinical signs, with death often occurring in less than 48 hours.

- Depression/somnolence.
- Anorexia.
- Dysphagia.
- Colic (moderate to severe).
- Absence of gut sounds.
- Tachycardia (heart rate 70–80/min, or more).
- Congestion and dryness of mucous membranes.
- Dehydration.
- ± Patchy sweating.
- ± Muscle fasciculations/tremors.
- Nasogastric reflux – occasionally spontaneous appearance of green fluid at the nostrils or several gallons foetid yellow/green fluid obtained via stomach tube.
- Gastric rupture may occur.
- Crusty nasal discharge/rhinitis.
- *per rectum* – dry, tacky rectal mucosa; meagre rectal contents which are hard with mucosal casts on surface. Large intestine (especially large colon) is shrunken down onto hard, knobbly, impacted contents. Distended, fluid-filled loops of small intestine extending toward the pelvic inlet may be palpable.

Sub-acute form

The clinical signs of the sub-acute form are similar but less severe than the acute form. The duration of clinical signs is longer, and the abdomen adopts a tucked-up appearance.

Chronic form

The onset of clinical signs in the chronic form is insidious. Severity of signs varies from day to day.

- Dullness/hypoaesthetic/sleepy.
- Reduced intestinal motility.
- Marked weight loss/emaciation (Figure 3.4).
- Minimal faecal production – faeces hard and dry.
- Marked reduction of abdominal size – 'greyhound-like'.
- Tachycardia.
- Crusty nasal discharge (*Rhintis sicca*).
- ± Ptosis.
- ± Abnormal stance – four feet placed closely together.
- ± Penile protrusion.
- ± Patchy sweating.
- ± Muscle fasciculation/tremor.
- ± Intermittent, mild colic.

Diagnosis of acute / sub-acute / chronic forms

- Clinical signs.
- Contrast radiography: prolonged (>5 secs) oesophageal transit time of barium given into proximal oesophagus. Also may see barium pooling or retrograde movement.
- Oesophageal endoscopy – linear ulcerations (reflux oesophagitis) (Figure 3.5).
- Phenylephrine eyedrops – eyelid elevation (compare with the drooping eyelid in the contralateral eye).
- Definitive diagnosis requires histopathology: neuronal vacuolation and chromatolysis with variable inflammatory infiltrate. Usual material for histopathologic examination is either coeliacomesenteric ganglion collected at post-mortem examination or ileal biopsy (at either exploratory celiotomy or post-mortem examination). Similar pathological changes occur in other autonomic ganglia as well as in brain and spinal cord.

Post-mortem examination

Acute and sub-acute form

- Gastric distension with foetid fluid.
- Gastric/oesophageal mucosal ulceration.
- Large colon impacted with dark, dry, hard content.

Chronic form

- Emaciated carcass.
- Marked reduction of intestinal size.
- Dry, firm, large intestinal content.

Treatment

- Euthanasia.
- Selected chronic cases may survive given nursing/supportive therapy and use of the intestinal prokinetic agent, cisapride.

Prevention

It might help to keep equine horses indoors for at least part of every day, and/or feed supplementary roughage, during periods of peak incidence. This applies particularly to young or newly arrived horses, or when a case of grass sickness has already occurred on the premises.

Figure 3.4 Chronic grass sickness. Emaciation and tucked-up abdomen, penile protrusion and abnormal base-narrow stance ('elephant on a tub').

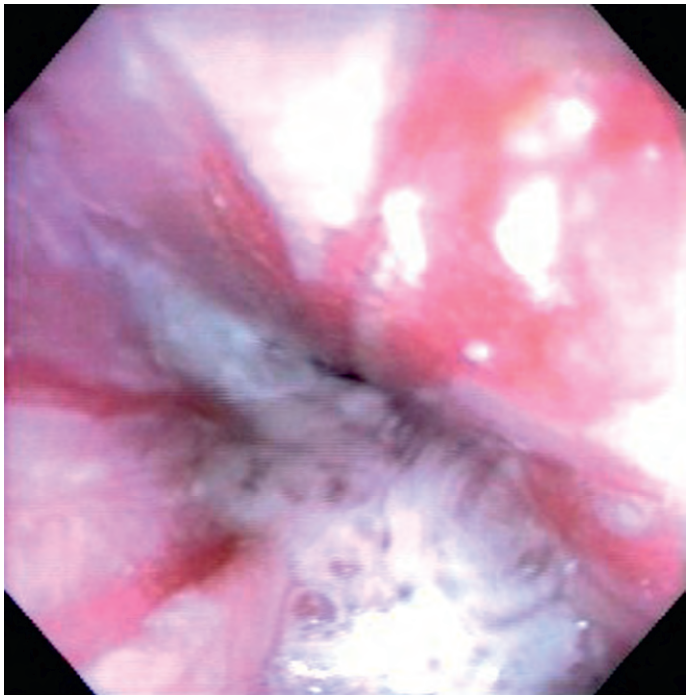


Figure 3.5 Endoscopic view of the oeseophagus of a horse with sub-acute grass sickness showing severe oesophagitis and linear ulceration due to reflux of gastric contents.

Diseases of the stomach (equine dysautonomia)

3.14 GASTRIC ULCERATION (EQUINE GASTRIC ULCER SYNDROME)

Epidemiology

Gastric ulceration can occur in any breed or age of horse but is most common in race horses (estimated to occur in >90%) and performance horses. Risk factors include:

- High concentrate / low roughage diet.
- Stress – e.g. racing, training, showing, transport.
- Other gastrointestinal diseases or illness.
- Anorexia or withholding feed.
- Non-steroidal anti-inflammatory drug therapy.

Pathogenesis

Hydrochloric acid is continuously secreted by the parietal cells in the gastric glandular epithelium. The gastric mucosa is typically protected from the acidic gastric contents by several mechanisms, including:

- A mucus/bicarbonate barrier.
- Gastric mucosal blood flow supported by prostaglandins.
- Eating, which raises gastric pH due to alkaline saliva and adsorption of gastric secretions by ingested roughage.

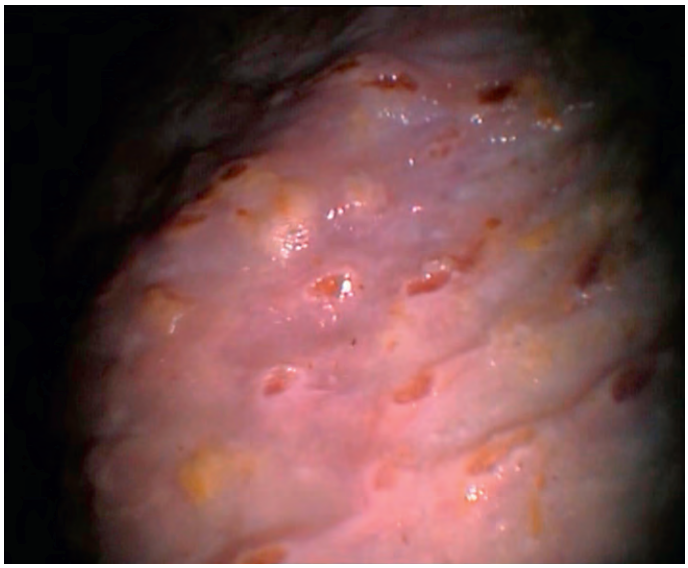


Figure 3.6 Gastric ulceration – endoscopic appearance of grade 1 ulcers.

Anything that disrupts these protective mechanisms can result in gastric ulceration. The squamous mucosa, especially that adjacent to the margo plicatus, is most commonly affected (80%); the glandular mucosa is less commonly involved (20%). Ulceration in the cardia and distal oesophagus is associated with gastro-oesophageal reflux and pyloric outflow obstructions.

- High-concentrate diets, infrequent feeding and withholding of feed or anorexia result in prolonged periods of low gastric pH, resulting in damage to the poorly protected squamous mucosa.
- NSAID therapy (by decreased production of protective prostaglandins).
- Intense exercise and systemic illness can result in alterations in mucosal blood flow, resulting in both squamous and glandular lesions.

Clinical signs

The clinical signs of gastric ulceration in the adult are variable.

- Many adult horses have no clinical signs, even with severe gastric ulceration.
- Inappetence.
- Colic – usually mild and may be associated with feeding.
- Bruxism or hypersalivation.
- Mild depression.
- Weight loss if chronic or severe ulceration.
- Rough hair coat.
- Behaviour and/or performance may change (not consistent feature).

Diagnosis

Diagnosis is achieved by gastroscopy (Figure 3.6) or response to specific treatment.

A 2–3 metre flexible endoscope is necessary to fully evaluate the stomach in adult horses and most ponies. Fasting for 12–24 hours is required prior to gastroscopy to ensure sufficient visualization of the gastric mucosa.

Gastric ulcers are often graded on a scale of 0 to 3:

- 0 = normal (no ulceration).
- 1 = mild ulceration with single or multifocal hyperaemic areas or small superficial ulcers.
- 2 = moderate ulceration, with moderately sized single or multifocal ulcers.
- 3 = severe ulceration, with large multifocal, coalescing or diffuse deep, often haemorrhagic, ulcers.
- Hyperkeratosis of the squamous mucosa in the absence of active ulceration is consistent with previous gastric ulceration.

Treatment

- Decrease or discontinue NSAID therapy if at all possible.
- Decrease or eliminate dietary concentrate and permit access to pasture if possible.
- Gastric acid suppression:
  1. Proton pump inhibitors:
     - Omeprazole (4 mg/kg PO q 24 hours). Very effective (>95% success rate).
       Requires up to 72 hours to effectively increase gastric pH. A lower dose (2 mg/kg PO q 24 hours) may be effective for long-term preventative therapy.
  2. Histamine H2-receptor antagonists.
     - Cimetidine (16–25 mg/kg PO or 6.6 mg/kg IV q 6–8 hours). Inhibits hepatic cytochrome p450 oxidase and thus alters metabolism of other drugs; use with caution in patients on concurrent medications.
     - Ranitidine (6.6 mg/kg PO or 1.5 mg/kg IV q 8 hours).
     - Famotidine (2.8–4 mg/kg PO or 0.23–0.5 mg/kg IV q 8–12 hours).
       Due to the need for more frequent administration, these agents are most useful to rapidly decrease gastric pH during the initial 72 hours of proton-pump inhibitor therapy, but may also be effective when used alone.
  3. Antacids (magnesium hydroxide, aluminium hydroxide) – require frequent administration (q 2–4 hours) to have any effect on raising gastric pH, thus not recommended.
     - Mucosal protectants.
       Sucralfate (20 mg/kg PO q 6–8 hours). Adheres to ulcerated mucosa, providing protection from gastric acid and stimulates local production of protective prostaglandins and cytokines to promote mucosal healing. Misoprostol (2.5–5 µg/kg). A PGE_2 analogue, which inhibits acid secretion, promotes mucosal blood flow, and enhances bicarbonate and mucus production. Useful in NSAID-induced gastric ulceration. Side-effects include abdominal pain and diarrhoea. Should not be used in pregnant mares.

3.15 GASTRIC IMPACTION

Gastric impaction is uncommon but may occur as a result of:

- Feeding poor quality roughage or certain feed stuffs like beet pulp and wheat bran, which may not be adequately hydrated by saliva and gastric fluid contents.
- Poor dentition resulting in inadequate mastication.
- Concurrent gastrointestinal disease, resulting in generalized decreased gastrointestinal motility.
- Pyloric outflow obstruction.
- Horses with acute or chronic hepatic disease have an increased incidence of gastric impaction, though the direct relationship between the two conditions is poorly understood.
- Bezoar formation. Persimmon seed or hair ingestion can result in formation of a phytobezoar or trichobezoar respectively. A large bezoar may obstruct the pylorus, or a small bezoar may obstruct the duodenum, resulting in impaired gastric outflow.

Figure 3.7 Gastric impaction – appearance at post-mortem examination.

Clinical signs of gastric impaction include inappetence/anorexia and colic (of variable severity). Idiopathic gastric impaction occasionally occurs as a primary cause of colic in the absence of a specific predisposing or concurrent cause (Figure 3.7).

Definitive diagnosis is difficult and is often made during exploratory celiotomy due to protracted and unrelenting pain. Trans-abdominal ultrasonography may be helpful (caudal displacement of the gastric contour). Gastroscopy does not allow accurate determination of gastric size, although identification of feed material at the level of the cardia in a fasted horse is consistent with a gastric impaction. The persistence of a large amount of feed material on serial examinations over 24–36 hours or longer in a fasted horse is needed for a presumptive gastroscopic diagnosis of gastric impaction.

Treatment

1. Withhold all feed and limit water intake (risk of gastric rupture)
2. Medical therapy:
   - Promote hydration of gastric contents and breakdown of the impaction:
     Intravenous fluid therapy.
     Intragastric administration of isotonic fluids and laxatives (small volumes via gravity flow).
     Administration of 0.5 to 1 L of Coca-cola™ via nasogastric tube every 12–24 hours can aid the breakdown of impacted material in both feed impactions and persimmon phytobezoars.
   - Gastric lavage via a large bore nasogastric tube.
   - Analgesic therapy.
   - Prokinetic therapy is not recommended unless a pyloric or intestinal obstruction has been ruled out.
3. Surgery. Surgical resolution of a gastric impaction or removal of a gastric bezoar via gastrotomy is extremely challenging in adult horses due to limited surgical exposure and inability to exteriorize the stomach, resulting in substantial risk for abdominal contamination at surgery.

3.16 GASTRIC NEOPLASIA

Gastric neoplasia is rare. The commonest neoplasm found at this site is squamous cell carcinoma.

Typical signs include:

- Chronic weight loss.
- Poor appetite.
- Abdominal discomfort – recurrent colic associated with feeding may occur.
- Lethargy.
- Ascites or oedema may occur in some cases.
- If the oesophagus or cardia is involved, dysphagia or ptyalism is the predominate sign.
- Metastasis to the thoracic cavity may cause tachypnoea and dyspnoea.

Many horses with gastric squamous cell carcinoma have anaemia, leukocytosis, and hyperfibrinogenaemia. Some have hypoproteinaemia due to bowel inflammation and protein exudation, whereas other cases will have hyperglobulinaemia. Gastroscopy may reveal a mass within the stomach protruding from the mucosa. Biopsy of the mass may be possible via the endoscope. Squamous cell carcinoma originates from the squamous mucosa and typically has the appearance of an ulcerated, cobblestone or florid mass. Ultrasonography, laparoscopy, and celiotomy can be used to further evaluate the patient.

Treatment of gastric squamous cell carcinoma in the horse is rarely possible.

Intestinal disorders

The most common clinical disorders of the gastrointestinal tract are those giving rise to signs of acute abdominal pain or colic which are dealt with in Chapter 2. There are other clinical manifestations of intestinal dysfunction in the horse of which the most important are *acute diarrhoea or chronic weight loss and/or chronic diarrhoea*. The presence of diarrhoea in an adult horse almost invariably indicates large intestinal disease whereas weight loss may be associated with disorders affecting either the small intestine or the large intestine or both.

The principal pathological processes of intestinal disease are *protein losing enteropathy and/or carbohydrate malabsorption*. If only the latter is present, the clinical presentation will usually be of insidious weight loss, whereas with protein losing enteropathy (PLE) there will usually be fairly rapid weight loss and commonly peripheral oedema will develop consequent upon hypoproteinaemia/hypoalbuminaemia. In acute disorders, fever, cardiovascular compromise and coagulopathy associated with endotoxaemia are common. Additional clinical signs which may be present in gastrointestinal disease include altered intestinal motility, anorexia, ptyalism, bruxism, dysphagia and, rarely, skin lesions.

A diverse group of gastrointestinal conditions may give rise to diarrhoea and/or weight loss but often these diseases cannot be readily differentiated on clinical findings. In practical circumstances the clinical management of such cases is often similar regardless of the diagnosis and is directed at symptomatic antidiarrhoeal medication and fluid/electrolyte/nutritional support. However, the prognosis for these conditions varies greatly, e.g. successful outcome following treatment of cases of larval cyathostominosis occurs in approximately 50% of cases whereas there is virtually 100% mortality in cases of intestinal lymphosarcoma. For this reason a specific diagnosis is preferable but unfortunately that can generally only be achieved by histopathological examination of intestinal biopsy obtained at exploratory celiotomy/laparoscopy (or rectal biopsy in a minority of cases). Horses with hypoproteinaemia secondary to protein losing enteropathy are high risk for wound dehiscence and this, together with the high cost of equine abdominal surgery, means that a specific diagnosis is often only achieved at postmortem examination.

3.17 DIAGNOSTIC APPROACH TO INTESTINAL DISEASE

History

1. Age – foals and yearlings are more susceptible to gastrointestinal infections such as rotavirus, *Lawsonia intracellularis* (proliferative enteropathy) and salmonellosis (Chapter 19). Larval cyathostominosis (Chapter 19) is more common in horses less than four years old, and grass sickness is also more prevalent in this age category. Chronic inflammatory bowel disease (CIBD) and intestinal neoplasia are more common in horses greater than ten years old.
2. Multiple cases are suggestive of an infectious cause, e.g. acute enterocolitis in salmonellosis (Chapter 19) or weight loss/diarrhoea of larval cyathostominosis or proliferative enteropathy (Chapter 19).
3. Duration/progression – peracute/acute systemic illness due to endotoxaemia occurs in various enterocolitides and also peritonitis. Cyathostominosis cases tend to have sudden onset, but rarely have marked systemic illness, and tend to have a timecourse of a few weeks. CIBD and intestinal neoplasia are generally insidious, with a duration of weeks to months.

Physical examination

There are virtually no characteristic physical findings for individual enteropathies, and the clinical approach is to rule out other disorders which may be associated with weight loss, e.g. liver disease with evidence of hepatic encephalopathy, then to undertake clinicopathological investigation relevant to intestinal dysfunction.

Colic, reduced borborygmi and cardiovascular compromise consequent upon endotoxaemia occur in acute enterocolitis and peritonitis which, therefore, must be differentiated from strangulating intestinal obstruction, non-strangulating infarction etc. (Chapter 2).

Various musculoskeletal signs which might be useful for differential diagnosis may be detectable in certain disorders primarily manifest as gastrointestinal in nature. In chronic grass sickness, cases often adopt a characteristic stance with all four feet placed close together under the trunk, and such horses will often exhibit intermittent muscle fasciculations. Due to parietal abdominal pain, cases of peritonitis are often reluctant to move and do so with a rather 'wooden' gait. This feature should be differentiated from endotoxaemia-associated laminitis which can complicate both peritonitis and also enterocolitis.

Laboratory investigation

Blood biochemistry/haematology. There are no specific markers of gastrointestinal disease. The most relevant analyses to measure are total protein, albumin, globulin, alkaline phosphatase, fibrinogen and a complete blood count (CBC) and differential. In acute gastrointestinal disease, hydration status, acid-base balance and electrolyte (K, Na, Cl) disturbances should be assessed, together with renal function (creatinine, urea and phosphate). Serum protein electrophoresis is sometimes useful for differentiation of parasitic colitis (cyathostominosis) from other enteropathies. The level of abnormality of analytes such as total protein, albumin, alkaline phosphatase and neutrophil concentratons relate to the severity of chronic enteropathies such that they may be helpful in assessing prognosis.

Peritoneal fluid cytology/biochemistry. Abdominocentesis is very useful in assessment of acute abdominal disorders (Chapter 2) but of limited value in chronic conditions. Peritonitis can be confirmed by detection of peritoneal fluid white cell count >5 × 10^9/L, total protein >10 g/L and alkaline phosphatase of >250 IU/L. Intestinal neoplasia and CIBD rarely exfoliate nucleated cells into peritoneal fluid in detectable numbers.

Intestinal function tests. Small intestinal absorptive dysfunction can be accurately detected by monosaccharide absorption tests, usually using glucose or, less commonly xylose. The protocol for the oral glucose tolerance test is:

- overnight fast and basal plasma glucose measurement time 0 min.
- glucose (20% solution) by stomach tube at dose of 1 g/kg body weight.
- plasma glucose measurement at times 30, 60, 90, 120, 180, 240, 360 min.

'Normal' small intestinal function is evidenced by a peak of plasma glucose at 120 min of 185-200% of basal level and return to basal levels by 360 min. A peak value of less than 120% basal value indicates total carbohydrate malabsorption and an increase of between 120 and 185% is classified as partial malabsorption.

Xylose absorption should give a similar shaped curve, but interpretation is by comparison with established reference ranges.

Detection of carbohydrate malabsorption confirms the presence of small intestinal disease, but the tests are not useful for differentiating the possible causes.

Histopathology. Chronic enteropathies can only be definitively diagnosed on the basis of histopathological features of biopsy material obtained by either celiotomy, rectal biopsy or post-mortem examination. The surgical risk and expense are high in horses with PLE, and surgery is often not undertaken. It is possible to obtain mucosal biopsies of the proximal duodenum by endoscopy, although the value of this technique in diagnosing inflammatory bowel diseases is currently uncertain. Rectal biopsy is safe and simple from intrapelvic sites on the lateral rectum: diagnostic samples are obtained in about one third of chronic enteropathy cases.

Imaging modalities. Ultrasonography and laparoscopy are occasionally useful for investigation of horses presented with possible gastrointestinal disease, mainly to rule out rare conditions such as splenic neoplasia or urogenital disease and to identify non-specific features such as intestinal wall thickening (Figure 3.9).

3.18 CHRONIC INFLAMMATORY BOWEL DISEASE

Chronic inflammatory bowel disease (CIBD) is a collective term for a group of chronic infiltrative enteropathies with very similar clinical presentations (usually of chronic weight loss) and clinical signs. These disorders are not as clearly defined in the horse as they may be in other animal species and humans. The major clinical differential diagnoses are intestinal neoplasia and chronic hepatopathies.

Epidemiology

Generally CIBD occurs spontaneously in individual, middle aged to old horses of any breed, but it has been suggested that granulomatous enterocolitis is more common in Standardbreds less than 5 years of age.

Pathogenesis

The aetiopathogenesis of equine CIBD is uncertain but may represent immune-mediated phenomena. Various infectious agents may play a role in these enteropathies including strongyle parasites, *Mycobacterium paratuberculosis* (granulomatous enteritis), *Rhodococcus equi* (granulomatous enteritis) and *Histoplasmum capsulatum* (granulomatous colitis).

Clinical signs

- Weight loss (Figure 3.8).
- ± Diarrhoea.
- ± Intermittent colic.
- ± Inappetence.
- ± Peripheral oedema.
- ± Pyrexia.
- ± Skin lesions.
- ± Mesenteric lymph node enlargement.

Figure 3.8 Marked weight loss and ventral (preputial) oedema due to small intestinal malabsorption.

Diagnosis

Clinical pathological findings are non-specific but may include:

- Hypoalbuminaemia.
- Hyperglobulinaemia.
- Neutrophilia.
- Anaemia.
- Hyperfibrinogenaemia.
- Raised serum alkaline phosphatase.
- Reduced glucose absorption (OGTT).

Trans-abdominal ultrasound may reveal diffuse or focal mural thickening of small intestine (Figure 3.9) (sometimes with concurrent large intestinal thickening). Definitive diagnosis is based on histopathological examination of intestinal (or rectal) biopsies (Figure 3.10). The infiltrate may constitute a mixed cellular population, or there may be a predominance of specific cell types such that CIBD may be classified as either granulomatous enteritis/colitis or eosinophilic gastroenteritis, eosinophilic granulomatous enteritis or lymphocytic plasmacytic enteritis. Similar pathological findings are commonly identified within mesenteric lymph nodes and also occasionally in skin lesions. The condition described as multisystemic eosinophilic epitheliotropic disease may have gastrointestinal involvement as well as cutaneous, hepatic and pancreatic lesions.

The major differential diagnoses for CIBD are alimentary lymphoma (lymphosarcoma), larval cyathostominosis, and chronic hepatopathies.

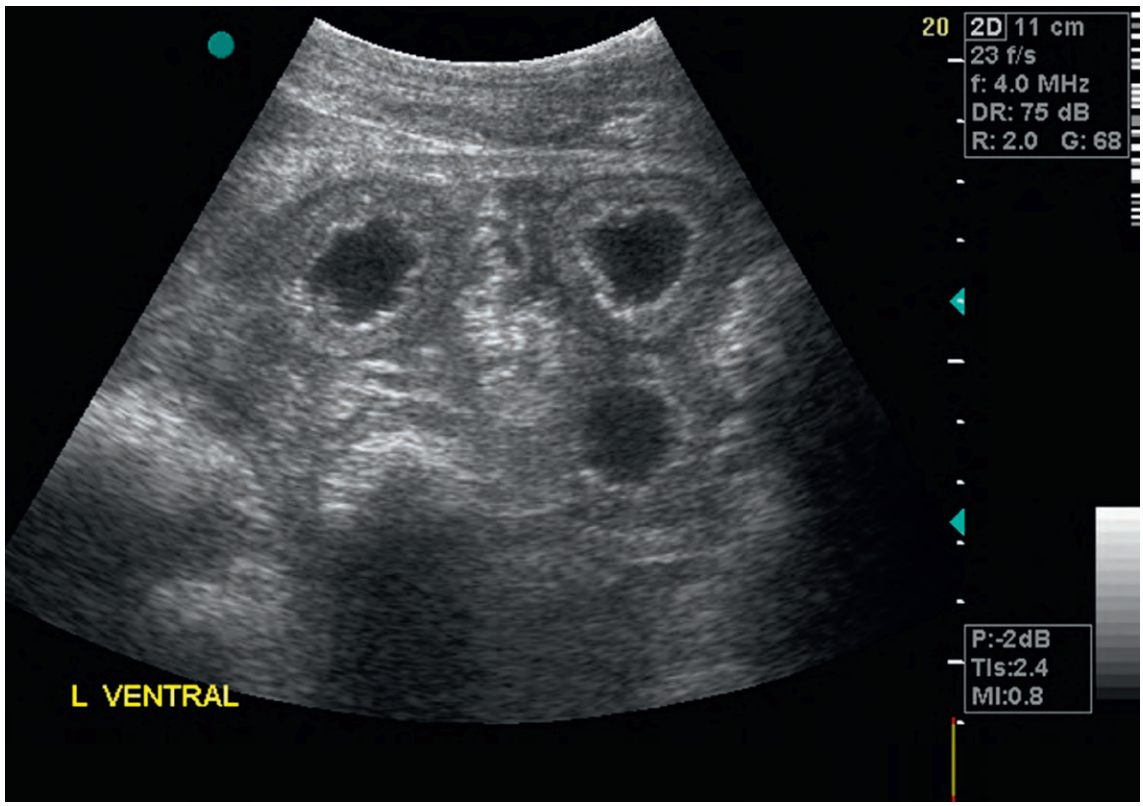


Figure 3.9 Trans-abdominal ultrasonography showing thickened loops of small intestine due to inflammatory bowel disease.

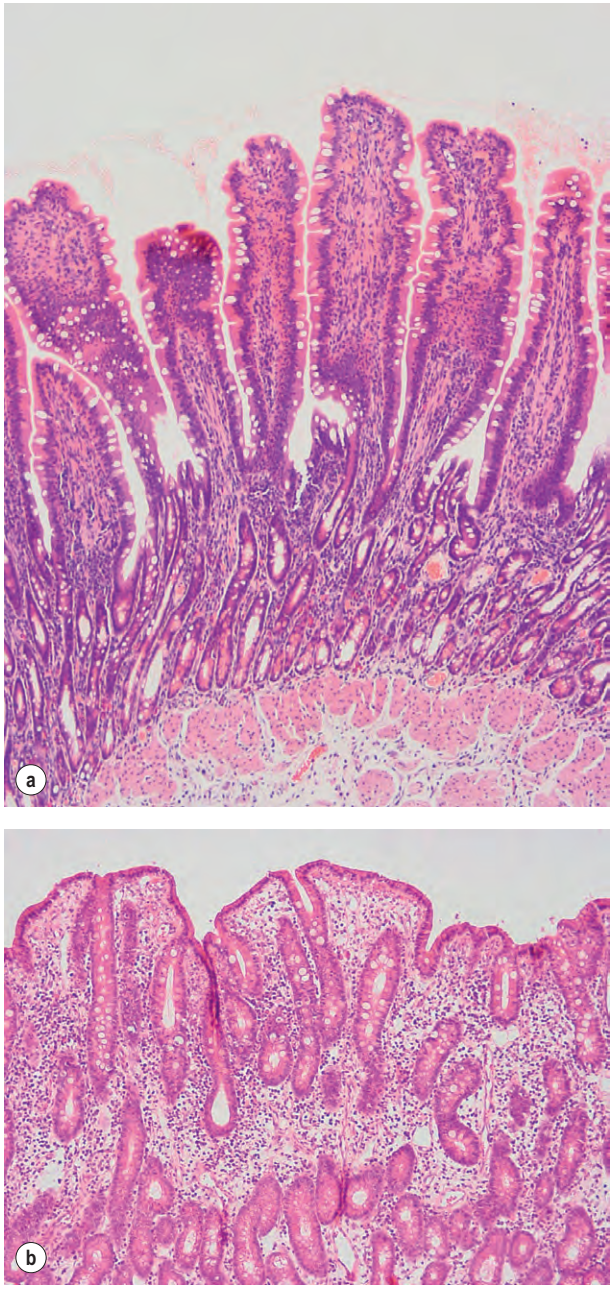


Figure 3.10 (a) Histological appearance of normal small intestine showing the long, finger-like villi. (b) Histological appearance of small intestine affected by granulomatous enteritis – there is an inflammatory cell infiltrate in the mucosa and submucosa with blunting and thickening of the villi.

Treatment

Long-term therapy with corticosteroids may result in clinical improvement during the course of treatment, but clinical relapses occur commonly, but not invariably, following cessation of medication. Parenteral corticosteroid treatment is usually more effective than oral treatment at least initially – dexamethasone (0.1 mg/kg iv or im q 24 hours) followed by oral prednisolone (1–2 mg/kg po q 24 hours). If a good response to the above therapy is observed, with improvement in serum protein concentration and weight gain, steroid therapy may be tapered slowly over 6–12 weeks, with a goal of reaching the lowest possible dosage that controls the horse's signs. If corticosteroids are ineffective, azothiaprine (2–3 mg/kg po q 24 hours) alone or in combination with corticosteroids may be effective. Occasionally, horses respond well to corticosteroid therapy and remain in remission following discontinuation of treatment. Unfortunately, the majority of horses, however, either do not respond sufficiently or require lifelong corticosteroid therapy.

Hay and fibrous feed may be poorly tolerated in many horses with CIBD. Maintainence on good quality pasture and a well-balanced complete pelleted feed with high protein supplementation may be effective.

In other species, allergy testing and exclusion diets can be helpful in determining the role dietary antigens may play in CIBD. In horses, designing an effective yet nutritionally balanced exclusion dietary regime is very difficult.

Other symptomatic treatments which may be useful include antidiarrhoeal agents such as codeine phosphate and/or anthelmintics and/or probiotics.

Prognosis

Sustained clinical improvement is rarely achieved, and many cases require euthanasia on humane grounds.

3.19 ALIMENTARY LYMPHOMA

Alimentary lymphoma is more common than either thoracic or cutaneous lymphoma (see Chapters 6 and 13), and most cases are presented with insidious weight loss, such that the major differential diagnoses are other chronic hepatopathies or chronic enteropathies.

Epidemiology

The disease may occur in young adults (2 to 6 years) but is apparently more common in horses more than 10 years old.

Pathogenesis

No infectious agent has been incriminated in equine lymphoma. Diffuse infiltration of the small intestinal (sometimes large intestinal) wall is the most frequent manifestation. The disease can result in both small intestinal carbohydrate malabsorption and/or protein losing enteropathy. In some cases, discrete masses of the intestinal wall or associated mesentery may occur, resulting in focal intestinal obstruction.

Clinical signs

- Weight loss.
- ± Inappetence.
- ± Intermittent/recurrent colic.
- ± Peripheral oedema.
- ± Diarrhoea.
- ± Pyrexia.
- ± Colic.
- ± palpable mesenteric lymph node enlargement.

Diagnosis

Clinical pathological findings are non-specific but may include:

- Hypoalbuminaemia.
- Hyperglobulinaemia.
- Neutrophilia.
- Anaemia.
- Hyperfibrinogenaemia.

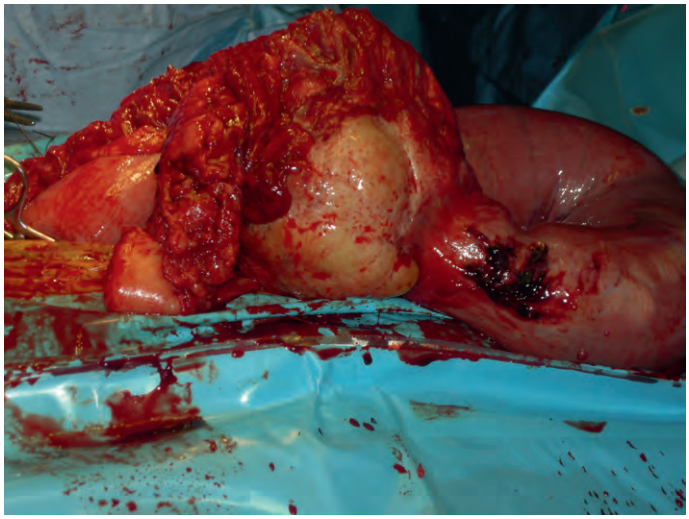


Figure 3.11 Intra-operative view of a focal small intestinal lesion due to lymphoma.

- Raised serum alkaline phosphatase.
- Reduced glucose absorption (OGTT) and xylose absorption.
- Peritoneal fluid cytology – rarely detect tumour cells.
- Hypercalcaemia in some cases.
- Decreased serum concentrations of IgM (and less commonly IgA or IgG) in some cases.

Abdominal ultrasonography and exploratory celiotomy/laparoscopy may be helpful. Definitive diagnosis is based on histopathology of intestinal (or rectal) biopsies.

Treatment

Temporary improvement can be achieved with palliative, symptomatic medication with either orally or systemically administered corticosteroids (dexamethasone, prednisolone), and antidiarrhoeic agents, such as codeine phosphate. Cytotoxic therapy for treatment of equine alimentary lymphoma has not been reported.

Surgical excision of focal lymphoma masses within the intestinal wall may occasionally be curative (Figure 3.11).

Prognosis

The prognosis is extremely poor – most cases are euthanased on humane grounds within 6–12 months of onset of signs.

3.20 SAND ENTEROPATHY

Accumulation of sand in the gastrointestinal tract is a well-recognized but fairly uncommon cause of diarrhoea and/or colic.

Epidemiology

The horses at risk of developing sand enteropathy are those grazing areas of sandy soil with little pasture cover (or horses turned out into sand arenas), and the risk is exacerbated if grain is fed from the ground to such horses. There is a relatively high prevalence in certain geographical regions, e.g. California, New Jersey.

Pathogenesis

Sand accumulates within the large colon and gives rise to either colonic irritation and/or physical obstruction which results in diarrhoea and/or colic.

Clinical signs

- Inappetence.
- ± Diarrhoea.
- ± Colic.
- ± (Rarely) weight loss.

Diagnosis

- Per rectum detection of sand impaction.
- Visualization of sand sediment from faecal solution.
- 'Pouring sand' sounds on abdominal auscultation of ventral abdomen.
- Abdominal radiography.
- Abdominal ultrasonography.

Treatment

- Bulk laxatives (especially psyllium hydrophilia), initially via nasogastric tube and then in-feed.
- Mineral/vegetable oils via nasogastric tube.
- Parenteral fluid therapy.
- (Rarely) surgical removal of impacted sand.

Prognosis

The prognosis is generally good following aggressive laxative therapy, although not all cases respond. Recurrence and/or cases in other horses under the same management can be prevented by avoiding overgrazing of pastures on sandy soil and feeding from fixed troughs.

3.21 IDIOPATHIC CHRONIC DIARRHOEA

In the horse, the cause of chronic diarrhoea often remains undiagnosed despite extensive investigative procedures.

Epidemiology

Some cases of idiopathic diarrhoea may be consequent upon earlier infectious enteropathies and/or fermentation disorders. Particularly in foals and yearlings, diarrhoea may persist for several months before resolution which can be associated with a change to a pasture-based diet.

Pathogenesis

By definition the pathogenesis of these cases is not known.

Clinical signs

- Diarrhoea.
- ± Weight loss.

Diagnosis

Diagnosis is by exclusion of identifiable causes of diarrhoea.

Treatment

Symptomatic therapy can be used with variable clinical effect – recurrence of diarrhoea is not uncommon. Symptomatic antidiarrhoeals (e.g. codeine phosphate, iodochlorohydroxyquin) administered concurrently with either probiotics or natural yoghurt or transfaunated caecal contents are appropriate. Absorbents such as chalk, kaolin or activated charcoal are only useful in foals.

Prognosis

The prognosis is generally good for survival, but recurrent, chronic diarrhoea may persist.

3.22 ACUTE ENTEROCOLITIS

The term incorporates a variety of large intestinal disorders with very similar presenting and clinical signs of acute onset diarrhoea and colic. Causes of enterocolitis include salmonellosis (also see Chapter 19), Potomac horse fever (i.e. *Neorickettsia risticii* infection, Chapter 19), clostridiosis (see Chapter 19), colitis X, antimicrobial therapy, non-steroidal anti-inflammatory drug (NSAID) therapy, and toxicities such as either monensin/salinomycin or heavy metals. These disorders cannot be readily differentiated on the basis of clinical features, and often there is rapid clinical progression with predisposition to sequelae of endotoxaemia and systemic inflammatory response syndrome, including circulatory collapse, laminitis and thrombophlebitis.

Epidemiology

- Salmonellosis – increased risk in foals, stressed/hospitalized horses and those receiving antimicrobials: 'outbreaks' may occur.
- Potomac horse fever – regional occurrence – Eastern USA. Probably involves an insect vector.
- Clostridiosis – associated with *Clostridium difficile* or *Clostridium perfringens* infection. Initiating factors similar to salmonellosis, especially antimicrobial therapy for *C. difficile*.
- Colitis X – peracute colitis, endotoxaemia and anaphylaxis of unknown aetiology. Typically involves single isolated cases rather than outbreaks.
- Antimicrobial therapy – disruption of intestinal flora leading to diarrhoea has been associated with either lincomycin or tetracycline therapy, but other antibiotics can have similar effects.
- NSAID toxicity – excessive or prolonged dosages and/or dehydrated horses.
- Monensin/salinomycin toxicity – ingestion of contaminated feedstuff.
- Heavy metal toxicity – rare.

Pathogenesis

- Salmonellosis – invasion of the primary pathogen; intestinal mucosal colonization results in inflammatory protein losing enteropathy (PLE) and endotoxaemia.
- Potomac horse fever – invasion of a primary pathogen; initial infection of peripheral monocytes and macrophages with later phase of infection within small intestinal and colonic mucosa. As a result there may be several days of fever and colic prior to onset of diarrhoea.
- Clostridiosis – overgrowth by potentially pathogenic normal inhabitants of the intestinal tract (normally present in low numbers); marked colonic oedema and PLE.
- Colitis X – the cause remains unknown although an infectious or toxic aetiology is assumed. Some cases may be forms of clostridiosis. Intestinal anaphylaxis to an unidentified antigen may be involved.
- Antimicrobial therapy – altered fermentation by disruption of normal intestinal microflora can give rise to diarrhoea. In addition, antibiotic therapy predisposes to both salmonellosis and clostridiosis.
- Monensin/salinomycin – ionophores alter ionic transport across cell membranes: gastrointestinal, cardiovascular and neurological effects occur.

The pathophysiology of equine enterocolitis has not been defined in detail. The mechanisms which are likely to be involved include:

- Loss of the colonic mucosal barrier allows intraluminal bacteria and bacterial toxins to invade the colonic wall, stimulating mural inflammation.
- Raised local concentrations of prostaglandins, prostacyclin and leukotrienes.
- Stimulation of fluid/electrolyte secretion and protein exudation into the bowel lumen.
- Compromised colonic absorptive capacity.
- Increased permeability of intestinal microvasculature.
- Endotoxaemia and stimulation of the systemic inflammatory response syndrome.

Clinical signs

The clinical signs are somewhat variable, especially in Potomac horse fever, but generally cases of enterocolitis are extremely ill with rapid progression of:

- Markedly dull attitude.
- Fever.
- Inappetence.
- Decreased intestinal motility (may be gross abdominal distension in some cases).
- Colic.
- Explosive watery diarrhoea (occasionally acute onset of severe colic signs and rapid systemic deterioration in the absence of diarrhoea). Diarrhoea may be bloody in clostridiosis, colitis X and salmonellosis (especially in foals).
- Endotoxaemia and hypovolaemic shock – tachycardia, prolonged capillary refill time, injected or cyanotic mucous membranes, cold extremities, etc.
- ± Peripheral oedema.
- ± Laminitis.
- Rapid weight loss in survivors.

Some severe per-acute cases may be found dead with no recognized clinical signs.

Diagnosis

The peracute nature of enterocolitis necessitates emergency treatment before a definitive, specific diagnosis is available. Successful intensive care of horses with enterocolitis is ideally monitored by use of biochemical and haematological findings which are extremely dynamic in this condition. Typical clinical pathological features are:

- Dehydration/haemoconcentration – raised packed cell volume (PCV) and total protein (TP), but as the disease progresses there may be progressive decrease in TP due to intestinal protein loss; raised plasma creatinine and urea.
- Electrolyte imbalances – usually hyponatraemia and/or hypochloraemia and/or hypokalaemia.
- Azotaemia – due to pre-renal or renal causes.
- Metabolic acidosis and high plasma lactate concentration due to impaired tissue perfusion.
- Leucopenia.
- ± Thrombocytopenia or disseminated intravascular coagulopathy in Potomac horse fever. Abdominal ultrasonography can be used to demonstrate fluid contents of the caecum and colon, and increased mural thickness (>5 mm).
- Peritoneal fluid is usually normal in acute colitis, but secondary septic peritonitis may occur in horses with persistent colic or fever (secondary to translocation of intestinal bacteria or infarction of the colonic vasculature).

Specific diagnosis may be obtained by:

- Salmonellosis – culture or polymerase chain reaction (PCR); may require multiple sequential faecal samples or post-mortem tissue/intestinal contents.

- Clostridiosis – faecal toxin assays for *Clostridium difficile* and *Clostridium perfringens* (serial samples may be required); culture/Gram stain of faeces/intestinal content with quantitative count of colony forming units.
- Potomac horse fever – serology (IFA or ELISA), PCR.

Gross post-mortem and/or histopathological findings vary depending on the time-course of the disease. Necropsy findings usually include:

- Serosal discoloration; petechiae purple to black.
- Mucosal discoloration; haemorrhagic or necrotic black/brown.
- Mucosal oedema/thickening and sloughing of fibronecrotic debris (mucosal oedema may be especially marked in colitis X).
- Watery, foul-smelling intestinal contents.
- Mesenteric lymph node enlargement.
- Hyperaemia and/or petechial haemorrhages of lungs, liver and adrenal glands.
- Changes consistent with proximal enteritis (Chapter 2) may be present in some cases of clostridiosis.

Treatment

- Intravenous fluid therapy with correction of specific electrolyte and acid–base disturbances, and consider use of colloidals or plasma. Initial fluid resuscitation with hypertonic saline solution may be necessary in severely hypovolaemic patients.
- Flunixin meglumine at anti-endotoxic dose of 0.25 mg/kg three times daily.
- Endotoxin antisera/purified immunoglobulin products.
- Intestinal adsorbents e.g. di-tri-octahedral smectite.
- Frog supports.
- Antibiotics.
  - generally contraindicated but appropriate in neonatal salmonellosis.
  - metronidazole may be helpful in clostridiosis.
  - oxytetracycline is drug of choice in Potomac horse fever.
- Nutritional support.
- Critical care (see Chapter 26).

Horses with colitis are particularly prone to thrombophlebitis. Long-term polyethylene intravenous catheters should be used, and removed at the first sign of a problem with the catheter or vein.

Prognosis

Mortality rate is high even after aggressive, expensive, intensive care, and there is a high prevalence of complications. Cases which are diarrhoeic for more than 7 days are unlikely to survive. Acute enterocolitis does *not* usually result in chronic intestinal/digestive dysfunction in surviving cases.

3.23 NON-STEROIDAL ANTI-INFLAMMATORY DRUG TOXICITY

Any non-steroidal anti-inflammatory drug (NSAID) can potentially lead to either gastrointestinal and/or renal disease. Mucosal ulceration may occur at any site of the alimentary tract from the mouth to the rectum, but the most common sites of damage are the stomach and the right dorsal colon. such that right dorsal colitis is a recognized clinical entity.

Epidemiology

Long-term treatment and/or overdosing with NSAIDs, which may arise from owner ignorance, often underlie NSAID toxicity. Horses with cardiovascular compromise and/or dehydration will are more prone to NSAID toxicity.

Pathogenesis

Inhibition of mucosal prostaglandins E_1 and E_2 which have a mucosal protective effect within the stomach, intestine and kidneys. Loss of mucosal protection can result in either protein losing enteropathy as a result of gastrointestinal ulceration or protein losing nephropathy.

Clinical signs

Variable severity of:

- Inappetence/anorexia.
- Peripheral oedema.
- Oral mucosal ulceration.
- Diarrhoea.
- Colic.
- Endotoxaemia.
- Laminitis.
- Decreased urine output and/or dilute urination.

Diagnosis

Usually a presumptive diagnosis is made from the history. Laboratory assessment of fluid/electrolyte and acid-base disturbances is useful to assist fluid therapy, and monitoring of renal function and plasma proteins concentrations is appropriate.

Endoscopy can allow visualization of oesophageal or gastric ulceration.

Treatment

- Discontinue NSAID therapy.
- Intravenous fluid/electrolyte therapy.
- Frog supports.
- Proton pump inhibitors (e.g. omeprazole) or histamine receptor type – two antagonists (H_2 antagonists) (e.g. ranitidine) and sucralphate for gastric ulceration.
- Analgesia with butorphanol tartrate and/or alpha-2 agonists for colic (and laminitis).
- Misoprostol (synthetic analogue of prostaglandin E2) (5 µg/kg; q 12 hours or 2 µg/kg; q 6 hours po) – may cause diarrhoea.

Prognosis

Guarded in severe cases, e.g. diarrhoea, colic and laminitis, but slow recovery if signs are mild. Biochemical evidence of severe renal compromise generally indicates a poor prognosis.

3.24 OTHER GASTROINTESTINAL DISORDERS

Many other conditions of the gastrointestinal tract occur with low prevalence in the horse. In addition, various disorders of other body systems may give rise to gastrointestinal signs and some of these are described elsewhere in the text.

- Equine proliferative enteropathy (*Lawsonia intracellularis*) (Chapters 19 and 20).
- Foal heat diarrhoea (Chapter 20).
- Rotavirus (Chapters 19 and 20).
- Coronavirus (Chapter 20).
- *Rhodococcus equi* (Chapters 6 and 20).
- *Ehrlichia coli* (Chapter 20).
- *Campylobacter spp.*
- *Strongyloides westeri* (Chapter 19).
- Strongylosis (Chapter 19).
- Cyathostominosis (Chapter 19).

- Cryptosporidiosis.
- Coccidiosis.
- Giardiasis.
- Histoplasmosis.
- Cardiac failure (Chapter 7).
- Cushing's disease (Chapter 9).
- Hyperlipaemia (Chapter 22).
- Peritonitis (Chapter 4).
- Intestinal lymphangectasia.
- Cantharadin (blister beetle) toxicosis (Chapter 22).

FURTHER READING

Brown CM (1989) Problems in equine medicine. Lea & Febiger, Philadelphia

Byars TD (1983) Chronic liver failure in horses. Compendium on continuing education for the practising veterinarian Compendium on continuing education for the practising veterinarian 5:S423

Love S, Mair TS, Hillyer MH (1992) Chronic diarrhoea in adult horses: A review of 51 referred cases. Veterinary Record 130:217

Roberts MC (1983) Protein-losing enteropathy in the horse. Compendium on Continuing Education for the Practising Veterinarian 5:S550

Whitlock RH (1986) Colitis: differential diagnosis and treatment. Equine Veterinary Journal 18:278

APPENDIX

Differential diagnoses

1. Jaundice.
   - Liver disease.
   - Reduced food intake.
   - Haemolysis.
   - Biliary tract obstruction.
   - Septicaemia.
   - Leptospirosis.
2. Acute diarrhoea in the adult horse.
   - Larval cyathostominosis.
   - Salmonellosis.
   - Potomac horse fever.
   - Clostridiosis.
   - Antimicrobial therapy.
   - NSAID toxicity.
   - Other toxicities, e.g.
     - heavy metals.
     - monensin/salinomycin.
     - plants.
   - Endotoxaemia/septicaemia.
   - Overfeeding or sudden dietary change.
   - Anthrax.
   - Purpura haemorrhagica.
   - Anaphylaxis.
   - Idiopathic.
3. Chronic diarrhoea in the adult horse.
   - Larval cyathostominosis.
   - Mixed strongyle infections.
   - Alimentary lymphoma.
   - Inflammatory bowel diseases.
   - Sand enteropathy.
   - Peritonitis.
   - Congestive heart failure.
   - Liver failure.
   - Hyperlipaemia.
   - Idiopathic.

Chapter 4

Abdominal cavity

G. Barrie Edwards

CONTENTS

4.1 Hernias 67
Definitions 67
Types of hernia 67
Umbilical hernias 67
Ventral hernias 69
Incisional hernias 69
Prepubic tendon rupture 70
Epiploic foramen incarceration 70
Diaphragmatic hernia 71
4.2 Peritonitis 71
4.3 Abdominal abscess 73
4.4 Abdominal neoplasia 74
Gastrointestinal neoplasia 74
Urogenital tract neoplasia 75
Further reading 75

4.1 HERNIAS

Definitions

A hernia comprises the protrusion of an organ or part of an organ through the wall of the cavity normally containing it. Hernias may be classified as direct or indirect, internal or external.

A *direct* hernia occurs through a rent or tear whereas an *indirect* hernia occurs through a natural passage such as the inguinal canal or umbilicus.

An *external hernia* occurs through the body wall producing a visible and palpable swelling covered by skin, whereas an *internal* hernia occurs within the abdominal cavity (Box 4.1).

Types of hernia

Hernias may be congenital or acquired. Most are reducible, but in some cases the contents of the hernias cannot be returned to their normal location due to incarceration, strangulation or adhesions.

An *incarcerated* hernia is one in which the passage of ingesta through the protruding loop of intestine is arrested. The blood flow in its wall, however, is maintained.

A *strangulated* hernia is one which is both irreducible and incarcerated, and in which the blood circulation is also arrested, resulting in gangrene unless speedy relief is afforded.

Hernias with intermediate sized rings minor are more prone to incarceration and strangulation because even minor distension of the herniated intestine with gas or ingesta may result in compression of its lymphatic and venous drainage.

Umbilical hernias

Umbilical hernias are significantly more common in fillies than in colts, and may be hereditary. Because the majority of small hernias eventually close spontaneously it has become customary to wait until the foal is 6–12 months old before attempting surgery. Large defects show little tendency to spontaneous closure, and early repair is rewarded by the greater ease with which repair can be achieved in the young foal.

Congenital umbilical hernias are present at birth and are due to failure of the abdominal wall to close. Acquired umbilical hernias develop at 3–4 weeks of age. Excessive straining to defecate or micturate, and infection of the umbilicus, may be causative factors. It has been suggested that umbilical hernias are often the result of improper management/handling of the foal at birth such as manual ligation of the cord.

The fibrous hernial ring is composed of the aponeurosis of the transverse muscle, the fused oblique muscles and the abdominal tunic.

Umbilical hernias may be oval in shape and vary in size from one to several centimetres in diameter.

The hernial sac comprises an inner peritoneal layer and an outer layer of skin linked by varying amounts of connective tissue. Intestine

© 2013 Elsevier Ltd
DOI: 10.1016/B978-0-7020-2801-4.00004-3

Box 4.1 **Types of hernia**

External hernias

- Umbilical.
- Inguinal/scrotal.
- Traumatic abdominal wall hernias.
- Ventral.
- Incisional hernias.

Internal hernias

- Epiploic foramen.
- Mesenteric.
- Diaphragm.
- Omentum.
- Mesocolon.
- Gastrosplenic ligament.
- Hepatogastric ligament.
- Hepatoduodenal ligament.
- Ductus deferens.

(small intestine or caecum), omentum or both may constitute the contents of the hernial sac.

Incarceration and strangulation of the herniated intestine is rare and most likely to occur when the hernial ring is intermediate in size (i.e., large enough to allow entry of intestine yet small enough that intestinal distension results in constriction of blood vessels and lymphatics).

A parietal or Richter's hernia is defined as incarceration of the antimesenteric portion of intestinal wall is trapped within a hernial orifice. The lumen of the intestine bowel is not completely obstructed, and pain may be less severe. If the hernia goes unrecognized and uncorrected, ischaemic necrosis of the incarcerated 'pouch' of intestinal wall may lead to the development of an enterocutaneous fistula. The ileum and less frequently caecum may be involved.

Umbilical hernias complicated by infection of the remnants of the umbilical stalk are encountered only rarely in foals compared with calves. The umbilical structures should close within a week of birth and only fibrous remnants should remain at 6 weeks. In the event of infection one or more of the umbilical structures (one umbilical vein, two umbilical arteries and the urachus) will remain patent. Ultrasonography provides the best means of identifying which structure(s) is involved.

Examination of the hernia

In the vast majority of cases, gentle pressure on the protruding swelling will reduce the hernia, allowing identification of the hernial ring, which should be assessed for size, shape and rigidity.

Incarcerated hernias present as tense, painful swellings which are usually irreducible. Occasionally gas in the entrapped intestine may have been dispersed during transport to a surgical facility or by gentle pressure allowing reduction to be achieved. Although the immediate risk to the foal's life may have been removed, whenever possible repair of the hernia should be carried out as soon as possible. Immediate repair will allow identification and removal of necrotic intestine which will eventually rupture.

Incision of the hernia sac and careful examination of the caecum and small intestine should be performed to rule out the possibility of ischaemic compromise which is particularly likely with a Richter hernia. Even if ischaemic damage is not considered likely, any delay in repair of the hernia should be kept to a minimum because of the possibility of a further episode of incarceration which on that occasion may progress to strangulation.

Closed reduction

A wide variety of methods of treatment have been used in the past, including blisters, transfixing skewers and wooden or metal clamps. Although clamps are still in use, the method of closed reduction most often used is the application of elastrator rings at the base of the sac after ensuring that any contents have been returned to the abdomen. There may be some oedema between the rubber ring and the abdominal wall on the day after application, but any local inflammation usually resolves in 7–10 days. Separation of the hernial sac vestige should be allowed to occur without assistance and takes approximately 3–4 weeks. This technique depends on subcutaneous fibrous scar tissue to obliterate the defect, and is only useful in treating small one-finger defects.

With the availability of safe anaesthetic techniques and improved suture materials, there is little indication for employing a method which is less than certain to correct the hernia. Open reduction, whereby the defect in the abdominal wall is closed by apposing the edges of the ring or by the use of an inlay prosthesis is a much more satisfactory method of treatment.

Open reduction

The surgery is performed under general anaesthesia and strict aseptic conditions with the patient in dorsal recumbency. An elliptical skin incision is made around the umbilical scar extending 2 cm beyond the margin of the hernial ring cranially and caudally. Care must be taken to avoid removing too much skin. The peritoneal sac is carefully dissected away from the overlying skin and the abdominal wall until the margin of the ring can be identified. Unless there is residual umbilical stalk present, or ischaemic damage to the intestine is suspected, the hernial sac need not be incised but rather simply returned to the abdominal cavity.

The majority of hernial rings are oval in shape and their edges are sufficiently pliable to allow them to be closed by a series of overlapping mattress (Mayo) sutures of absorbable or non-absorbable material.

The sutures are inserted 1–2 cm from the margin of the ring and are carried through the full thickness of the abdominal wall on both sides of the ring. A finger inserted into the inverted sac serves to guide the needle and prevent inadvertent damage to the intestine (Figure 4.1). The ends of each suture are held with haemostats until they are all in place. Simultaneous traction on the sutures overlap the edges of the ring and this is maintained while each individual suture is tied. The subcutaneous tissue is apposed with a continuous suture of polyglactin, and the skin with a subcuticular suture of the same material. The application of an elastic bandage encircling the abdomen (or a stent bandage in the case of small hernias) will provide protection from contamination and eliminate dead space.

With large hernial rings, it may be necessary to bridge the defect with a prosthetic mesh or gauze. The mesh, which must be inert and nonreactive to the tissues, stimulates fibroplasia, resulting in it being enveloped in a thick fibrous bed that fills the defect and strengthens the abdominal wall.

The mesh is best placed in an extraperitoneal position between the internal rectus abdominal sheath and the peritoneum. The peritoneal sac is dissected down to the hernial ring and inverted into the abdominal cavity as described above. If the sac is sufficiently large, the mesh can be placed within it (Figure 4.2), but if it is not, the peritoneum is reflected peripherally from the deep fascial sheath of the rectus

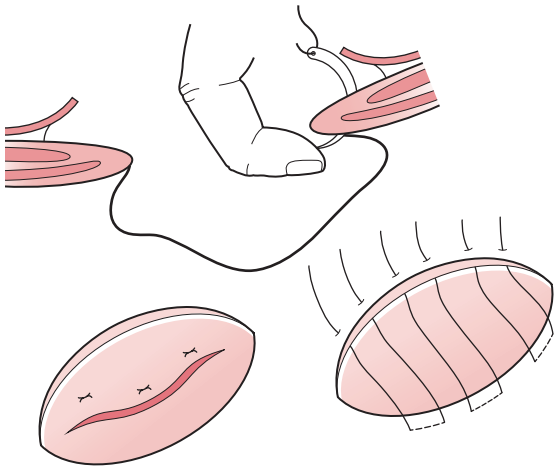


Figure 4.1 Repair of umbilical hernia by overlapping the margins using modified Mayo sutures.

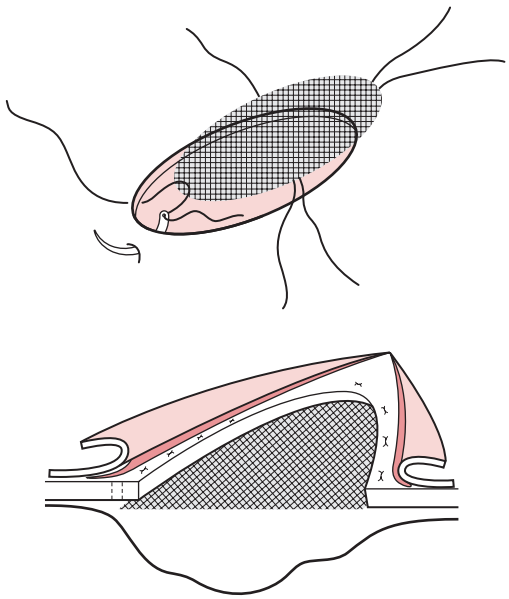


Figure 4.2 Repair of umbilical hernia using mesh prosthesis inserted within the inverted peritoneal sac.

abdominis muscle for 1–3 cm to create a space for the mesh. The mesh is cut so that it overlaps the margin of the ring by the same amount. The sutures are preplaced before tying. Sufficient tension is applied so that the mesh is kept taut and flat. The subcutaneous tissues and skin are then carefully apposed over the mesh.

Ventral hernias

A ventral hernia is one that occurs through any part of the abdominal wall other than the umbilicus or inguinal canal. They may be traumatic in origin or incisional. Kicks, collisions with blunt objects and straddling gates are common causes. Common sites are low in the flank (just dorsal to the edge of the rectus muscle), along the costal arch and along the ventral abdomen.

The abdominal wall is weakest just dorsal to the rectus abdominis muscle, since the oblique abdominal muscle is aponeurotic here, and only the transverse abdominal muscle is present. Flank trauma usually causes the abdominal wall to split just above the rectus muscle. Usually the tear in each layer occurs along the direction of the fibres, and if damage to the external oblique is extensive, the inguinal ring may be involved. If the peritoneum is also torn, herniated intestine will come to lie beneath the skin and panniculus muscle.

A large tear will allow a large mass of viscera (e.g. large colon) to escape from the abdomen, but the size of the swelling is not necessarily an indication of the size of the defect because a considerable length of small intestine can escape through a small rent.

Inflammatory exudate accumulates in the surrounding tissues, but little change may occur in the herniated intestine during the first 2–3 weeks after injury. Nevertheless, the risk of strangulation of intestine necessitates early diagnosis and surgical treatment.

Diagnosis

Signs of depression, abdominal discomfort and cessation of defecation indicate intestinal obstruction requiring prompt intervention.

Diagnosis is not always straightforward, and a number of diagnostic procedures should be considered to differentiate a hernia from a haematoma or abscess:

- Palpation of the swelling.
- Rectal examination.
- Auscultation.
- Ultrasonography.
- Exploratory surgery.

Palpation is complicated by the associated haemorrhage and oedema. Most defects are too ventral to be palpated *per rectum*. Aspiration, although helpful in identifying abscesses, is not advisable because of the risk of penetrating the bowel or introducing infection into a haematoma. Auscultation may reveal intestinal sounds which are more easily heard than over the remainder of the abdominal wall. *Ultrasonography* provides the most useful way of detecting herniated intestine.

Treatment

When intestinal obstruction necessitates immediate surgery, the tissues surrounding the rent or hernia are very friable and have little suture holding power. It is not uncommon for disruption of the surgical repair to occur during recovery from anaesthesia. Therefore, when the hernia is not accompanied by signs of intestinal obstruction, it is advisable to delay surgery for 3–6 weeks until swelling has subsided and deposition of collagen has increased the tensile strength of the damaged tissues.

Under general anaesthesia, the skin and subcutaneous muscle are carefully excised to reveal the herniated intestine. If examination of the intestine reveals no irreversible damage it is returned to the abdominal cavity. A midline laparotomy incision is helpful in identifying the precise site of the tear and allowing traction to be applied to the herniated gut.

The defect is closed by suturing each layer in turn.

When the defect is very large or its edges are too rigid to appose by suture, satisfactory repair may be achieved using mesh. Following closure of the peritoneum and transverse muscle by suture, a piece of polypropylene mesh is sutured to the deep face of the internal oblique aponeurosis.

Incisional hernias

Incisional or postoperative hernias are encountered relatively frequently following abdominal surgery, particularly in large, heavy horses.

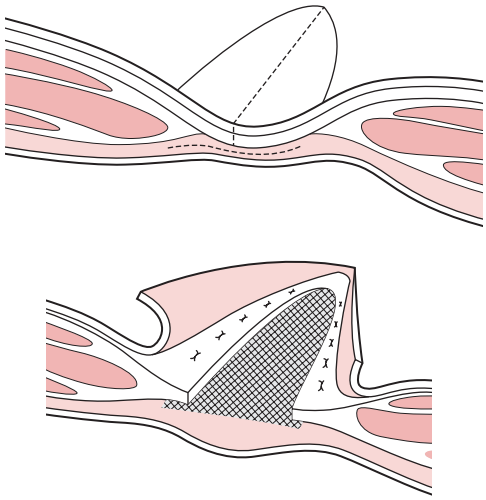


Figure 4.3 Repair of an incisional hernia using mesh prosthesis inserted into the space created by dissecting the peritoneum away from the margin of the abdominal wall defect.

Improper closure of the incision, sutures breaking or tearing through tissues and, of greatest importance, postoperative wound infection, are contributory factors.

The weakened abdominal wall undergoes loss of continuity and a hernia develops often after a delay of several weeks or months. The hernial rings vary considerably in size and can be very large.

Forceful approximation of the edges of a large fascial defect inevitably leads to failure. To repair large hernias a mesh prosthesis is required. Since the margin of the defect is less well defined than in umbilical hernias, it is necessary to dissect the peritoneum from the inner sheath of the rectus to create an adequate 'shelf' to support the mesh (Figure 4.3). Alternatively, a mesh inlay graft with the onlay apposition of supportive hernial sac fascia can be used (Figure 4.4).

Healing is facilitated by obliterating dead space and preventing accumulation of serum. When the surgery site is the ventral abdomen an elastic bandage or a belly bandage can be applied over the wound for 2 to 3 weeks after surgery. The horse should be given box rest with daily walking in hand for 2 months.

Prepubic tendon rupture

A tendinous rupture of the rectus abdominis muscles can occur in mares in late gestation or in early lactation. The prepubic tendon appears to degenerate at its attachment to the pelvis. The onset of rupture is gradual. A swelling appears cranial to the udder and may at first be mistaken for periparturient oedema. The damage progresses (may be associated with further pregnancy) and eventually the contour of the ventral abdomen drops to the level of the hock accompanied by oedematous swelling of the subcutaneous connective tissue.

The large hernial ring runs transversely just anterior to the pelvis. Not all of the ring can be palpated *per rectum*. It can only be felt externally with the mare in dorsal recumbency. *It is not possible to repair the rupture.*

Epiploic foramen incarceration

Entrapment of the small intestine through the epiploic foramen is the most common form of internal hernia in the horse and is the cause of colic in 2–8% of all horses undergoing colic surgery. Previous suggestions that increasing age is a risk factor have recently been refuted.

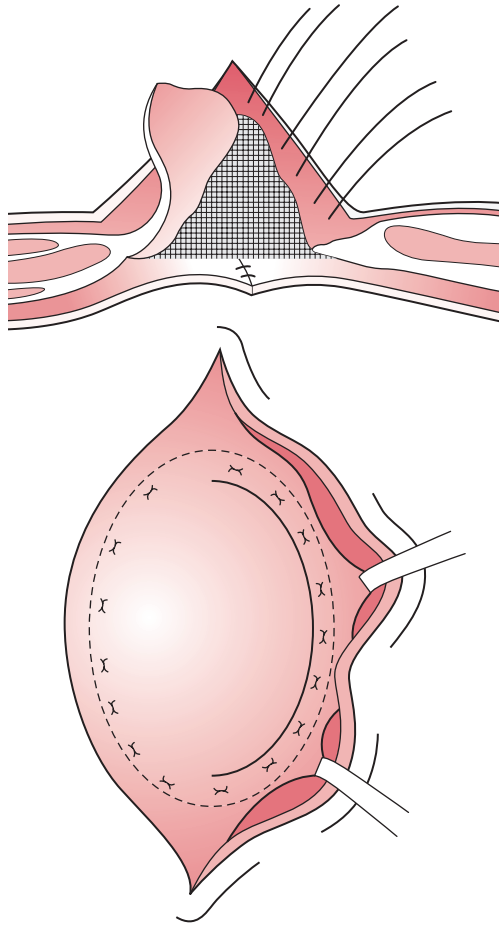


Figure 4.4 Repair of an incisional hernia using a mesh and fascial flap overlay technique.

Although more common in older horses, it can occur in horses of any age and has been reported in a foal 5 months of age.

Recent epidemiological studies have shown that crib biting or wind-sucking behaviour is significantly associated with risk of epiploic foramen entrapment (EFE). Horses that exhibited crib biting or wind-sucking behaviour were 72 times more likely to develop EFE than horses which did not exhibit this behaviour. Horses of greater height were also identified to be at increased likelihood of EFE. A horse 17 hh was 11.4 times more likely to develop EFE compared to a pony.

There is evidence of a seasonal pattern of EFE in the UK, with more cases occurring in January than in any other month; this might be explained by the fact that increased stabling in the previous 28 days was identified as a risk factor.

Horses that had access to a mineral/salt lick were found to have a significantly reduced likelihood of EFE.

The epiploic foramen (the relatively narrow passage through which the greater and lesser peritoneal sacs communicate) is situated on the visceral surface of the liver dorsal to the portal fissure. It is bounded dorsally by the caudate lobe of the liver and the posterior vena cava, and ventrally by the right lobe of the pancreas, gastropancreatic fold and portal vein. The foramen is limited cranially by the hepatoduodenal ligament and caudally by the juncture of the pancreas and duodenum.

In the adult horse the foramen allows introduction of the tips of three fingers. The suggestion that it enlarges with age due to atrophy of the caudate lobe of the liver has not been confirmed.

In >90% of horse with EFE the intestine passes through the foramen from left to right. In the process the lesser omentum is torn. In the remaining cases the intestine enters the foramen from the right side and enters the lesser omental bursa. The length of gut involved can vary from a few centimetres to 17 metres.

Clinical signs

- The clinical signs are those of small intestinal obstruction (see Chapter 2) and vary with the nature and severity of the damage to the incarcerated bowel.
- Approximately 80% have irreversible vascular compromise of the herniated small intestine.

Diagnosis

- Diagnosis is usually made at exploratory laparotomy (see Chapter 2 for indications for exploratory laparotomy in acute abdominal pain).
- Sanguineous peritoneal fluid with a raised protein level is indicative of a strangulating lesion but when herniation occurs from right to left the transudation enters the omental bursa and may not be collected by paracentesis.

Treatment

Conservative treatment is of no value, and surgery should be performed at the earliest opportunity.

During laparotomy the epiploic foramen is most easily performed from an operating position on the patient's left side using the right hand to explore the right hypochondral region. The hand is passed lateral and dorsal to the right dorsal colon and forward to the visceral surface of the liver. The only bowel that normally occupies the gutter between the right dorsal colon and the dorsal abdominal wall is the duodenum which is suspended by the short mesoduodenum permitting little lateral movement or mobility of this fixed part of the small intestine. If the ileum is located and the small intestine is traced orally, it will identify jejunum leaving the foramen and the distended afferent arm entering it. In >40% of cases the ileum itself is involved in the herniation.

Distension of the herniated intestine with fluid and gas, and oedema of its wall, can make reduction difficult. *Attempts should never be made to enlarge the foramen, since this would result in fatal haemorrhage.* Traction applied to either the afferent or efferent intestine on the parietal side of the foramen should be gentle and along the plane of the foramen. As the trapped intestine is withdrawn, gas and fluid tends to dam back in the remaining loop increasing its diameter and tension. To counter this it is necessary to gently massage the intestine to encourage gas and fluid to pass through to the left side. Strangulated bowel is resected and an anastomosis is performed. In cases where the short loop of entrapped intestine is impacted with firm ingesta, a further length of fluid-filled afferent intestine must be pulled through the foramen to permit dispersion of the ingesta before the herniated ileum can be reduced.

Survival studies have shown that both the short term and long term prognosis is less favourable than for other strangulating obstructions.

Diaphragmatic hernia

Diaphragmatic hernia is an uncommon occurrence in horses and is usually discovered during laparotomy or autopsy.

Diaphragmatic defects may be congenital or acquired but in most reported cases there was convincing evidence that the diaphragm had ruptured either spontaneously or as a result of trauma. Most diaphragmatic hernias follow thoracic or abdominal trauma or strenuous exercise, parturition or extreme gastrointestinal distension.

The absence of haemorrhage at the margins of a defect found at autopsy identifies rupture which has resulted from post-mortem tympany.

Displacement into the thorax of large and small intestine, stomach, omentum, liver and spleen in one combination or another may occur.

Clinical signs

The clinical effects of diaphragmatic hernia are very variable.

- If only small intestine is displaced, signs of intestinal obstruction predominate, often with a rapidly fatal outcome.
- If large intestine moves into the chest, its greater bulk results in compression of the lungs and respiratory distress is more likely. In such cases respiratory and digestive signs may coexist, and the illness may be more protracted because large bowel obstruction is less rapidly fatal than is incarceration of small intestine.

Diagnosis

- Absence of lung sounds or increased intestinal sounds in the chest.
- Dullness on percussion over the ventral thorax.
- Sanguineous fluid on thoracocentesis.
- A relative emptiness of the abdomen may be apparent during abdominal examination *per rectum* (provided that the remaining segments of gut are not distended as a result of bowel obstruction).
- Thoracic radiography.
- Ultrasonography.

Treatment

- Surgical correction presents considerable problems and the prognosis in most cases of acquired diaphragmatic hernia is grave.
- The severe respiratory and cardiac embarrassment caused by the displaced viscera, and the large volume of fluid present in the chest, may result in death during anaesthetic induction.
- Adequate pulmonary ventilation during surgery is possible only by using intermittent positive pressure ventilation.
- Bilateral pneumothorax can develop when the abdomen is opened or, more frequently, when the hernia is reduced.
- Large dorsal diaphragmatic rents are relatively inaccessible and retraction of herniated viscera is difficult. Small rents may need to be enlarged before the herniated small intestine can be withdrawn from the chest.
- Once reduced, the infarcted bowel can be resected provided the length involved is not too great. However, it is not uncommon for all but the relatively fixed duodenum and ileum to pass into the chest.
- Any attempt at closure requires extending the abdominal incision cranially to the xiphisternum. Even then, access to the defect may be very restricted making closure by suture or the use of prosthetic mesh difficult.

4.2 PERITONITIS

The peritoneum normally secretes a serous fluid which lubricates the abdominal cavity, minimizes adhesion formation, has minimal antibacterial properties and acts as a semipermeable barrier between the blood and the abdominal cavity. Inflammation of this mesothelial lining is termed peritonitis and is characterized by hyperaemia, peritoneal effusion and fibrin deposition, chemotactic phagocytosis and increased peritoneal permeability to toxins.

Table 4.1 Causes of peritonitis

Infectious or septic	Non-septic	Parasitic	Traumatic	Iatrogenic
Surgical complications: • failure of anastomosis. • non-viable tissue. • poor asepsis.	Ruptured bladder, ureter or kidney	Verminous arteritis	Breeding or foaling accident e.g. uterine artery haemorrhage	Rectal tear
Intestinal accidents with perforation	Chemical agents: • bile. • gastric juice. • pancreatic juice.	Parasitic larval migration	Penetrating abdominal wound	Uterine perforation: • infusion. • biopsy. • artificial insemination.
Uterine rupture or perforation	Foreign body	Perforating lesions (ascarids, tapeworms)	Blunt abdominal trauma	Enterocentesis
Metritis	Neoplasia: • ovarian. • abdominal.		Ruptured diaphragm	Caecal trocharization
Urachal infection	Urolithiasis			Liver biopsy
Post-castration infection	Hepatitis			
Enteritis	Gastric rupture			
Septicaemia				
Cholangitis				

Although it may occur as a primary condition, it is more commonly seen as a secondary complication associated with infectious (bacterial, viral, fungal, parasitic) or non-infectious (traumatic, chemical, neoplastic) stimuli (Table 4.1). In primary peritonitis, the route of bacterial spread is not evident, but impaired defences may be involved. Bacteria enter the abdominal cavity in secondary peritonitis most commonly following disruption of the integrity of the gastrointestinal tract, or less frequently the genitourinary tract or abdominal wall.

Peritonitis may be further classified according to onset (peracute, acute, chronic) and the region affected (localized or diffuse). Its severity is related to a number of factors including the underlying problem, the nature of the infectious agent, the resistance of the host, speed of recognition and intervention, and response to initial therapy.

The inflammatory responses mobilize leucocytes and immunoglobulins, and cause a profound relocation of proteins, fluid and electrolytes from plasma. These fluid shifts can lead to cardiovascular collapse. Fibrinolytic activity is depressed and fibrin is rapidly precipitated either diffusely in septic peritonitis or following major gastrointestinal leakage, or focally around an intestinal perforation. Although initially of benefit by confining the contamination and infection, these processes may become deleterious, resulting in hypovolaemia, hypoproteinaemia, reflex ileus with bowel distension, ischaemia of bowel wall allowing absorption of bacteria and toxins, and adhesion formation.

Clinical signs

The clinical signs vary considerably depending on the cause and the extent of the disease.

1. Horses with peracute peritonitis (e.g. following gastric rupture) may be found dead or present with profound toxaemia, which rapidly leads to circulatory failure and death in 4–12 hours.
2. Acute diffuse peritonitis:
   - tachycardia.
   - tachypnoea.
   - congested to cyanotic mucous membranes.
   - cold extremities.
   - thready pulse.
   - dehydration.
   - depression.
   - sweating.

   Signs of both visceral and parietal pain may be present but parietal pain characterized by immobility, splinting of the abdominal wall and sensitivity to extreme pressure is usually predominate.
3. Localized, subacute or chronic peritonitis:
   - dullness.
   - decreased appetite.
   - progressive weight loss.
   - abdominal pain may be low-grade, intermittent or absent.
   - intermittent fever.
   - variable bowel sounds.
   - chronic diarrhoea in some cases.

Diagnosis

A definitive diagnosis can usually be made simply by examination of a peritoneal fluid sample for cytology and total protein. It may be turbid and off-white suggesting a high white blood cell count, homogeneously bloodstained suggesting haemoperitoneum or intestinal infarction, or turbid and brown/green in colour suggesting contamination with intestinal contents.

Early in the inflammatory process, the elevated white blood cell (WBC) count is due primarily to an increase in polymorphs while in chronic cases mononuclear cells and macrophages are increased.

Peritoneal fluid protein is significantly higher than normal.

Examination of Gram-stained smears may show free or phagocytosed bacteria.

Further evaluation of the degree of severity of peritonitis depends on laboratory estimation of fluid and electrolyte balance, blood gas analysis and haematology:

- In peracute and acute peritonitis, there is leucopenia with neutropenia and a degenerative left shift.

- Protein sequestration and fluid exudation into the peritoneal cavity leads to hypoproteinaemia and dehydration.
- In acute peritonitis of longer duration, and in localized or chronic peritonitis the changes in total WBC count are less dramatic.
- A decrease in albumin/globulin ratio is frequently present.

Rectal findings in cases of peritonitis are variable:

- Contamination of the abdominal cavity with gastrointestinal contents causes a gritty feeling to the serosal surface of bowel.
- In mares with uterine rupture, a fibrinous adhesion may be noted over the affected area.
- Distended bowel or secondary impaction may be evident while in other cases there may be no abnormalities palpable.

In foals, radiographs may show free fluid, ileus or free gas. Ultrasonography and laparoscopy can also be of diagnostic value.

Early exploratory laparotomy may be indicated for diagnostic, therapeutic and prognostic reasons, and is best performed while the horse is in reasonable condition.

Therapy

General therapy

- In many cases treatment of peritionitis is not necessary when it is the expected result of procedures such as castration or inadvertent enterocentesis during abdominocentesis.
- Early aggressive therapy is required if treatment is to be of benefit when peritonitis is caused by bacterial infection.
- The objective should be to correct the cause of the peritonitis if possible, and overcome the infection and the effects of inflammation.
- Pain relief, correction of fluid, electrolyte and acid–base disturbances, and treatment of cardiovascular and endotoxic shock should be addressed.
- Medical treatment is unlikely to be successful without surgical correction of the cause.
- Peritonitis may be associated with infection by any of the aerobic or anaerobic bacteria normally found in the intestinal tract. Alternatively, as in the case of abscesses, specific organisms such as *Streptococcus equi* or *Rhodococcus equi* may be involved. A positive culture is not always possible, and antibiotic therapy should be started as soon as possible. Penicillin is suitable as a first choice antibiotic because it is well tolerated and produces peritoneal fluid concentrations in excess of minimum inhibitory concentration and is bactericidal against most Gram-positive aerobic organisms and most anaerobic organisms. For Gram-negative aerobic organisms and *R. equi*, gentamicin is recommended. Metronidazole can also be used to treat anaerobic infections.
- Non-steroidal anti-inflammatory drugs are useful in combating endotoxaemia and SIRS.
- Larvicidal anthelmintics such as ivermectin or moxidectin are indicated when parasitism is thought to be the cause.

Abdominal drainage and lavage

- Drainage of peritoneal fluid can be achieved using a cannula, Foley catheter or indwelling drain, but serious doubts exist about our ability to achieve mechanical removal of contaminants in the horse related to the size and limited access to many parts of the abdominal cavity.
- Premature obstruction of the drain with omentum or fibrin is often a problem, and local tissue irritation and cellulitis resulting from leakage of fluid into the subcutaneous tissues are other possible complications.
- Effective lavage of the peritoneal cavity requires large volumes (20–30 L) of sterile Hartmann's solution.
- Failure to drain these large volumes effectively can increase morbidity and mortality.

Prognosis

If the causal lesion can be rapidly identified and corrected, the prognosis is fair to good. However, it is very poor if the peritonitis is the result of intestinal perforation. Few cases of localized adhesion formation or abscessation have a successful outcome; even if the immediate crisis is overcome, chronic ill-thrift and recurrent colic are frequent sequelae.

4.3 ABDOMINAL ABSCESS

The majority of internal abdominal abscesses involve the mesenteric lymph nodes and are believed to be the result of systemic spread of respiratory infections, but may be the sequel of foreign body penetration of the intestine or umbilical infection.

The causative organisms most frequently encountered are *Streptococcus equi*, *Streptococcus zooepidemicus*, *Escherichia coli*, *Salmonella* spp., and, in foals, *Rhodococcus equi*. Infrequently, anaerobes may be involved.

Horses of any age may be affected, and those exposed to strangles are at greatest risk.

Heavily parasitized horses may be predisposed to abscesses as the result of migrating larvae invading mesenteric tissues allowing for secondary bacterial contamination. As the abscess enlarges and the associated adhesive peritonitis becomes more extensive, the adjacent small intestine may become compressed and/or constricted.

Clinical signs

The most common clinical signs are:

- Chronic weight loss.
- Intermittent low-grade colic.
- Inappetence.
- Low-grade fevers.
- Intermittent diarrhoea.

The colic may be the result of stretching of the mesentery, or adhesion or scarring of the intestine.

Occasionally, a large abdominal abscess may go unsuspected until the horse develops acute obstruction. Rectal examination may reveal a localized well-encapsulated mobile mass which may be painful to the touch. More frequently the abscess is very large, involves loops of bowel and is firmly adherent to the roof of the abdomen.

Peritoneal fluid may be normal, or reveal elevated white blood cells (predominantly neutrophils) and protein. If the abscess is mobile, it may be possible to obtain a needle aspirate percutaneously under ultrasound guidance. The purulent material should be submitted for both aerobic and anaerobic culture.

Treatment

- Long-term antibiotic treatment for a minimum of 30 days is recommended. Oral administration of antibiotics such as rifampicin, potentiated sulphonamides and metronidazole avoids many of the complications of long-term administration of parenteral medication.
- Surgical intervention aimed at resecting the abscess is not recommended because of the serious risk of rupture and peritoneal contamination. However, surgical drainage can be possible in some cases.

- In cases with small intestinal involvement, the obstructed intestine can be bypassed by side-to-side anastomosis of a proximal and distal segment, but horses in which numerous loops of bowel are found to be involved in extensive adhesions to the abscess should be euthanased.

4.4 ABDOMINAL NEOPLASIA

Neoplasms of the abdominal cavity are uncommon, and present a considerable diagnostic challenge. They include primary and metastatic tumours, and are generally seen in middle-aged to older animals. The majority of neoplasms involve the gastrointestinal system. Neoplasia of the urogenital system is much less common.

Gastrointestinal neoplasia

Neoplasia accounts for approximately 5% of horses presenting with signs of acute or chronic abdominal disease. Of the primary neoplasms encountered squamous cell carcinoma of the stomach, the alimentary form of lymphoma, and lipomas are the most common. Other types are seen rarely (Box 4.2). Metastatic spread of malignant melanoma, transitional cell carcinoma, keratoma or others occurs occasionally.

Clinical signs

The presentation and clinical signs of abdominal neoplasia depend on the location and type of neoplasm. Since treatment is rarely possible or feasible, an early diagnosis is desirable to prevent unnecessary suffering. However, the signs are often vague, making early recognition and diagnosis difficult.

Clinical signs can include:

- Progressive weight loss.
- Depression.
- Reduced exercise tolerance.
- Pyrexia.
- Ventral oedema.
- Colic.
- Ascites.

Box 4.2 **Neoplasia of the gastrointestinal tract**

Stomach

- Squamous cell carcinoma.
- Adenocarcinoma.
- Leiomyoma.
- Fibrosarcoma.

Intestine

- Lymphosarcoma.
- Leiomyoma.
- Gastrointestinal stromal tumour.
- Leiomyosarcoma.
- Adenocarcinoma.
- Hamartoma.
- Myosarcoma.

Omentum and mesentery

- Lipoma.
- Mesothelioma.

- Abdominal distension.
- Diarrhoea.

Diagnosis

Cases suspected of having abdominal neoplasia should undergo a thorough clinical examination supported, where applicable, by haematology, serum biochemistry, peritoneal fluid evaluation, rectal palpation and abdominal ultrasonography (see Chapter 3).

Further diagnostic procedures that may be required include organ function tests, gastroscopy, biopsy, laparoscopy, and exploratory surgery (see Chapter 3).

Gastric squamous cell carcinoma

Squamous cell carcinoma is the most common gastric neoplasm in the horse. Middle-aged and older horses are most susceptible. There is a reported 4 : 1 male : female ratio.

The tumour usually originates in the oesophageal portion of the stomach, infiltrates the wall and projects like a cauliflower into the lumen. Metastatic nodules may occur in the liver, spleen, omentum, intestines, pleura and lungs, and there may be adhesions between the stomach and the liver or diaphragm.

Clinical features

- Chronic weight loss over a 1–2 month period.
- Anorexia.
- Depression.
- Anaemia (PCV 12–28%) may develop as a result of haemorrhage into the stomach or depressed erythrogenesis.
- Pain is not usually present.
- Often, excessive salivation.

Diagnosis

- Passage of a nasogastric tube may meet resistance if the tumour involves the stomach adjacent to the cardia.
- Neoplastic cells may be found in fluid recovered by gastric lavage.
- A video or fibreoptic endoscope 1–3 m in length will enable a definitive diagnosis by direct visualization and biopsy of the tumour.
- Ultrasonography from the left cranial abdomen may show thickening and abnormal echogenicity of the stomach wall.
- Ascites is commonly associated with the tumour, and secondary spread may be recognized as nodules in the liver, spleen, etc. Palpable masses or adhesions may be evident on rectal examination around the cranial mesentery.
- Exploratory laparotomy or laparoscopy allows more complete examination of the stomach region, and allows biopsy of the primary tumour mass or metastatic nodules.

Prognosis

By the time a diagnosis is made, the tumour has progressed beyond the point where any treatment is feasible. Euthanasia is the only option.

Lymphoma

Lymphoma is seen most commonly in horses more than 10 years of age, although it has been seen in animals as young as yearlings. It accounts for between 1.3 and 2.8% of all tumours seen in the horse.

Most lymphomas appear in lymph nodes or in tissues rich in lymphoid cells such as the spleen, bone marrow, pharynx and gastrointestinal tract. The alimentary form (see Chapter 3) occurs with slightly greater frequency than the mediastinal (thoracic) form (see Chapter

6). Alimentary lymphoma may occur as either a diffuse or a focal lesion.

Diffuse infiltration of either the small intestine and/or large intestine with involvement of local lymph nodes can result in both small intestinal carbohydrate malabsorption and/or protein losing enteropathy with a typical clinical presentation of progressive weight loss and/or diarrhoea.

Alimentary lymphoma may also result in single or multiple focal infiltration of the small intestine in focal lesions which are typically annular and progress in size to occlude the lumen. Enlargement of the mesenteric lymph nodes may sometimes accompany the focal intestinal lesion. The clinical course of focal intestinal lymphoma is chronic, extending over several weeks or months with intermittent, increasingly severe bouts of abdominal pain as the degree of obstruction increases.

Diagnosis

Clinical pathological findings are non-specific but may include:

- Hypoalbuminaemia.
- Hyperglobulinaemia.
- Neutrophilia.
- Anaemia.
- Hyperfibrinogenaemia.
- Raised serum alkaline phosphase.
- Reduced glucose absorption (OGTT).
- Peritoneal fluid cytology – rarely detect tumour cells.
- Decreased plasma concentration of IgM.

In addition, in focal lymphoma diagnosis may be suspected on the basis of:

- Marked muscular hypertrophy of several metres of intestine proximal to the lesion. Hypertrophy occurs in response to the increased workload required to force ingesta through the narrowed intestine. This enlarged, thick-walled intestine may be palpable *per rectum* (the neoplastic mass itself is less likely to be palpable).
- Palpable enlargement of mesenteric lymph nodes.
- Demonstration of the bowel wall thickening by use of trans-rectal or trans-abdominal ultrasonography.

Prognosis

Horses with widespread lesions should be euthanased but others with a single primary lesion of small intestine sometimes make a long-term recovery following resection of the affected portion of bowel.

Lipomas

Pedunculated lipomas, benign fat aggregations attached to the mesentery, are common findings in horses and ponies in their mid teens or older, and have the potential to obstruct the small intestine, or, less frequently, the small colon. The risk of this disease is increased by obese body condition which is common in elderly ponies.

Clinical signs and treatment

Lipomas with a long pedicle can cause strangulation of varying lengths of gut by wrapping tightly around a loop of intestine. This is accompanied by a sudden onset of severe, persistent colic and rapid degeneration of the obstructed intestine. Resection of the infarcted bowel is almost invariably necessary.

In a small proportion of cases, the pedicle is attached at the mesenteric attachment of the intestine. When the lipoma reaches a large size, its weight can cause partial simple obstruction of the intestine, resulting in moderately severe, sometimes intermittent, colic. At laparotomy, the lipoma can be removed but resection of bowel is rarely necessary.

Urogenital tract neoplasia

Ovarian neoplasia

See Chapter 14.

Testicular neoplasia

See Chapter 14.

Renal neoplasia

See Chapter 8.

Bladder neoplasia

Neoplasia of the bladder is rare. Cystoscopy using a video or fibreoptic endoscope is the preferred means of obtaining a tissue sample from an intraluminal mass. Ultrasonography is also useful, particularly in distinguishing a cystic calculus from a tumour.

Phaeochromocytoma

See Chapter 9.

FURTHER READING

Auer JA (1992) Equine surgery. W.B. Saunders, Philadelphia

Bristol DG (1986) Diaphragmatic hernias in horses and cattle. Compendium on Continuing Education for the Practicing Veterinarian 8:S407

Holt PE (1986) Hernias and ruptures in the horse. Equine Practice 8:13

Scott EA (1979) Repair of incisional hernias in the horse. Journal of the American Veterinary Medicine Association 175:1203

Chapter 5

Disorders of the ear, nose and throat

J. Geoffrey Lane

CONTENTS

5.1 Diseases of the external ear 78
Anatomical considerations 78
Clinical signs of otitis externa 78
Investigation of ear disorders 78
Temporal teratoma 78
Parasitic otitis 79
Chronic keratinization plaques 79
Foreign bodies 79
Neoplasia: sarcoids 79
Neoplasia: other ear tumours 79
5.2 Diseases of the middle ear 79
Anatomy and function of middle ear 79
Otitis media 79
Temporohyoid osteoarthropathy (THO) 79
Otitis interna/peripheral vestibular disease 79
5.3 Diseases of the auditory tube diverticulum (ATD) (guttural pouches) 80
Anatomy and function of ATDs 80
Signs of ATD diseases 80
Physical examination of ATDs 80
Endoscopy of ATDs 80
Radiographic examination of ATDs 80
Topical treatment of ATDs 80
Surgical approaches to ATD 80
5.4 Guttural pouch tympany 81
5.5 Diverticulitis of the guttural pouch 81
'Strangles' 81
Chronic ATD empyema and chondroids 81
Chronic ATD diverticulitis 81
5.6 Guttural pouch mycosis 82
5.7 Other ATD disorders 83
Trauma 83
Neoplasia 83
5.8 Disorders of the external nares 83
Anatomical features 83
'Wry nose' 83
Hypertrophy of alar folds 83
'Atheroma' of false nostril 83
Facial paralysis 83
Trauma 83
Alar cartilage necrosis 84
5.9 Diagnostic approach to nasal and paranasal sinus disease 84
Anatomical considerations 84
Common presenting signs 84
History 84
Physical examination 84
Endoscopy 84
Radiography 85
Other imaging modalities 85
Biopsy 85
5.10 Treatment of sinu-nasal disorders 86
Treatment objectives 86
Medical management 86
Trephination 86
Facial flap surgery 86
5.11 Primary and secondary empyema 87
5.12 Progressive ethmoidal haematoma (PEH) 88
5.13 Sinus cysts 89
5.14 Mycotic rhinitis and sinusitis 89
5.15 Sinus and nasal neoplasia and polyps 90
5.16 Other sinu-nasal disorders 90
Nasal septal deviation 90

© 2013 Elsevier Ltd
DOI: 10.1016/B978-0-7020-2801-4.00006-7

Other nasal septal disorders 90
Conchal necrosis and metaplasia 91
Facial and sinus trauma 91
Intra-nasal foreign bodies 91
Subcutaneous emphysema 91
5.17 Idiopathic headshaking in horses 91
5.18 Diagnostic approach to conditions causing airway obstructions in horses 92
Functional concepts of conducting airways 92
Respiration and locomotion 92
Intra-narial larynx and role of the soft palate 92
Clinical signs of obstructive dyspnoea 93
Respiratory noises at exercise 93
History 93
Palpation tests 93
Endoscopy at rest 93
Radiography of the throat 93
Conduct of exercise tests 93
Endoscopy after exercise 94
Treadmill and overground endoscopy of horses during exercise 94
Ultrasonography of the upper respiratory tract 94
5.19 Common upper respiratory tract obstructive disorders of horses 95
5.20 Recurrent laryngeal neuropathy (RLN) 95
5.21 Dorsal displacement of the soft palate (DDSP) 97
5.22 Epiglottal entrapment (EE) 99
5.23 Sub-epiglottal cysts 100
5.24 Arytenoid chondropathy 100
5.25 Fourth branchial arch defects (4-BAD) 101
5.26 Axial deviation of the ary-epiglottal folds (ADAF) 102
5.27 Other causes of dynamic airway collapse 102
Pharyngeal collapse 102
Ventro-axial luxation of the corniculate process of the arytenoid cartilage (VALCPA) 102
Epiglottal retroversion 103
5.28 Other causes of airway obstruction in horses 103
Choanal stenosis 103
Pharyngeal lymphoid hyperplasia (PLH) 103
Palatal cysts 103
Palatal defects 103
Pharyngeal cysts 103
Pharyngeal paralysis 103
Pharyngeal and laryngeal neoplasia 103
Congenital tracheal collapse 103
Acquired tracheal obstructions 103
Tracheotomy intubation 104
5.29 Miscellaneous throat conditions of horses 104
Wind-sucking and crib-biting 104
Salivary gland disorders 104
Thyroid neoplasia 104
Parotid melanomas 104
Facial palsy 105
Horner's syndrome 105
Jugular thrombophlebitis 105
Nuchal fractures 105
Cervical injection infection and abscesses 105
Further reading 105
Appendix 1 105
Appendix 2 106

5.1 DISEASES OF THE EXTERNAL EAR

Anatomical considerations

The external ear consists of a semi-rigid open-ended tube supported by the auricular and annular cartilages and is lined by modified integument. The canal comprises a vertical segment covered by pigmented skin and rich in wax-producing cells, and a very narrow horizontal section which is non-pigmented and leads to the pretympanic region.

The pinna is a highly mobile structure to optimize the directional appreciation of sound. The auricular muscles are innervated by the auriculo-palpebral branch of the facial nerve and loss of ear mobility indicates an injury close to the emergence of the nerve trunk from the facial canal.

Clinical signs of otitis externa

Abnormal ear carriage may indicate aural pain or malfunction of the auricular muscles.

Discharge at the natural opening of the meati or staining of the skin of the parotid region may be present with or without an unpleasant smell.

- Pale keratinized plaques of the aural integument.
- Soft-tissue swellings.
- Resentment of handling around the ears.

Investigation of ear disorders

Horses resent detailed inspections of their ears, and care must be taken in the interpretation of perceived otalgia.

A distant inspection may reveal abnormal carriage of the ear or of the head as a whole.

Swellings or overt otorrhoea voided over the parotid area or a discharging sinus tract at the rostral margin of the pinna (see 'Temporal teratoma', below) may be seen. The discharge may be malodorous if secondary infection is present.

Digital palpation may reveal soft tissue swellings in the canal. It is normal for the lining of the vertical ear canal to be covered with dark waxy secretions.

Smears may be taken to identify parasitic mites under low-power microscopy.

Detailed otoscopy can be performed only under general anaesthesia. Appropriate equipment must be on hand to inspect the very narrow horizontal canals in this species. A 4-mm, rigid arthroscope makes an effective otoscope.

Temporal teratoma

Teratomatous lesions may develop adjacent to the external ear in horses. While the majority of teratomas at this site contain identifiable dental tissues some are of dermal origin.

The characteristic presenting sign of temporal teratoma consists of a persistent discharging sinus tract which opens at the rostral margin of the pinna 2–3 cm from the natural opening of the ear.

Young horses are invariably involved, i.e. under 1 year of age when signs first appear.

There may or may not be a visible or palpable swelling.

Radiographs are useful to differentiate dermal from dental teratomas, to locate the sac where the teratoma lies, and to establish the size of the lesion which is to be resected.

Treatment consists of resection following dissection along the discharging tract. Where deep lesions are involved care is required to avoid damage to the facial nerve and the ear canal itself.

Parasitic otitis

Otodectic mites may cause aural irritation but are easily identified either with a hand lens or by examination of smears. The acaricidal preparations used to treat parasitic otitis in small animals are effective in horses.

The ears appear to be attractive to small biting flies during the summer months, and behaviour typical of that caused by local irritation will be exhibited. Insect repellents applied to the skin at the base of the ears provide effective protection.

Chronic keratinization plaques

A form of papillomatosis with the formation of discrete white plaques on the underside of the pinnae is common. The aetiology of these plaques is not known, but some clinicians believe that they are the result of repeated insect harassment (see 13.7).

Foreign bodies

Foreign bodies rarely become trapped in the external auditory meati of horses. Even horses fed from hay racks seldom gather grass seeds at this site. *Acute onset* of aural discomfort, holding the affected side of the head down, rubbing, otorrhoea and headshaking are the likely presenting signs.

Neoplasia: sarcoids

The pinnae and surrounding tissues are common sites for the development of equine sarcoids. The full range of lesions including occult, verrucose, nodular and fibroblastic may be found at this location.

The diagnosis is most often based on physical appearance, but differentiation from other rarer integumental tumours requires biopsy sampling.

Surgical excision at this site can be difficult and is likely to be followed by recurrence. The pinna is not a good site for cryotherapy as distortion of the auricular cartilage leaves a poor cosmetic result.

Topical application of heavy-metal cytotoxic cream has been advocated for this condition, or solitary sarcoids may be treated by intralesional BCG (bacillus Calmette–Guérin) injections.

Neoplasia: other ear tumours

Other tumours which may occur in and around the external ear include squamous cell carcinoma, adenoma, adenocarcinoma and, in grey horses, melanoma.

5.2 DISEASES OF THE MIDDLE EAR

Anatomy and function of the middle ear

The middle ear comprises the space within the tympanic bulla and includes the ear drum, auditory ossicles and the tensor tympani and stapedius muscles. The internal auditory tube runs from the rostral wall of the middle ear to the nasopharynx and provides the means for pressure equilibration across the tympanic membrane and mucus clearance from an enclosed space. The middle portion of this tube passes through a diverticulum, the guttural pouch, which is considered separately (see 5.3). Adjacent to the middle ear lie the facial nerve as it emerges from the skull and the tympano-hyoid articulation. The outer wall of the tympanic bulla is visible during endoscopy of the guttural pouch.

Otitis media

Infections may become established in the middle ear after rupture of the ear drum, by microbial passage from the guttural pouch, and by haematogenous spread. Otitis media is rarely identified as an isolated entity in the horse, but more frequently it is recognized after infection has extended to involve adjacent structures (see the next two sections).

Horses with otitis media are likely to present with aural discomfort and a tendency to hold the head rotated with the afflicted ear down.

Purulent otorrhoea may arise when the ear drum has ruptured from the middle ear outwards. The middle ear is a difficult structure to investigate in the living horse. Otoscopy and palpation of the ear drum are unreliable techniques to confirm defects.

Endoscopy of the auditory tube diverticulum (ATD) (see 5.3) permits inspection of the ventral aspect of the tympanic bulla and of the internal os of the eustachian tube.

The tympanic bulla is not a good subject for diagnostic radiographs because of superimposition of the petrous temporal bones. However, where the facilities are available, scintigraphic and CT scanning may produce useful images.

Temporohyoid osteoarthropathy (THO)

THO comprises a proliferative osteitis of the petrous temporal and proximal stylohyoid bones. The aetiology of the underlying disorder is not known, and the presence of infection is rarely confirmed. A degenerative inflammatory process is most likely involved. Ankylosis of the joint between the stylo-hyoid and temporal bones is a common feature of THO. The consequences of this ankylosis include pathological fractures through the middle and inner ears causing peripheral vestibular signs (see Chapter 11) or through the stylo-hyoid the effect of which is to limit the horse's ability to move the tongue. The first sign of temporo-hyoid osteitis may be facial palsy when the facial nerve is compressed by the expanding bony lesion as it passes through the dorsal recess of the lateral compartment of the ATD. The diagnosis can be confirmed by endoscopy of the ATD, and the consequences can be relieved by osteotomy of the keratohyoid, thus pre-empting the pathological fracture.

Otitis interna/peripheral vestibular disease

The inner ear includes the end organs of hearing and proprioception, i.e. the cochlea, semi-circular canals, utricle and saccule.

Deafness is practically unrecorded in horses but whether this reflects an absolute rarity or a lack of observation by attendants is unclear.

The term otitis media should *not* be used to describe the state of neurological disturbance where there is head tilt, ataxia, circling and nystagmus; this is indicative of disturbance of the vestibular system, particularly the peripheral vestibular apparatus of the inner ear.

Otitis interna may arise as an extension of suppurative otitis media or be concurrent with osteitis of the petrous temporal bone (THO – see above).

The diagnosis is based on the presenting signs, the exclusion of other neuropathies such as cervical vertebral stenotic myelopathy ('wobbler syndrome'), endoscopy of the ATDs and diagnostic imaging of the area.

5.3 DISEASES OF THE AUDITORY TUBE DIVERTICULUM (ATD) (GUTTURAL POUCHES)

Anatomy and function of ATDs

The ATDs are balloon-like structures lying between the base of the cranium dorsally and the pharynx and oesophagus ventrally. The volume of each pouch is approximately 300 mL, and medially the two ATDs are in contact with one another, divided only by a thin layer of areolar tissue. The stylohyoid bone divides each pouch into lateral and medial compartments.

Each pouch is in contact with the base of the skull so that those structures which enter and leave the cranium through the foramen lacerum must cross the pouch: internal carotid artery, cervical sympathetic nerve and cranial nerves IX (glossopharyngeal), X (vagus) and XI (accessory).

The facial nerve (VII) lies in the submucosa of the lateral compartment dorsally.

The internal maxillary artery and vein cross the wall of the lateral compartment, and here the pouch lies beneath Viborg's triangle.

The walls of the ATDs are lined by ciliated columnar mucous membrane, and a dynamic clearance system removes mucus and particulate debris.

The drainage ostia are slit-like openings under cartilaginous flaps which lie on the dorso-lateral wall of the pharynx, and they are quite close together, separated by the pharyngeal recess. Thus, although slight discharges from one pouch may produce a predominantly unilateral nasal discharge, the more copious the discharge, the more likely it is to be bilateral.

Signs of ATD diseases

The signs of disorders of the guttural pouches reflect either compression of adjacent organs when they become distended or damage to the structures which cross them.

Swellings of the ATDs may be visible externally at the parotid region or will partially obstruct the pharynx leading to dyspnoea or dysphagia.

Erosion of the internal carotid artery can cause spontaneous haemorrhage at the nares which on occasions is so severe that the horse exsanguinates.

ATD disorders provide the potential for a very wide range of neuropathies to such an extent that the possibility of guttural pouch disease should be considered practically whenever a horse is presented with a cranial nerve deficit.

Physical examination of ATDs

External palpation in the parotid area is helpful to detect swellings produced by tympany, empyema, infection of adjacent lymph nodes or neoplastic foci particularly in the parotid lymph nodes.

On occasions guttural pouch mycosis may produce painful foci deep to the base of the ear when the head of the stylohyoid bone or the tympanohyoid articulation has become involved. However, this condition rarely produces an external swelling.

Endoscopy of ATDs

The simplest way to pass an endoscope into each pouch is by using a wire leader passed through the biopsy channel of the endoscope. This channel is invariably eccentric, and thus the wire can be used to raise the cartilage flap before advancing into the duct beyond. This manoeuvre can only be successful when the ostium is approached via the ventral nasal meatus.

Apart from looking for abnormalities inside the ATDs, endoscopy should be used to examine the larynx and pharynx for extension of ATD disease into those structures.

Depression at the pharyngeal recess and partial obscuring of the larynx may result from distension within the ATD.

Pharyngeal and laryngeal hemiplegia can result from mycotic infections.

Radiographic examination of ATDs

The ATDs are normally filled by air, and the contrast thus provided makes for good diagnostic radiographs.

The erect lateral projection is used and may reveal free gas/fluid interfaces, loss of air contrast through replacement by inspissated pus or soft tissue substitution e.g. indentations from lymph node swelling in the caudal wall.

Complete occlusion of an ATD can occur in cases of chronic empyema or chondroid formation.

Topical treatment of ATDs

A 35-cm long, 30-mL Foley balloon catheter offers a suitable indwelling device for repeated topical infusion of medication. The catheter is advanced up the ventral nasal meatus using a metal stiffener slightly bent at the leading end. This is rotated under the cartilage flap of the ostium which lies on the lateral pharyngeal wall at the same level as the eye.

Long-term catheterization may lead to weakening of the ostium and erosion of the cartilage flap.

Surgical approaches to ATD

1. **Hyovertebrotomy.** An incision is made parallel and immediately cranial to the wing of the atlas. The parotid salivary gland is reflected forwards. An endoscope introduced into the ATD *per nasum* serves to illuminate the membranous lining deep in the surgical site once the loose connective tissue has been bluntly separated. When this approach is used the ATD is entered through the lateral wall of the medial compartment where it projects caudal to the stylohyoid bone.
2. **Viborg's Triangle.** Access to the ATDs by this approach is very restricted except in conditions where there has been stretching of the tissues through distension of the pouch which in turn increases the overall size of Viborg's triangle.
3. **Paralaryngeal (Whitehouse) approach.** With the horse in dorsal recumbency, a ventral midline incision is made over the larynx. The dissection passes lateral to the larynx, trachea and cricopharyngeal muscle to reach the pouches ventro-medially. Entry to the ATD is again made medial to the stylohyoid bone. The depth of incision limits the value of this approach.
4. **Modified Whitehouse approach.** The site of the incision corresponds to that used for prosthetic laryngoplasty, i.e. it lies

ventral to the linguo-facial vein and then follows the same route to enter the pouch. This approach may be used in the standing sedated patient for the removal of chondroids when the horse is considered a poor risk for general anaesthesia.

5.4 GUTTURAL POUCH TYMPANY

Aetiology

In this condition the ATD ostium acts as a non-return valve so that air can enter the pouch but cannot escape.

Clinical signs and diagnosis

This is a condition of foals which usually manifests itself within a few days of birth and it is thought to arise from a congenital malfunction of pharyngeal opening of the pouch rather than a physical obstruction.

The disorder appears to be more common in fillies than in colts and is almost invariably unilateral. Arabian horses are most susceptible.

Air accumulates and produces a tympanitic swelling in the parotid region which is initially non-painful and non-inflammatory.

Established cases invariably show evidence of opportunistic infection because a mucopurulent nasal discharge is generally present by the time afflicted foals are submitted for corrective surgery.

The laxity of the medial septum between the ATDs may lead to swelling on the normal side, and hence false diagnoses of bilateral tympany may be made.

Dysphagia and dyspnoea may be exhibited by virtue of the size of the distension.

Occasionally ATD tympany is an acquired disorder of the adult horse.

Treatment

Three principles have been applied for the relief of ATD tympany:

1. Dilation of the pharyngeal ostium on the affected side.
2. The creation of a fistula between the normal and distended pouches by the removal of a section of the medial septum.
3. Blunt fistulation between the ATD and the pharyngeal recess.

A simple conservative technique to remedy the disorder consists of the long-term implantation of an indwelling Foley catheter placed through the defective pharyngeal ostium *per nasum* and left in place for up to 8 weeks.

The purpose of the medial wall fistulation technique is to facilitate the egress of air from the abnormal ATD through the pharyngeal ostium of the normal side. Transendoscopic laser surgery provides an option for non-invasive fistulation through the medial septum when this facility is available.

Prognosis

The prognosis for ATD tympany is usually favourable regardless of whether the catheterization or fistulation technique is used. Delayed treatment may lead to dyspnoea in later life if the tissues have become irreversibly over-stretched.

5.5 DIVERTICULITIS OF THE GUTTURAL POUCH

Aetiopathogenesis

These are poorly defined and poorly understood conditions in which inflammation of the mucous membrane lining of the ATDs occurs and which can loosely be termed 'diverticulitis'. They include strangles infection in the lymphoid tissue of the walls of the pouches, empyema, chondroid formation and chronic diverticulitis.

A catarrhal inflammation of the ATD mucosae probably accompanies most upper respiratory tract bacterial and viral infections, and the tympany described above is also likely to be accompanied by inflammatory changes, hence the term 'tympanitis'.

'Strangles'

See Chapter 19.

This is an infectious condition of horses caused by *Streptococcus equi* which consists of a suppurative lymphadenitis, particularly of the lymph nodes, associated with the upper respiratory tract including the ATDs.

Occasionally the free movement of air through the drainage ostia of the ATDs is obstructed by the physical presence of the lymphadenopathy, and a variable degree of tympany may be exhibited.

Temporary tracheotomy is indicated for those horses which show life-threatening airway obstruction.

Chronic ATD empyema and chondroids

Empyema of the ATDs occurs when mucus and/or pus accumulates within the pouches because it fails to drain satisfactorily.

The primary aetiological factor in ATD empyema is a dysfunction of muco-ciliary clearance followed by stagnation of mucus, opportunist bacterial infection and finally purulent exudation. There is a possible aetiological association with tympany.

Regardless of the precise aetiology of empyema, pus which is stagnant within the pouch eventually becomes inspissated and progressively leads to the formation of solid concretions – *chondroids.*

Horses with chondroids should be regarded as carriers for *Streptococcus equi* infection unless proven otherwise.

- The clinical signs of empyema include a bilateral purulent nasal discharge and swelling of the parotid region.
- The distension of the affected pouch into the pharynx may produce obstructive dyspnoea. The nasal discharge is sometimes malodorous.
- Lateral radiographs confirm the loss of air contrast from within the ATD, and if the pus is still fluid, an air/fluid interface will be demonstrable.
- An indwelling self-retaining Foley balloon catheter may be used for drainage of the ATD and for long term irrigation in the management of chronic cases.
- Inspissated caseous pus/chondroids may be liquefied by a process of repeated lavage via the pharyngeal ostium aided by the instillation of acetylcysteine.
- Chondroids which do not respond to conservative management may be removed individually using trans-endoscopic grasping forceps if they are small in number; otherwise surgical removal is required to extirpate chondroids.
- Whenever chondroids are bilateral the ventral Whitehouse approach is preferred so that both pouches can be entered through the same incision.

Chronic ATD diverticulitis

Chronic catarrhal inflammation of the ATDs in horses may be encountered with no history to suggest previous strangles infection.

Chronic diverticulitis without the presence of empyema may present with a syndrome of neuropathies where any combination of deficits of the glossopharyngeus, vagus, facial, spinal accessory and sympathetic nerves may be present. It is assumed that the nervous pathways

are damaged as an extension of the inflammatory process in the ATD walls.

The diagnosis is established by a functional assessment of the cranial nerves mentioned, combined with the endoscopic identification of a roughened thickening of the ATD lining. The nervous form of diverticulitis carries a poor prognosis.

5.6 GUTTURAL POUCH MYCOSIS

Aetiopathogenesis

The fungal plaques of guttural pouch mycosis (GPM) are usually found in one of two characteristic sites:

- The majority on the roof of the medial compartment.
- Others on the lateral wall of the lateral compartment.

There is a close association between the predilection sites and the underlying internal carotid (ICA) and external carotid (ECA) arteries respectively.

Clinical signs

- The development of an invasive fungal plaque on the mucosal wall of the ATD of a horse will produce consequences ranging from nil to fatal.
- Spontaneous epistaxis at rest is the most frequent sign noted by owners and usually consists of a small quantity of fresh blood at one nostril in the first instance.
- A number of further minor haemorrhages may follow but, if untreated, exsanguination is a probable final outcome.
- It is unusual for the first episode of epistaxis to be fatal, but the course of the disease from first to final haemorrhage rarely spans more than three weeks and is more likely to be a matter of days.

Pharyngeal paralysis is the most frequent neuropathy which accompanies GPM and the inclusion of ingesta in a nasal discharge from any horse is an indication to inspect both ATDs by endoscopy as well as to assess pharyngeal function.

Endoscopic evidence of pharyngeal paralysis includes persistent dorsal displacement of the palatal arch, the presence of saliva and ingesta in the nasopharynx, weak pharyngeal contractions and a failure of one or both of the pharyngeal ostia of the ATDs to dilate during deglutition (see Figure 1.2).

Laryngeal hemiplegia is the next most frequent cranial nerve deficit encountered in horses with GPM but it is rarely responsible for the only signs observed by the owner.

GPM may produce a wide range of other signs referable to the head and upper neck. These include facial palsy and Horner's syndrome; reluctance to lower the head to the ground and stiffness in the upper neck; parotid pain; otorrhoea; epiphora and photophobia.

Abnormal head posture may be associated with pain in the atlanto-occipital joint when the mycosis has extended into this joint.

Diagnosis

The clinical signs are not specific, but whenever a horse is presented with spontaneous epistaxis the possibility of GPM should be explored because delayed treatment may result in a fatal outcome.

A definitive endoscopic diagnosis of a mycotic plaque in the ATD (Figure 5.1) is not always straightforward; two caveats should be heeded:

- First, the stress of handling the horse may precipitate a fatal epistaxis.

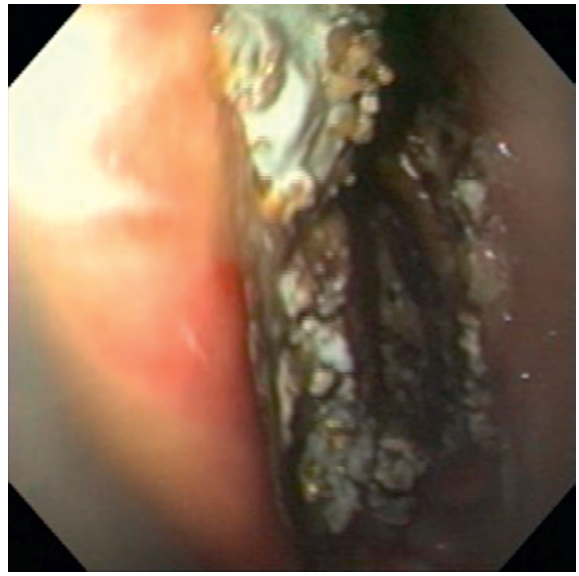


Figure 5.1 Guttural pouch mycosis. The mycotic plaque lies on the internal carotid artery in the dorsal region of the medial compartment of the right ATD.

- Second, endoscopic visibility within the affected pouch may be poor after a recent haemorrhage, and accurate location of the lesion will not be possible.

If the epistaxis has been recent, it is sufficient to identify the stream of blood flowing from the pharyngeal drainage ostium.

In all cases of mycosis, the contra-lateral pouch should be checked for extension of the disease through the medial septum and for concurrent bilateral mycosis.

A full endoscopic assessment of laryngeal and pharyngeal function is required for an accurate prognosis.

Treatment

- Medical treatment of GPM, be it by the use of topical or systemically administered antimycotic drugs or by a combination of both, is likely to be unsuccessful.
- Surgical occlusion of the branches of the carotid artery is recommended.
- The treatment of choice is transarterial coil embolization.
- The internal carotid is not an end-artery and therefore the placement of a simple ligature on the cardiac side of the lesion will not always be successful because retrograde haemorrhage may occur.
- Occlusion of the distal segment with a balloon-tipped catheter combined with proximal ligation of the ICA is superior.
- Topical antimycotic medication as an adjunct to arterial occlusion is probably not necessary.

Prognosis

Those cases of GPM not showing neurological complications can usually be brought to a successful conclusion by arterial occlusion surgery.

Although a small proportion of horses showing pharyngeal paralysis recover normal swallowing function, destruction on humane grounds is a more likely outcome and is indicated as soon as the patient shows signs of dehydration or aspiration pneumonia.

Laryngeal hemiplegia resulting from GPM can be managed as for the idiopathic form recurrent laryngeal neuropathy (see 'Treatment' in 5.20).

5.7 OTHER ATD DISORDERS

Trauma

The rectus capitis ventralis muscles insert onto the basisphenoid bones which form a narrow bridge between the foramina lacera in the dorso-medial aspect of the ATDs.

Fractures at the junction of the basisphenoid and occipital bones may occur in horses which have sustained violent head trauma by rearing over backwards. Some patients with this injury are unable to stand but show collapse and epistaxis.

Endoscopy can be used to confirm that the origin of the epistaxis lies in the ATDs, and radiography can also make a valuable contribution to diagnosis.

Apart from blood showing as free fluid in the floor of the ATDs, a step in the base of the skull will be visible on radiographs.

The condition is irreparable, but those horses which are able to stand may make a full recovery albeit after a prolonged period. Others are rendered partly quadriplegic, and humane destruction is indicated.

Foreign body penetration into the retropharyngeal tissues by wire can occur in horses. The clinical signs include epistaxis, nasal discharge, dyspnoea, dysphagia and pain in the upper neck with restricted movement. The radiographic identification of metallic material in the caudal wall of the ATD is straightforward but projections in two planes are required for an accurate stereotactic location of the object.

Neoplasia

All regions of the horse richly endowed with lymphoreticular tissue are potential sites for lymphoma development, and this tumour can arise in the tissues abutting onto the ATDs.

Ectopic melanosis is a common feature of the mucosa of the ATD of grey horses. The usual site for this is in the lateral compartment over the internal maxillary vessels. Primary melanomas can arise here also (see 5.29).

5.8 DISORDERS OF THE EXTERNAL NARES

Anatomical features

The nostrils contribute more than 50% to the total resistance to flow of the entire upper respiratory tract during quiet breathing. This can be reduced considerably by active dilation during exertion. The C-shaped alar cartilages, back-to-back at the midline, provide rigidity for the otherwise soft structures of the external nares. Dilation of the nostril margins is achieved through the action of the nasolabialis muscles which receive their motor supply through the dorsal buccal branches of the facial nerves. The alar folds which attach to the ventral conchus mark the dorsal margin of the airway through the nasal vestibule, although the blind pocket of the false nostril lies above this.

The nostril margins and the structures which support and dilate them are of paramount importance to the sports horse. Any disorder which leads to collapse at this level is likely to render a horse useless for athletic pursuits.

'Wry nose'

- A congenital deformity of the nose resulting from gross foreshortening of the premaxilla on one side.
- The deformity is not thought to be genetically transmitted but may be due to abnormal positioning in utero.
- The deformity is externally obvious.
- Even radical surgery is unlikely to render the horse suitable for exercise.
- Deviation of the nasal septum is usually accompanied by a degree of wry nose which may be very subtle on occasion and is most easily recognized through incisor malocclusion.

Hypertrophy of alar folds

- It is normal for the alar folds which form the floor of the false nostril sometimes to vibrate during exhalation. This is the origin of the vibrant expiratory noise known as 'high blowing'. Occasional horses exhibit a comparable vibrant sound during inspiration.
- Confirmation that the origin of the noise lies at the alar folds is achieved by placing full thickness mattress sutures from the skin at the dorsal aspect of the nose across the openings of the false nostrils. The technique is performed under local anaesthesia so that the noise can be compared before and after suture placement. A positive result is an indication for resection of the alar folds.

'Atheroma' of false nostril

- Epidermal inclusion cysts (sebacious cysts) occasionally develop in the lining of the false nostril.
- These are painless and can be seen at the dorsal aspect of the nose rostral to the naso-maxillary notch.
- Although the swellings can be sizeable they do not obstruct respiration and are of cosmetic significance only.
- Chemical ablation of the lining by flushing with 10% formalin solution after evacuation of the contents is usually effective. The formalin is left in situ for 5 minutes before being flushed clear with sterile saline solution.
- Otherwise removal by careful dissection from the dorsal skin surface is straightforward, and recurrence is improbable.

Facial paralysis

See Chapter 11.

Flaccidity of a nostril margin through dysfunction of the nasolabialis muscle represents an aerodynamic disaster to an athletic horse. Many cases of facial palsy are of iatrogenic origin, and during facial surgery, i.e. dental extractions, the branches of the facial nerve should be respected. Care should be taken to avoid trauma to the nerve when horses are recumbent or recovering from general anaesthesia.

Trauma

- Wounds to the nostrils are typically sustained when a horse pulls backwards having caught the margin of its nose on a hook.
- These injuries demand meticulous management in the acute stage with layer-by-layer anatomical restoration.
- Inadequately managed nostril tears lead to stricture of the nostril aperture and a major reduction in athletic capacity.

Alar cartilage necrosis

Neglected penetration injuries of the alar cartilages may be followed by the extension of suppuration into the cartilage matrix. Careful resection of the diseased tissue is required because loss of mechanical stability at this location represents a serious complication.

5.9 DIAGNOSTIC APPROACH TO NASAL AND PARANASAL SINUS DISEASE

Anatomical considerations

The paranasal sinuses are extensive air-filled spaces lined by mucoperiosteum. The normal removal of mucus is a dynamic process depending on muco-ciliary flow to the drainage ostia which do not lie at the lowest points in the sinuses. Once the nasal meati are reached mucus is lost by a combination of evaporation and further muco-ciliary flow towards the nasopharynx. The aetiopathogenesis of primary sinusitis in horses hinges on stagnation of mucus in the sinus cavities through inhibited dynamic clearance.

The five paired paranasal sinuses of the horse are:

1. Frontal/conchofrontal.
2. Caudal maxillary.
3. Rostral maxillary.
4. Ethmoidal.
5. Sphenopalatine.

The frontal sinus is divided into conchofrontal (CFS) and frontal (FS) portions. Drainage takes place through the fronto-maxillary foramen into the caudal maxillary sinus (CMS). The ethmoidal and sphenopalatine sinuses also drain via the CMS into the middle nasal meatus.

The rostral maxillary sinus (RMS) has an independent drainage ostium, again into the middle nasal meatus.

The RMS is divided into a lateral bony and a medial turbinate portion within the ventral conchus (ventral conchal sinus, VCS). They are separated by the infra-orbital canal and a sheet of bone joining it ventrally to the roots of the cheek teeth. In the young horse the lateral bony compartment is largely occupied by the roots and reserve crowns of the cheek teeth, and regardless of age, the VCS is not easily accessible for surgery other than via the CFS.

- The apices of the 4th, 5th and 6th cheek teeth (109, 110, 111, 209, 210 and 211) lie within the maxillary sinuses; they are most prominent in young horses and recede towards the floor of the sinuses with age.
- The roots of the 3rd cheek tooth (108 and 208) form the rostral wall of the RMS.
- When dental periapical suppuration is the cause of sinusitis and oral extraction is unsuccessful, surgical exodontia requires that the roots are approached through the sinuses.
- Structures such as the nasolacrimal canal, infra-orbital canal, vein and artery of the angle of the eye are vulnerable to iatrogenic insult.

Common presenting signs

- The clinical signs of paranasal sinus diseases almost invariably include a nasal discharge, which may be mucoid, purulent, haemorrhagic or a combination of these.
- There may also be facial swelling and obstructive dyspnoea.
- Some expansive lesions in this area displace orbital tissues resulting in exophthalmos, but it is exceptional for a sinus disorder to extend caudally into the cranium to provoke central nervous signs.
- The intranasal structures are richly vascular, and it is not surprising that trauma and destructive conditions frequently lead to epistaxis.

History

- Note should be made of possible contact with infectious respiratory disease and of the duration and nature of any nasal discharge.
- It is unusual for sinusitis to be bilateral, and it is logical that the discharge will be largely unilateral when its origin lies proximal to the caudal limit of the midline septum.
- When a horse is presented with unilateral epistaxis enquiries should be made regarding associations with exercise to eliminate a diagnosis of exercise-induced pulmonary haemorrhage (see 6.7).
- Epistaxis due to guttural pouch mycosis may be acute, and, even if episodic, the course of the history is unlikely to exceed 3 weeks (see 5.6).
- A diagnosis of progressive ethmoidal haematoma (PEH) is more likely to be correct when episodes of epistaxis span a longer period, especially if the blood is not fresh.
- A foetid nasal discharge points to suppuration, but this could arise from a wide range of chronic sinus lesions.

Physical examination

- The facial area should be inspected for evidence of deformity of the supporting bones through swelling or trauma.
- Subcutaneous emphysema may be detected after trauma in some cases where the sinus walls have been disrupted.
- Percussion of the walls of the paranasal sinuses is an unreliable technique, but increased resonance may be perceived when the walls become thin, or dullness may develop when the sinuses are completely filled by fluid or soft tissue.
- The airflow at each nostril should be checked to assess obstruction of the nasal meati.
- The clinical crowns of the cheek teeth are examined for the presence of fracture, displacement or impaction of degenerate ingesta.
- The patency of the nasolacrimal duct can be checked by catheterisation and infusion of saline solution from either end.

Endoscopy

Endoscopy of the nasal area is performed in two ways:

- First, by conventional passage of the instrument into the nasal meati.
- Second, by direct inspection of the sinus contents through small trephine holes.

The latter technique is most commonly performed into the CFS or CMS. All endoscopy of this region is best performed on the standing horse because orientation is straightforward and the nasal tissues of a recumbent horse become discoloured and engorged.

Nasal endoscopic checklist:

1. Nasal meati – are they narrowed? Compare with contralateral side.
2. Is narrowing of the meati the result of conchal distension?
   - Dorsal concha – CFS.
   - Ventral concha – VCS.

3. Is narrowing due to a soft-tissue mass? Does the colour of the mass indicate a PEH, cyst or tumour? Can a mass be seen extending caudal to the midline septum when the endoscope is passed via the opposite nostril?
4. Sinu-nasal drainage ostium in caudal middle meatus:
   - Is there discharge/blood present?
   - Is the ostium dilated or compressed?
5. Ethmoidal labyrinth:
   - Is there any sign of blood/PEH?
6. Conchal mucosa, check for:
   - Mycosis.
   - Ulceration/haemorrhage.
   - Necrosis.
7. After surgery, check for:
   - Evidence of persistent infection including mycosis.
   - Satisfactory sinu-nasal fistulation.
8. Direct sinus endoscopy, check for:
   - Empyema.
   - Dental periapical reactions.
   - Mycosis.
   - PEH.

Radiography

The good contrast provided between bone and air renders the nasal chambers and paranasal sinuses excellent candidates for radiographic diagnosis (Figure 5.2). Erect lateral, lateral oblique, lesion-oriented oblique and ventro-dorsal views may be required for a comprehensive investigation. The radiographic signs of sinu-nasal disease include:

- Free fluid interfaces in the sinuses.
- Loss of normal air contrast through substitution by fluid or soft tissue.
- Depression or elevation of the supporting bones of the face.
- Distortion of normal structures such as the tooth roots, sinus walls, midline septum and infra-orbital canals.

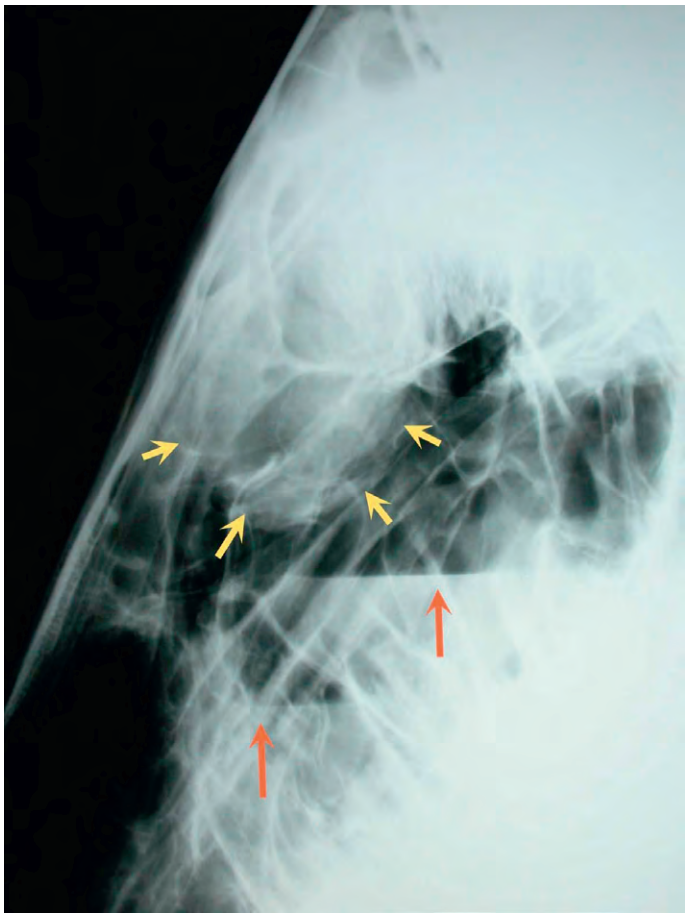


Figure 5.2 Erect lateral radiograph of the paranasal sinuses showing free fluid lines (long arrows) and a soft tissue mass (short arrows).

Other imaging modalities

- Scintigraphy.
- Computed tomography (CT).

Scintigraphy has emerged as a useful means to identify active bone turnover, often before radiographic changes are seen, and it is particularly applicable to discriminate between dental apical disease and primary sinusitis.

The complex anatomy of the equine head, with the frequent superimposition of normal and abnormal structures, renders the interpretation of radiographs difficult. CT scanning offers a precise means to locate abnormalities (Figure 5.3).

Biopsy

Even after full endoscopic and imaging investigations, surgical exploration may be performed before a specific diagnosis has been made. Suspect tissues should be biopsied to differentiate neoplastic disorders from other disease processes.

Figure 5.3 A transverse CT section through the head at the level of the first upper molars. The arrows point to three sites of progressive ethmoidal haematoma development.
Courtesy of Dr Renate Weller, Royal Veterinary College.

5.10 TREATMENT FOR SINU-NASAL DISORDERS

Treatment objectives

- Accurate diagnosis of the primary sinu-nasal disorder and removal of diseased tissue.
- Restoration of the normal drainage mechanisms or the creation of alternative drainage through a sinu-nasal fistula.
- Adequate visibility within the sinuses and nasal meati for accurate diagnosis and surgery.
- Means to control haemorrhage during surgery and in the recovery period.
- A safe airway during surgery and recovery.
- Facilities for topical post-operative treatments and for monitoring progress.
- An early return to exercise.
- A pleasing cosmetic result.

Medical management

Non-surgical treatments for sinusitis include antibiotics, mucolytics, steam inhalations, volatile inhalations and continued controlled exercise. The objective is the return of normal muco-ciliary clearance. Most cases of simple primary sinusitis will be resolved either naturally or with minimal veterinary assistance provided that the necessary supportive management is instituted promptly.

Trephination

- Simple trephination also aims to restore normal sinus drainage but with the addition of irrigation and topical antibiotic infusion to clear stagnant mucus and eliminate secondary infection.
- Trephination is performed using local anaesthetic infiltration with the horse standing.
- The preferred site for trephination is into the CMS in the angle formed between the margin of the bony orbit and the facial crest.
- In-dwelling balloon catheters provide a good means for regular irrigation over a number of days until the discharge to the nostril ceases.
- The RMS is less available for simple trephination especially in young horses where the lateral compartment is largely occupied by the roots and reserve crowns of the third and fourth cheek teeth.

Facial flap surgery

In the face of chronic sinusitis, sinus cyst and PEH the natural drainage system may well be physically obstructed. Fistulae can be made by removal of the floor of the CFS and medial wall of the VCS so that there is free communication between the sinus cavities and the nasal meati. Extensive fronto-nasal flap surgery is required for this and additional drainage from the CMS is achieved by removal of the septum dividing it from the RMS.

- The bulla of the RMS may bulge caudally into the CMS when it is inflated by pus, and this is easily punctured and excised.
- This form of fistulation may be performed through a trephine hole into the CFS before introducing tubing for subsequent irrigation and drainage.
- In surgical practice the fistulae described provide convenient routes by which to lead sock-and-bandage pressure packs to the nostrils (see below).
- Fronto-nasal or maxillary flap surgery is required for extensive excisional procedures such as the removal of sinus cysts, PEHs and selected tumours as well as for the relief of chronic sinusitis and fistulation techniques.
- Radical exposure of the nasal chambers, paranasal sinuses and their contents can be achieved through the bony walls of the supporting bones.

The surgical options are:

- whether to perform the surgery with the patient standing using local anaesthetic infiltration or under general anaesthesia. In general, standing surgery is preferred, and general anaesthesia should be reserved for those instances where haemorrhage is expected to be severe and when exceptional sensitivity is possible where the infra-orbital nerve may be exposed.
- between frontal and lateral (maxillary) approaches.
- whether to reject or preserve the bone flap.

Lesser decisions relate to the shape of the bone flap and the direction in which the skin/periosteal flap is raised.

The frontal approach into the sinus compartments (Figure 5.4) is preferable because it is more versatile and generally provides superior access. The lateral route into the maxillary sinuses provides limited access and should be reserved for those instances where the disease process is restricted to the lateral areas of the maxillary compartments.

Incisions are made through skin and periosteum in the same plane before the periosteum is peeled away from the underlying bone.

The bone flap is best fashioned with a 5-cm diameter trephine or an oscillating saw, but the osteotomy should be made at least 1 cm inside the skin/periostium incision so that the later repair is supported by bone.

Once the sinus contents are exposed, the disease focus is identified and removed.

Closure of the incision can be achieved with a single layer of mattress sutures, but accurate alignment is required for the best cosmetic result in horses with natural facial markings.

Figure 5.4 Frontal flap exposure of the sinus compartments – in this case to drain a large quantity of stagnant pus.

Care must be taken to provide circulatory support by aggressive intravenous fluid administration particularly when prolonged surgery may render an anaesthetized horse vulnerable to rhabdomyolysis of the dependent limbs.

Pressure packing within the sinus cavities and nasal chambers is essential to control haemorrhage on completion of the surgery and during the initial recovery period, i.e. 48–72 hours. Sock-and-bandage packing consisting of lengths of cotton bandage packed into tubular stockinet socks is suitable. The open end of the stockinet is led to the nostril.

For anaesthesia, a cuffed endotracheal tube is essential to protect the lower airways from the inhalation of blood, pus, surgical debris and irrigation fluids during surgery.

Vigorous lavage, possibly from a hosepipe, is frequently used to displace inspissated pus from the recesses of the sinuses.

During anaesthetic recovery, until the horse regains its feet, a nasopharyngeal tube should be placed through the contra-lateral nasal chamber and secured at the nostril.

A Foley balloon catheter implanted into the CMS (see above) offers a convenient route for post-surgical irrigation and medication.

Blood clots and devitalized tissue are inevitably left after facial flap surgery and opportunist infections, frequently mycoses, are likely.

The topical medication should include an antimycotic agent.

Inspection of the sinus contents to monitor progress after sinus flap surgery can be achieved by endoscopy *per nasum* through the iatrogenic fistulae or laterally through the trephine hole into the CMS.

In spite of the radical nature of some sinus surgical procedures an early return to exercise is recommended as an integral part of treatment.

Forced nasal ventilation increases the evaporation of residual discharges when stagnation might otherwise encourage post-operative infections to become established.

The cosmetic results of trephination and facial flap surgery are usually satisfactory with no more than a slight depression at the operative site.

5.11 PRIMARY AND SECONDARY EMPYEMA

Aetiopathogenesis

Primary sinus empyema results from the stagnation of mucus through inhibited dynamic muco-ciliary clearance, generally by infection with upper respiratory tract viral agents. Initially the dependent portions of the sinuses fill with mucus which passively spills through the drainage ostia into the nasal meati.

Bacterial opportunism follows exacerbating the mucosal inflammation and leading to purulent exudation (Figure 5.4).

- Hyperplasia of the sinus lining, a feature of chronic sinusitis, narrows the drainage ostia.
- Inspissation of pus develops in the later stages and also occludes the ostia.
- The conchal walls of the sinuses are not rigid, and in the face of obstructed drainage there is a tendency to inflation and thereby to obstruction of the nasal airways.
- Secondary (dental) sinusitis is a sequel to dental periapical suppuration.
- The roots of the fourth to sixth maxillary cheek teeth lie within the maxillary sinuses and are covered by a thin layer of alveolar bone and mucosa. Whenever these teeth are devitalized by fracture or infundibular necrosis there is the possibility of secondary sinusitis.
- The third maxillary cheek tooth forms the rostral wall of the RMS and apical suppuration here is more likely to lead to a facial swelling than to sinusitis.

Clinical signs

- In the acute stages primary sinus empyema produces a mucus-based unilateral nasal discharge.
- As the condition becomes more chronic, the discharge becomes more purulent and malodorous.
- The discharge of secondary (dental) sinusitis is invariably purulent and malodorous. It is generally less profuse than with primary empyema.
- Primary and secondary sinusitis are rarely bilateral conditions.
- The discharge is occasionally blood-flecked but rarely primarily bloody.
- Facial swelling, over the maxillary sinuses, and nasal obstruction are later features of primary empyema.

Swelling over the maxillary sinuses rarely occurs with secondary dental sinusitis. By contrast, facial swellings rostral to the facial crest are usually caused by dental periapical suppuration.

Diagnosis

- Based on the history and clinical signs.
- Physical examination takes note of the nasal discharge and any facial deformity.
- The airflow at the external nares is compared.
- Percussion may show loss of resonance if the sinus spaces are filled by pus but is unreliable when filling is incomplete.
- An oral inspection, preferably using a dental endoscope, is used to check the cheek teeth for fractures, displacement or infundibular necrosis (see 1.2).
- Endoscopic features have been outlined above (see 'Endoscopy' in 5.9), but at least endoscopic examination will show a flow of discharge from the caudal region of the middle nasal meatus where the sinus drainage ostium is located.

Radiographs either show free horizontal gas/fluid interfaces or, when the sinuses are full, total loss of air contrast. It is always important to assess the possibility of dental suppuration as the cause of the sinusitis.

Treatment

- Early cases can be treated non-surgically using systemically administered antibiotics, volatile or steam inhalations and continued light exercise. (Figure 5.5).
- Those which do not respond to conservative measures should be managed by the implantation of a catheter into the appropriate sinus compartment(s), but this usually means the CMS which communicates directly with all of the sinuses except for the RMS.
- The catheter is used for topical antibiotic infusion in addition to physical irrigation. A free flow of lavage to the nostrils implies that the drainage ostia are clear, and this is an optimistic sign.
- Refractory cases, typically where there is poor drainage during irrigation and a putrid nasal discharge, should be considered potential candidates for standing facial flap surgery. Endoscopic and radiological re-assessment is used to confirm the exact location of the empyema and to decide which technique to use.
- The objective of this radical surgery in the management of chronic sinusitis is to break down the intercompartmental barriers thus converting the sinuses into a common air space and to create generous fistulae into the nasal meati.

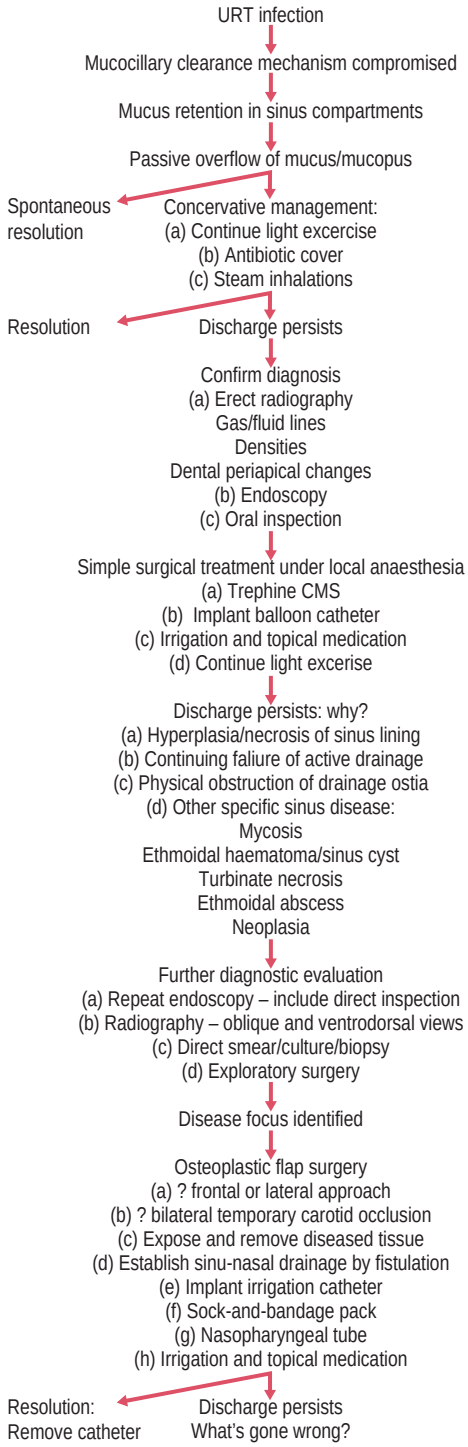


Figure 5.5 Management of primary sinusitis in the horse. URT = upper respiratory tract; CMS = caudal maxillary sinus. Reproduced with permission from Lane JG 1993 The management of sinus disorders of horses – part 1. Equine Veterinary Education 5:5–9.

Prognosis

A minority of cases of sinus empyema require any form of surgical intervention, and of those which do, the overwhelming majority are resolved by simple trephination and catheterization. Although facial flap surgery is a major undertaking it is generally successful and the few failures which do occur can generally be attributed to inadequate restoration of drainage or to incorrect diagnosis.

5.12 PROGRESSIVE ETHMOIDAL HAEMATOMA (PEH)

Aetiopathogenesis

- The aetiology of PEH is not known.
- There is no evidence that any form of neoplastic process is responsible for the repeated sub-mucosal haemorrhages which cause these relentlessly expanding lesions to develop on the surfaces of the ethmoidal turbinate labyrinth.
- Lesions may arise on the nasal or sinus aspects of the ethmoidal 'onion'.
- The mucosal capsule splits intermittently to release a bloody discharge, but the overall trend is towards expansion.
- This in turn may compromise sinus drainage with a secondary retention of mucus, or the nasal airways may become obstructed causing dyspnoea.
- In extreme cases the dyspnoea may cause distress even at rest, and the mass may be extruded at the nares.

Clinical signs

- Afflicted horses are likely to be 4 years of age or older, the prevalence increasing with age. Repeated low-grade haemorrhage from one nostril is the most common presenting sign.
- The blood is not fresh, and epistaxis is not related to exercise.
- Between haemorrhages a dirty nasal discharge is often present.
- Varying degrees of nasal obstruction will be present, and in competition horses noisy breathing may be the first symptom noted by the rider.
- A facial swelling is a late feature of PEH.
- Rare cases cause pressure on, or infiltrate through, the cribriform plate, typically ventrally so that central nervous signs including blindness are occasionally reported.

Diagnosis

Diagnosis is based on the clinical history, physical examination, endoscopy, radiography, and computed radiography (see Figures 5.2 and 5.3). Percussion of the sinuses may show increased resonance if the walls are becoming thin. Endoscopy per nasum will reveal those PEHs based on the nasal surfaces of the ethmoid. The lesions appear as smooth-walled masses pushing forward from the caudal nasal region. The colour of the lesions is very variable ranging from yellow/orange to grey/green (Figure 5.6).

Some PEHs arise on the sinus aspect of the ethmoids and are not visible *per nasum* although a stream of blood may be seen emerging from the sinus drainage ostium in the caudal middle meatus.

Direct sinus endoscopy through the CMS is required for a definitive diagnosis of these PEHs. Radiography in the standing position should reveal large PEH lesions as soft tissue masses extending rostral or dorsal to the ethmoidal 'onion' into the CFS or FS respectively (see Figure 5.2).

Treatment

- Chemical ablation using intra-lesional 4% formaldehyde solution (i.e., 10% formalin) passed though a transendoscopic needle catheter is the treatment of choice for those PEH lesions which can be seen by endoscopy.

Figure 5.6 Endoscopy *per nasum* showing an ethmoidal haematoma emerging ventral to the great ethmo-turbinate bone.

- Treatments are repeated at two-week intervals until resolved, and at least 3 treatments are usually needed.
- Lesions arising within the sinuses are problematic for chemical ablation because repeated access may be needed.
- Lesions greater than 3 cm diameter are unlikely to be successfully resolved by this technique.
- When intra-lesional chemical ablation is inappropriate or unsuccessful surgical removal via a frontal facial flap is used.
- Lesions within the sinuses are easily accessible but nasal aspect PEHs require removal of the floor of the CFS to provide the access necessary for removal.
- The lesions are rarely withdrawn without rupture of the mucosal sac but the basal area should be subjected to thorough curettage.
- Transendoscopic laser destruction is suitable for small lesions but has no advantage over chemical ablation.

Prognosis

- PEH is best considered as a multifocal lifetime disorder.
- The prognosis for successful ablation by facial flap surgery is fair because recurrence may occur in up to 30% of cases within 18 months.
- PEH can afflict horses bilaterally, and recurrences which arise after surgical ablation are as likely to be new lesions rather than the original focus re-growing.

5.13 SINUS CYSTS

Aetiopathogenesis

- The aetiology of sinus cysts is not known, but there appear to be features in common with PEHs inasmuch as the two conditions may occur concurrently, and both show histopathological evidence of repeated haemorrhage.
- Cysts typically contain vivid yellow fluid indicative of blood pigment degradation.
- Sinus cysts tend to arise in the region of the drainage ostium so that expansion either occurs into the sinus or into the nasal meati.

Presenting signs

- Nasal obstruction and facial swelling are the typical presentation signs of sinus cysts.
- The facial swelling is generally over the concho-frontal or maxillary sinuses, but a small number of cases show a midline frontal distension.
- Rarely ocular proptosis may be seen.
- A mucoid nasal discharge may be present and reflects partial obstruction of muco-ciliary clearance.

Diagnosis

- Sinus cysts tend to occur either in the first year of life or in horses over 4 years old.
- The presenting signs, physical examination, endoscopy and radiography provide the necessary diagnostic information.
- Sinus cysts can be large space-occupying lesions and can easily be confused with a neoplastic disease. However, cysts are very much more frequent in horses than nasal tumours or PEHs.
- The physical examination may reveal a considerable swelling over the facial region with or without obstruction to nasal air flow.
- Increased resonance on percussion can be expected if there is thinning of the nasal bones. Cysts which have expanded in a nasal direction can be seen at endoscopy as discrete rounded masses typically in the middle meatus.
- Those which are confined to the sinuses cause a more diffuse narrowing of the nasal airways to the point where passage of the endoscope to the nasopharynx is obstructed.
- Radiographs reveal a homogeneous soft tissue density with at least some of its margins well demarcated.
- On lateral standing radiographic views free gas/fluid lines may be seen indicative of impeded sinus drainage.
- Ventro-dorsal radiographic views are helpful to demonstrate the limits of the lesion in a rostrocaudal dimension as well as the extent of any septal deviation.
- If there is a safe available site, aspiration into the cyst provides definitive confirmation of the diagnosis by the release of characteristic vivid yellow fluid.
- Trans-ocular ultrasonography will show that the bony wall of the orbit is intact, in contrast to advanced sinu-nasal tumours which may destroy the bony orbit.

Treatment

- Fronto-nasal flap surgery is effective, and the cyst wall can be peeled away from the inside of the sinuses. Many sinus cysts can be removed in this way with the horse standing.
- Advanced cysts may cause atrophy of the infra-orbital canal with exposure of the nerve trunk within. This renders standing surgery uncontrollably painful, and general anaesthesia is required.
- In the event of incomplete ablation small areas of residual cyst tissue do not appear to cause complications.

Prognosis

The prognosis for successful surgical ablation is excellent, and recurrence is not likely.

5.14 MYCOTIC RHINITIS AND SINUSITIS

Aetiopathogenesis

Although mycotic opportunism is common after surgery or secondary to other suppurative conditions such as dental periapical infection,

horses are encountered where these infections arise on the sinu-nasal tissues without obvious underlying disease. The aetiology is not known, but the infection consists of a destructive rhinitis/sinusitis occasionally producing sinu-nasal fistulae.

Clinical signs

Horses with mycotic infections in the nasal region usually show a low-grade unilateral purulent discharge which may be malodorous. On the rare occasions when erosion of a significant blood vessel has occurred, there may be epistaxis.

Diagnosis

- The external physical examination is unlikely to be helpful.
- Similarly, radiographic findings may appear to be relatively normal with no more than small gas/fluid lines and such a finding in a horse with a notable nasal discharge is highly suggestive of a mycotic infection.
- Mycotic plaques can be identified by endoscopy either per nasum or directly into the CMS.

Treatment

- Topical medication with the benzimidazole agent enilconazole provides an effective and simple remedy for small lesions.
- A Foley balloon catheter is placed into the CMS in the usual manner, and the sinus cavity acts as a reservoir for the medication which is infused twice daily.
- Resolution may require prolonged treatment, i.e. 4–6 weeks.
- Extensive mycoses are better treated by surgical curettage through a standing frontal osteoplastic flap followed by topical medication to prevent recurrence.

Prognosis

Primary sinu-nasal mycosis carries an excellent prognosis provided that the course of treatment is sufficiently long.

5.15 SINUS AND NASAL NEOPLASIA AND POLYPS

Aetiopathogenesis

- True tumours of the sinus and nasal regions are uncommon in the horse.
- Squamous cell carcinoma, adenocarcinoma and osteomas and osteosarcomas have all been reported.
- Odontogenic tumours, i.e. tumours derived from the tooth-forming tissues are rarely encountered, usually in young horses.
- Polyps, i.e. pedunculated inflammatory proliferations enclosed in mucous membrane, are likely to represent a complication of dental periapical disease.

Clinical signs

The clinical signs are likely to be confused with those of a severe primary sinus empyema, cyst or progressive ethmoidal haematoma, i.e. a putrid nasal discharge possibly mixed with blood; nasal obstruction and facial swelling. In addition there may be ocular proptosis when the tumour has infiltrated the bony orbit and displaced the globe outwards. The more benign lesions are likely to present as causes of nasal obstruction. Some nasal polyps protrude to the nostrils.

Diagnosis

- The usual physical, endoscopic, radiographic and computed tomographic procedures which might be applicable to a case of empyema, cyst or PEH are indicated.
- In horses with proptosis, trans-ocular ultrasonography should be used to assess the integrity of the bony orbit.
- Attempts to aspirate fluid from the lesion fail to produce pus or cyst fluid, but needle aspiration could be a means to obtain biopsy material, for example after exploratory trephination.
- Radiographs may show a lesion with an outline less well demarcated than with a cyst or empyema. Identifiable mineralized dental tissue may be evident in cases of well differentiated odontogenic tumour. In cases of suspected polyp the dental features on radiographs should be reviewed and the clinical dental crowns checked by oral inspection. Computed tomography provides an effective diagnostic and prognostic technique.

Treatment

Fronto-nasal flap surgery may be used on an exploratory basis, and this may or may not lead on to extirpation depending on the feasibility of total removal.

Prognosis

The prognosis for sinu-nasal tumours in horses is highly variable and each case must be treated on its merits. Well circumscribed benign lesions often lend themselves to successful removal by fronto-nasal flap surgery, and the size of the lesion need not be a deterrent.

5.16 OTHER SINU-NASAL DISORDERS

Nasal septal deviation

Congenital deviation of the nasal septum is invariably present in cases of wry-nose but may also occur as an independent entity. It may not come to light until the patient commences training and investigations into the cause of abnormal respiratory noise take place. Some cases are not discovered until obstruction is met during routine endoscopy or naso-gastric intubation.

While a diagnosis of septal deviation may be suggested by endoscopy, the diagnosis can only be confirmed by radiography in the ventro-dorsal plane showing that the septum does not adhere to a midline position.

Resection is a feasible treatment for nasal septal disorders in horses.

Other nasal septal disorders

Hyperplasia of the nasal submucosa occasionally causes nasal obstruction in young horses, i.e. under two years of age, and is an indication for resection of the septum. Infections of the nasal septum are rare in Europe, but in other countries this is a site where granulomatous thickening may occur in response to infections such as cryptococcosis. In addition to specific antifungal medication, septal resection may be indicated to relieve the obstructive effect.

Other indications for septal resection include cystic degeneration and amyloidosis.

Nasal septal resection

A tracheotomy tube should be placed routinely before this surgery because blood loss will be significant, and post-operative bilateral nasal packing is required. Septal resection is performed, firstly, by two vertical incisions of the septum (i) just caudal to its rostral attachment to the nares, and (ii) through a frontal midline trephine hole at a level rostral to the medial canthi. These vertical incisions are joined by horizontal incisions along the ventral and dorsal margins of the septum, using either a protected chisel or embryotomy wire.

Conchal necrosis and metaplasia

Metaplastic calcification of the conchal cartilages may occur as an end-stage of dental suppuration which has extended into the nasal tissues. Horses afflicted with this condition will be presented with a foetid nasal discharge, but signs directly referable to dental disease may not have been noted. Endoscopy will show necrotic debris on the surfaces of the conchal scrolls, but radiographs provide the definitive diagnosis in the form of turbinate 'coral' formation.

Facial and sinus trauma

Horses involved in all forms of athletic sport are vulnerable to facial trauma in falls, stick-and-ball injuries as well as kicks.

The guiding principles which relate to the management of cases of facial trauma are no different to those which apply to other wounds:

- control of haemorrhage and preservation of vital functions such as respiration in the acute phase.
- anatomical restoration where desirable for functional or cosmetic reasons.
- removal of devitalized tissue.
- control of secondary infection.

Intra-nasal foreign bodies

Intranasal foreign bodies are uncommon in horses. The sudden onset of acute nasal discomfort with intense sneezing and facial rubbing are the likely signs, particularly if there has been recent epistaxis. Some foreign bodies are visible as soon as the alar margins are raised but others are only identified at endoscopy.

Sub-cutaneous emphysema

The presence of air in the sub-cutaneous tissues suggests one of three events: (1) leakage of air from within the respiratory tract; (2) repeated entrapment of air in an external wound; (3) the presence of gas-forming organisms.

1. Trauma and surgery of the upper respiratory tract, including the paranasal sinuses, larynx, and trachea, may lead to air being forced between the skin and the underlying layers. Emphysema typically arises when the air cannot move freely to the nares during expiration and is therefore forced through a defect in the wall of the tract into the sub-cutaneous tissues. Oedema and spasm after laryngeal surgery, or nasal packing after fronto-nasal surgery are examples of iatrogenic causes of emphysema.
2. Wounds in the axillary region frequently cause progressive entrapment of air with each movement of the forelimb.
3. Clostridial (gas gangrene) infections typically stem from the deep inoculation of the infective agent, and unless identified promptly, they are rapidly fatal.

The objectives in treatment of subcutaneous emphysema are straightforward: either prevent further air entering the sub-cutaneous space by closure of the defect, or allow the air to escape more easily by an alternative route. Facilities for emergency tracheotomy or laryngotomy should always be on hand in establishments where airway surgery is performed. Neglected sub-cutaneous emphysema can lead to pneumothorax and fatal pulmonary collapse after air has entered by way of the mediastinum.

5.17 IDIOPATHIC HEADSHAKING IN HORSES

Clinical signs

The term 'headshaking' is used to describe the abnormal condition when a horse shakes its head in the absence of obvious external stimuli and with such frequency and violence that it becomes difficult or dangerous to ride or appears to be distressed.

The typical presenting features of a headshaker include:

- The headshaking consists of rapid vertical head flicking, and rarely are the movements horizontal.
- The behaviour occurs during ridden exercise once the horse has warmed up.
- The horse is inclined to sneeze/snort at the walk and trot.
- There is an obvious seasonal tendency for the condition, and in the northern hemisphere most cases begin between March and June. The condition generally is at its worst in late spring but can last until the early winter. During the first few seasons the winter resolution and spring relapses are obvious but many horses eventually become afflicted throughout the year.
- The horse was purchased during the winter months.
- There is almost invariably evidence of irritation at the nostrils. This may consist of sneezing, snorting, rubbing the nose or even striking at it with the forefeet.
- When being walked the horse will rub its nose along the ground or constantly nudge its handler in the back.
- The headshaking is particularly likely to occur on warm humid days when the horse is exercised under trees.
- There is sometimes engorgement of the superficial veins on the face together with excess lacrimation.

Horses of all breeds, and over a wide age range, may be afflicted by headshaking. It is unusual to encounter idiopathic headshaking (IHS) in horses racing on the flat or over jumps. Most appear to be involved in restrained exercise such as dressage, eventing, showing or general purpose riding.

Horses rarely exhibit IHS when at rest in the stable or at grass, but signs generally begin once the horse has warmed up at exercise. Owners often describe the headshaking behaviour as resembling a horse with a bee sitting on the end of the nose.

Aetiology

The aetiology of IHS is not known, but it is generally accepted that the disorder is mediated through the sensory branches of the trigeminal nerve, particularly the infra-orbital. The level at which it is triggered remains uncertain. The hypothesis that IHS is an expression of allergic rhinitis has not yet been supported by specific immunological tests.

Clinical investigation

A review of the presentation and history is of paramount importance. The major diagnostic challenge is to eliminate demonstrable disorders of the ears, eyes, cervical spine, auditory tube diverticula, upper respiratory tract, central nervous system (CNS), cranial nerves, teeth and oral cavity.

Thus, the clinical examination includes a general health assessment; a neurological examination; external inspection of the head; flexion tests of the neck in both horizontal and vertical planes; endoscopy of the upper respiratory tract, including a tracheal wash; and an ophthalmoscopic examination. A demonstration of the behaviour is staged within the limits of safety to the rider and the effect of a bilateral infra-orbital nerve block assessed. At a later stage intradermal monospecific antigen testing is applied. Under a general anaesthetic a detailed otoscopic inspection of both ear canals and smear examinations for mites should be performed (see 5.1). The oral cavity and teeth are checked carefully for potential sources of discomfort, particularly those which could interfere with the bit, and, when indicated, radiographs of the dental arcades are made. I n typical cases of IHS most of the investigations above are unhelpful.

A photic form of headshaking has been described in which a pattern of behaviour very similar to that described above has been attributed to exposure to bright sunlight. Obviously, photic headshaking could be expected in the summer months also, and care is required to differentiate the condition from IHS. The use of a visor with tinted lenses or testing the horse at dusk as well as in the middle of the day produces a dramatic improvement of photic headshaking.

Treatment

- The use of aids such as nose fringes can be helpful in the short term.
- A number of medical regimes have been tried to control IHS, and these have included systemic corticosteroids, inhaled corticosteroid (beclomethasone), inhaled sodium chromoglycate, systemic antihistamines and flunixin meglumine. None is at all effective, either because the treatment has no effect whatsoever or because of impracticalities of administration.

The results achieved by bilateral infra-orbital neurectomy, either by section of the external nerve trunks as they emerge from their foramen, or by chemical destruction within the infra-orbital canal or in the floor of the orbit, have been inconsistent.

Cyproheptadine may be useful for the management of photic headshaking.

5.18 DIAGNOSTIC APPROACH TO CONDITIONS CAUSING AIRWAY OBSTRUCTIONS IN HORSES

Functional concepts of conducting airways

Olfaction is relatively unimportant to the horse, and the conchal scrolls of the nasal chambers are anatomically simple, and the nasal meati are streamlined for a species which is an obligatory nasal breather.

- The movement of air through the respiratory tract is achieved by the creation of pressure gradients during inspiration and expiration.
- During the inspiratory phase the pressure within the lumen of the airways is lower than that within the tissues of the walls of the tract as well as in the external environment.
- During expiration the gradients are reversed.
- The greater the work of breathing, the greater are the collapsing forces applied to the walls of the upper respiratory tract, so that during inspiration at exercise they are very considerable, as much as 40 cm H_2O.
- During quiet breathing, the upper respiratory tract, principally the nostrils and larynx, provides 70–85% of total resistance, but when required this can be reduced to less than 50% by strategies such as the active dilation of the nares and larynx and by straightening the airway.
- Airflow through the conducting airways increases from 80 L/min at rest to 2500 L/min at the gallop, and video-endoscopy of horses exercising on a high-speed treadmill confirms that the airways dilate in response to increased demand.
- An essential component of the overall airway dilation is the maximal abduction of the rima glottidis achieved by the action of the intrinsic laryngeal musculature. Exercising endoscopy confirms that the full abduction is sustained through all phases of the respiratory cycle and continues during the immediate post-exercise recovery period.
- Failure of this function, for example in recurrent laryngeal neuropathy or arytenoid chondropathy, leads to dynamic collapse of the flaccid structures into the airway during inspiration with resultant restriction of airflow with its associated noise.
- Dynamic dysfunction at the level of the pharynx and larynx accounts for the majority of cases of obstructive dyspnoea in athletic horses.
- The structural rigidity provided by the conchal cartilages and the tracheal rings provides stability through much of the upper respiratory tract but in three key areas of the airway, the nostrils, pharynx and larynx, resistance to collapse is provided by active muscular dilation. These are coincidentally the sites at which much of the resting airway resistance occurs, and it is suggested that relatively minor lesions or dysfunctions at these sites of high resistance may significantly increase total impedance to airflow.

Respiration and locomotion

At the walk, trot, and when pacing, there is no fixed relationship between locomotion and the respiratory cycle. However, at the canter and gallop a 1 : 1 relationship exists, i.e. one respiratory cycle is completed in the time taken for one stride. Some unfit horses and others with airway obstructions may exhibit 2 : 1 cycles with two strides for each complete respiratory cycle. At the canter and gallop inspiration occurs during the extensor phase of the forelimbs. Conversely, expiration occurs during the weight bearing phase of the forelimbs which in ridden exercise coincides with the rider moving forwards in the saddle.

Horses may require to swallow during exercise, and respiration cannot continue through deglutition; it is suspended after the inspiratory phase and resumes with expiration on completion of swallowing. For horses at the canter and gallop deglutition must be completed exactly in the time taken for a whole number of strides, usually one.

Intra-narial larynx and role of the soft palate

The horse is designed to be an obligatory nose-breathing animal and never to breathe through its mouth. It is equipped with an intra-narial larynx whereby the cartilages of the larynx are locked into the caudal wall of the nasopharynx by the palatopharyngeal arch which acts as

an air-tight seal. The intrinsic musculature of the soft palate provides a second, possibly more important role in the stabilization of the pharyngeal airway whereby tension in the soft palate eliminates air from the oro-pharynx. Thus, the ventral surface of the soft palate and the dorsal surface of the tongue are held in contact with one another, and the only stage when they separate is during swallowing. The intranarial larynx is also required to be able to disengage momentarily for deglutition.

Clinical signs of obstructive dyspnoea

The signs of airway obstruction include increased respiratory effort, increased respiratory noise and compromised athletic performance. The increased work of breathing exaggerates the collapsing forces so that some horses which appear to be normal at rest show signs only during exercise. Dynamic collapse of the soft structures abutting onto the walls of the airway may exacerbate the obstruction, and audible respiratory sounds are produced. Occasionally the airways are so severely obstructed that breathing at rest is embarrassed, for example in the face of an intranasal mass or arytenoid chondropathy.

Respiratory noises at exercise

When horses are exercised, the process of inspiration should be almost silent to the unaided human ear, whereas exhalation produces non-specific sound. Vibrant sounds generated by the alar folds frequently make expiration a noisy process in some normal horses – 'high blowing'.

Respiratory obstructions cause turbulence in the airways, and these are translated as the sounds which are heard. They may arise through structural distortion of the walls of the airways or through abnormal function. When there is structural change or when the malfunction is consistent the inspiratory sounds which are heard will be reliably present at a given speed, provided that ambient conditions of temperature and humidity remain unchanged. However, in some circumstances the dysfunction is momentary, typically at the time of greatest effort, and therefore the sound which is heard has a relatively abrupt onset and often an equally abrupt conclusion.

Analysis of the frequency and intensity of the noises produced by horses at exercise can be helpful in the diagnosis of some specific disorders such as recurrent laryngeal neuropathy, but more often than not there is lack of specificity as other airway obstructions do not provide a sound 'fingerprint'.

History

Horses are presented for respiratory assessments at pre-purchase examinations and because of nasal discharge, untoward respiratory noises, acute and chronic coughing, prolonged post-exercise tachypnoea, fatigue at exercise and disappointing performances. In the context of poor performance, it is important to discover whether the performance is unproven and may result from an inherent lack of ability, or whether the patient has been an effective athlete but is no longer successful.

Information about the duration of clinical signs, history of previous treatments, including surgery, should be obtained and the rider's description of the noises produced can be invaluable. Note should be taken of management factors such as the feeding and stabling routines and of the riding aids which have been used, i.e. bits, nosebands, tongue straps, martingales.

Before commencing an examination of the upper respiratory tract the bodily condition of the patient should be noted as overweight, unfit horses are inclined to produce 'stuffy' respiratory noises resulting from pharyngeal flaccidity. The temperature should be taken and any nasal discharge noted; the lymph glands should also be palpated looking for evidence of current URT infections.

Palpation tests

During the examination at rest palpation of the larynx should check for:

- the cartilage structure of the larynx (see 5.25).
- the presence of atrophy of the intrinsic musculature, typically the crico-arytenoideus dorsalis muscle, and resultant prominence of the muscular process of the arytenoid cartilage.
- evidence of previous surgery, i.e. a ventral laryngotomy cicatrix or scar from laryngoplasty surgery.
- responses to the 'slap' test; these are better assessed by palpation than by endoscopy.
- the arytenoid depression test whereby the right muscular process is depressed to close the right side of the larynx. In cases of severe recurrent laryngeal neuropathy the left side of the larynx will already be closed. Thus, closure of the right side will markedly restrict the airway and provoke stridorous sounds.

Palpation of the trachea should seek evidence of disruption of the normal ring architecture by previous trauma or surgery. Congenitally flattened rings may also be detected.

Endoscopy at rest

An endoscopic inspection of the conducting airways during quiet breathing forms the basis for most diagnoses of airway obstructions in practice. A full endoscopic examination should include both nasal chambers, the nasopharynx, epiglottis, soft palate, larynx and trachea. Apart from the overall conformation of the lumen of the airway, the relationships between structures such as the soft palate and epiglottis may be noted and the resting movements of the larynx observed. Note is also made of the presence and origin of any discharge including blood.

During the examination the patient should be stimulated to swallow on multiple occasions to give the best opportunity to identify those anomalies such as epiglottal entrapment and small sub-epiglottic cysts which may not be consistently visible.

Radiography of the throat

Radiographs can be used to provide an additional dimension to respiratory investigations particularly to reveal structures obscured from endoscopic view, for example the tissues ventral to a soft palate which is permanently displaced dorsal to the epiglottis.

Conduct of exercise tests

Although an exercise test is an important part of the overall examination, and no pre-purchase assessment is complete without one, the consent of the owner of the horse, the suitability of the location, the fitness of the horse and the competence of the rider/handler should all be established before the test begins.

All ridden and lunged exercise tests to detect respiratory sounds should include a canter in both directions in a confined area. A ridden test is preferred because in a collected canter the poll is flexed and the resultant curvature of the airway exaggerates any untoward noises produced. Any adventitious noises can be timed with respect to the phase of the respiratory cycle (see 'Respiration and locomotion', above), i.e. inspiratory versus expiratory.

Figure 5.7 Overground endoscopy of a horse during exercise. The control unit with this instrument is contained within a pouch on the visor.

Endoscopy after exercise

Although it is standard procedure to include an examination as soon as possible after completion of the exercise test, this is probably the least informative time to perform endoscopy. No dynamic collapse is likely to be seen, and some of the subtler anomalies apparent during quiet breathing will have been abolished especially by the full sustained abduction of the arytenoid cartilages.

Treadmill and overground endoscopy of horses during exercise

There is an obvious flaw in the concept of URT endoscopy of a horse standing still to speculate on dynamic events occurring only at exercise. Endoscopy at rest in the stable is used by the majority of clinicians as the first, and often only, stage in URT diagnosis, and yet the technique has been shown to provide incomplete or incorrect diagnoses in at least 40% of cases. Clearly, there are major structural abnormalities which can be identified in the tract at rest and when an accurate diagnosis can be given. In addition, the function of the respiratory organs of some horses may be so abnormal that a diagnosis can be made even during quiet breathing.

The advent of improved endoscopic equipment and treadmills upon which horses can exercise at speeds up to 14 m/second has provided the means by which to study the dynamic changes which occur in the conducting airways at exercise. At the same time physiological tests of respiratory function run in parallel with endoscopy have made it possible to validate the impact of the abnormalities which have been observed on horses' athletic capacity.

The advantages of high speed treadmill endoscopy (HSTE) include improved specificity of diagnosis and an increased repertoire of diagnoses. It also gives the opportunity for simultaneous physiological tests, not only to assess whether the anomalies observed endoscopically are compromising respiratory function but as the means to test the efficacy of surgical methods of correction.

Overground (OG) endoscopes, whereby the endoscope is inserted into the URT of an exercising horse and its light source and control unit are carried either by the rider, in a pouch on a visor (Figure 5.7), or in the saddle cloth, are now becoming more widely available. The advantage of the OG endoscope over HSTE is that the horse is not required to visit a specialist centre and little familiarization training is required. However, research has suggested that OG exercise of the horse in training is less strenuous than on a treadmill so that those anomalies such as dorsal displacement of the soft palate which arise with maximal effort are less frequently diagnosed. Nevertheless the diagnostic 'gap' can be narrowed when experienced riders and a reproducible and exacting exercise test are employed.

Ultrasonography of the upper respiratory tract

Ultrasonography is a useful modality in the diagnosis of laryngeal and pharyngeal function and structure and can contribute in a non-invasive manner to the assessment of poor performance. It provides additional information on the extra-luminal structures of the upper airways and can be used, for example, to assess the positions of the laryngeal cartilages and the hyoid apparatus in relation to one another, the movements of the vocal cords and the relative mass and/or density of the intrinsic laryngeal musculature so that left and right sides can be compared.

5.19 COMMON UPPER RESPIRATORY TRACT OBSTRUCTIVE DISORDERS OF HORSES

Obstructions of the conducting airways are diagnosed in three circumstances:

1. in the investigation of horses which produce abnormal respiratory noises during exercise.
2. in the investigation of horses showing poor, reduced or sub-optimal performance.
3. during routine pre-purchase examinations.

In order of frequency in Thoroughbred horses, the major causes of respiratory obstructions during exercise – dynamic airway collapse – are:

1. malfunctions of the soft palate – palatal billowing and instability (PI) and dorsal displacement of the free border (DDSP).
2. recurrent laryngeal neuropathy (RLN) leading to vocal cord collapse (VCC) and/or arytenoid cartilage collapse (ACC).
3. axial deviation of the ary-epiglottal folds (ADAF).
4. ventro-axial luxation of the corniculate process of the arytenoid cartilage (VALCPA).
5. epiglottal entrapment (EE).
6. fourth branchial arch defects (4-BAD).
7. arytenoid chondropathy (AC).
8. Subepiglottic cysts.

The prevalence of each of these entities in equine athletes is not known, and horses are commonly afflicted with complex forms of dynamic airway collapse wherein the causes of obstruction during exercise are multiple.

5.20 RECURRENT LARYNGEAL NEUROPATHY (RLN)

Aetiopathogenesis

RLN causes a permanent dysfunction of the intrinsic muscles of the larynx which receive their motor innervation through the recurrent laryngeal branch of the vagus nerve. The result is partial obstruction of the airway evident during exercise and compromised athletic performance through hypoxia. The condition almost invariably involves the left side of the larynx, and very rarely right-sided or bilateral cases are encountered. The disease manifests itself as a failure to achieve or to maintain full symmetrical vocal cord and/or arytenoid abduction under conditions of greatest respiratory demand. The resistance to normal airflow causes turbulence in the airstream which is the source of the characteristic abnormal inspiratory sounds – 'whistling' or 'roaring'.

The term recurrent laryngeal neuropathy is preferred to that of idiopathic laryngeal hemiplegia because 'hemiplegia' should be reserved to describe those disorders where one side of a structure has no active motor function whatsoever. This is often not the case with horses afflicted with RLN.

RLN consists of a distal axonopathy whereby the larger myelinated nerve fibres degenerate from the motor end-plate proximally towards the cell body of the neuron. The effect is one of atrophy of those muscles predominantly supplied by the large myelinated fibres, and for this reason the major adductor, the crico-arytenoideus lateralis (CAL) is afflicted before the major dilator of the rima glottidis, the crico-arytenoideus dorsalis (CAD). However, defects of adduction are rarely noted by owners although they form the basis of some clinical tests such as the 'slap' reflex. It has been suggested that the structural changes in the nerves arise through an underlying defect of axonal transport so that, to some extent, the condition may be related to the absolute length of the recurrent nerve fibres. The recurrent laryngeal nerves include by far the longest lower motor neurons in the horse, and the left nerve is significantly longer than the right. Clinical signs of RLN, exercise intolerance and stridor at exercise, become apparent with involvement of the principle abductor, the CAD muscle, and the degree of neurogenic myopathy correlates with the severity of the nerve lesion.

Less common causes of RLN include:

- trauma to the recurrent nerve by perivascular injection of irritant medicaments.
- other direct trauma to the recurrent nerve.
- damage to the vagal trunk by guttural pouch mycosis or strangles infection.

Toxic and metabolic insults to the recurrent nerves are likely to be bilateral:

- toxicity by heavy metals, such as lead, and organophosphate poisoning.
- nutritional deficiencies such as thiamine.
- liver failure.

Distal axonopathies often show an inherited basis in other mammalian species, and there is a wealth of anecdotal evidence to support this contention for RLN in the horse. Genetic research is on-going to establish the mode of inheritance, if it exists, in the hope that it will lead to an enlightened approach to management or prevention of the disorder.

During exercise the arytenoid cartilages are normally held in full symmetrical abduction which is sustained throughout all stages of the respiratory cycle, and this abduction will be present for a period after completion of the exercise dependent on the duration and degree of the exertion and the fitness of the horse. Failure of the neuromuscular unit results in partial obstruction of the airway. As the cross-sectional area of the rima glottidis is reduced, the pressure differentials within the larynx increase to maintain the airflow necessary to sustain exercise. The collapsing forces rise, and the paralysed vocal cord is drawn even further across the airway (Venturi effect). When the demands for inspiratory flow reach their peak, afflicted horses show flow limitation, increased inspiratory resistance and depression of arterial oxygen tension.

Prevalence

Horses of any age from birth onwards may be afflicted with RLN, and it has been shown that the disorder can be progressive, i.e. normal or slightly afflicted horses may deteriorate. Horses over 16 hands tall are most susceptible, and RLN becomes increasingly rare in horses below 15.2 hands. The prevalence of RLN in the Thoroughbred is not known, and there are widely varying estimates (0.96–95%) in the literature. The frequency of true hemiplegia, i.e. where there are no active abductory or adductory movements whatever by the left arytenoid cartilage and vocal fold, is in the order of 2%.

The clinical signs of RLN usually appear before the horse is 6 years of age.

Clinical signs

- Horses afflicted with RLN usually produce consistent inspiratory sounds which can be heard throughout the period of exertion at the canter and gallop.
- The sounds themselves may range from a low-grade musical 'whistle' similar to the noise produced by blowing over the top of an empty bottle, to a harsh 'roaring' noise like sawing wood.

- Disappearance of the sounds can be expected within a short period of pulling up; horses with RLN recover a resting respiratory rate in a normal period.
- Occasionally horses with RLN produce adventitious respiratory noises only under extreme exertion, and, unless examined by OG or HST endoscopy, it is not possible to determine whether the noise stems from abrupt dynamic collapse of the paralysed arytenoid cartilage or from secondary DDSP.
- The athletic potential of horses which exercise aerobically is likely to be compromised by RLN. However, the impact on the performance of horses which are involved in anaerobic sports, such as sprint racing, will be less marked.

Diagnosis

1. Palpation

Palpation of the larynx should seek evidence of atrophy of the intrinsic laryngeal musculature especially on the left side.

The arytenoid depression test, whereby the right side of the larynx is forced to adduct by pressure on the right arytenoid muscular process, is more likely to provide a convincing increase in stridor at the conclusion of exercise than at rest.

Palpation should also seek evidence of a cicatrix from previous ventral laryngofissure surgery. The area ventral to the left linguo-facial vein should be checked for a prosthetic laryngoplasty ('tie-back') scar.

During palpation of the larynx note should be taken of the spacing between the cricoid and thyroid cartilages. Deformities of the thyroid laminae are frequently present as part of the fourth branchial arch defect syndrome (see 5.25), and particular attention should be paid in cases of apparent right-sided RLN.

An assessment of the strength of the 'slap' response is more accurately judged by palpation than by endoscopy. The test should not be applied during the inspiratory phase of respiration.

2. The 'Grunt-to-the-stick' test

This test depends upon startling the horse by threatening it. Laryngeal fixation in an incompletely closed position, together with a rapid rise in pressure within the airway, produces a low-pitched grunt. This is a test of the competence of laryngeal adduction, but the results are inconsistent.

3. Resting endoscopy

The asymmetry of the rima glottidis in cases of true left laryngeal hemiplegia is usually obvious.

During equine laryngoscopy, the perspective distortion which arises from the eccentric position of the endoscope in the nasopharynx must be taken into account. Thus, when the endoscope is introduced through the right nostril, false negative diagnoses are possible, but from the left side the left arytenoid cartilage may give the false impression of inadequate abduction. Whenever doubt exists, the endoscopy should be performed through each nostril in turn.

A grading system of laryngeal function with reproducible values is necessary if the subjectivity of endoscopy of the larynx is to be eliminated, particularly when left laryngeal dysfunction is incomplete. A suggested laryngeal function score (LFS) grading system is provided below:

Grade 1: All movements by the left and right arytenoid cartilages (both adductory and abductory) are synchronized and symmetrical.

Grade 2: All major movements of the arytenoid cartilages are symmetrical with a full range of adduction and abduction. Transient asynchrony, flutter, or delayed abduction, especially by the left arytenoid cartilage, may be seen.

Grade 3: The rima glottidis is asymmetric during quiet breathing, but the left arytenoid cartilage and vocal fold are capable of full abduction, typically in response to the nostril occlusion manoeuvre or after swallowing.

Grade 4: There is consistent asymmetry of the rima glottidis at rest: the left arytenoid cartilage is not capable of full abduction, but some residual movements are present.

Grade 5: True hemiplegia: active movement is absent on the left side with the arytenoid cartilage resting on or near the midline. In addition, the 'slap' test does not provoke any adductory response (Figure 5.8).

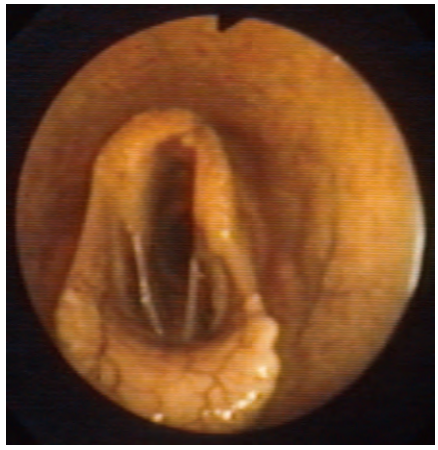


Figure 5.8 Recurrent laryngeal neuropathy (Grade 5 out of 5 LFS) where there is obvious asymmetry of the rima glottidis and no active motility by the left arytenoid cartilage or vocal fold – true hemiplegia.

This five-point scheme of LFS grading is that which is most commonly used in the United Kingdom, Ireland, and Australasia, but other systems with up to 7 grades are in use in the USA., e.g. the Havermeyer system.

In the context of pre-purchase or auction sales examinations, where dynamic investigations are generally impracticable, Grades 1 and 2 are deemed to be within the limits of normality. Grades 4 and 5 are considered abnormal and Grade 3 comprises equivocal dysfunction.

The correlation between LFS grading at rest and dynamic endoscopic findings is inexact. For example, while all horses with Grade 5 LFS at rest show dynamic collapse by the arytenoid cartilage (ACC) and vocal cord (VCC) at exercise, approximately 20% of horses showing Grade 4 LFS are able to achieve and maintain full abduction throughout exercise. Furthermore only 39% of horses with Grade 3 LFS show ACC and/or VCC at exercise. Conversely 7% of horses with Grades 1 and 2 LFS will sustain progressive ACC and/or VCC at exercise; all of these 7% produce an inspiratory 'whistle' or 'roar' when exerted.

It is helpful that dynamic endoscopic studies have established that, in the context of RLN, a horse is normal unless an audible noise is produced.

4. Dynamic endoscopy

HSTE or OG endoscopy is necessary to provide a complete assessment of laryngeal function because of the inconsistencies of interpretation of endoscopy at rest – see above (Figure 5.9).

5. Exercise test

See 'Endoscopy after exercise' in 5.18.

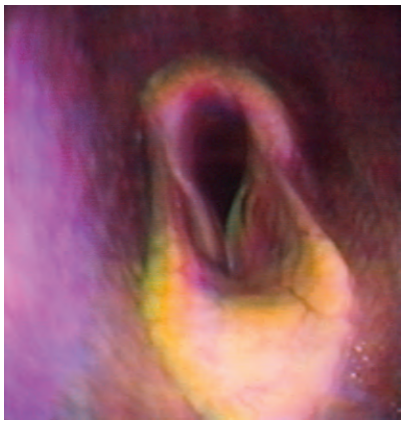


Figure 5.9 Dynamic collapse of both vocal cords and the left arytenoid cartilage in a horse which appeared to be normal at rest, confirmed by overground endoscopy during fast exercise.

6. Other diagnostic tests

These are not yet widely used, but are likely to be of future interest; they include ultrasonography (see 'Ultrasonography of the upper respiratory tract' in 5.18), the radio-stethoscope with sound frequency analysis, electro-myographic recording of laryngeal muscle activity and measurement of conduction time in the 'slap' reflex.

Treatment

A number of procedures have been proposed to relieve the obstructive effects of RLN: the most notable of these are:

1. Ventriculo-cordectomy (Hobday or Williams procedure) (performed by conventional or laser surgery) depends upon the removal of the mucous membrane lining from the laryngeal ventricle(s) and excision of the vocal fold(s). The benefits of the Hobday procedure are at best slight, and the technique should be reserved for horses with less marked RLN and those horses which, on dynamic endoscopy, are shown to be obstructed by the collapse of the vocal fold rather than by collapse of the arytenoid cartilage.
2. Prosthetic laryngoplasty (abductor prosthesis operation – 'tie-back') is considered to be the treatment of choice for RLN in most countries.

   The procedure and its variants aim to implant a suture between the caudal border of the cricoid cartilage and the muscular process of the arytenoid to mimic the action of the CAD muscle as if it were in a semi-contracted state.

   However, the procedure should be regarded as a gross physiological disturbance because when the rima glottidis is fixed in an abducted position, the ability of the larynx to protect the lower airways during deglutition is compromised, and a degree of dysphagia is inevitable. Nevertheless, most horses show relief of laryngeal obstruction and are only sub-clinically dysphagic.

   Physiological studies have confirmed that prosthetic laryngoplasty is effective in the restoration of normal respiratory function and in the prevention of dynamic collapse of the paralysed arytenoid in cases of RLN.
3. Nerve/muscle pedicle grafting aims to transplant small cubes of muscle taken from the omo-hyoideus together with their motor supply through the first and second cervical nerves into the atrophied CAD muscle to restore abductory function to the larynx. Following surgery the grafts grow in response to mechanical stimulation so that at least a year must be allowed to achieve optimum results. Abduction of the arytenoid cartilage only occurs during exertion because the omo-hyoid is an accessory muscle of respiration, and the technique has the advantage over prosthetic laryngoplasty that no complications can arise from aspiration through a permanently abducted rima glottidis.
4. Total, partial and sub-total arytenoidectomy aim to remove the intra-laryngeal structures which are causing obstruction. The usual indications for arytenoidectomy are the removal of infected cartilage in cases of chronic chondropathy (see 5.24) and the removal of the left arytenoid cartilage when other techniques to treat RLN have failed.
5. Tracheotomy intubation (see 'Tracheotomy intubation' in section 5.28). The purpose of these tubes is to provide an alternative airway and to by-pass the site of airway obstruction. Tracheotomy tubes are not permitted under the rules of some racing and equestrian authorities. When permitted they may be used where other surgical techniques for RLN have failed. Their major virtue is that intubation is performed under local analgesia, and disruption of the training programme is minimal.

   Tracheotomy tubing provides a short-term expedient to racehorses which would otherwise be side-lined by alternative surgery.

   When the tracheotomy tube is eventually removed the defect heals quickly by second intention, and the option to perform a more enduring surgical correction will not have been compromised.
6. Permanent tracheostomy, the creation of a fistula between the tracheal lumen and the skin surface of the ventral neck, has been used for horses and ponies, but the results are generally not aesthetically acceptable. There is a regular requirement for nursing to remove exudation from the skin adjacent to the stoma and to maintain local hygiene.

Prognosis

Prosthetic laryngoplasty is far from ideal as a treatment for RLN but it remains the best practicable option currently available. Refinements are required to provide consistent and enduring abduction without dysphagia. Although it is recognized that this surgery can produce complications in the forms of coughing, nasal reflux of ingesta or recurrence of dyspnoea, the risks are justifiable in horses which cannot otherwise be effective athletes.

Nerve/muscle pedicle grafting is a potential alternative, but its application is most likely to be limited to horses where RLN is confirmed at an early stage and when the prolonged convalescent period is less restrictive.

Functional electrical stimulation of the recurrent nerve using an implanted stimulator with an external control unit is a novel approach suggested as a future potential remedy for RLN.

5.21 DORSAL DISPLACEMENT OF THE SOFT PALATE (DDSP)

Aetiopathogenesis

The major function of the arcus palato-pharyngeus – the soft palate – of the horse is to stabilize the pharyngeal section of the airway. Not only does it provide an airtight seal that locks the larynx into the caudal wall of the nasopharynx, but the tension provided by its intrinsic musculature helps to eliminate air from the oropharynx. Thus, the dorsal surface of the tongue and the ventral surface of the palate remain in intimate contact at all times other than during the passage of food and fluid boluses through the oropharynx. This mechanism renders the horse at all times an obligatory nose-breather where the

intra-narial siting of the larynx provides excellent streamlining of the airway.

Rostrally the palatal arch passes ventral to the epiglottis; laterally, lateral to the aryepiglottic folds; and caudally, caudal to the corniculate processes of the arytenoid cartilages. Thus, the free border of the palate is not visible by endoscopy when the larynx occupies its normal intra-narial position.

DDSP is the end-stage of a sequence of events which occurs during vigorous exercise and which commences with the entry of air into the oro-pharynx. This causes a lack of stability in the soft palate which is seen on dynamic endoscopy as waves of billowing passing from the junction of the hard and soft palates caudally. The epiglottis is seen to flatten against the dorsal surface of the palate partly by the action of the hyo-epiglotticus muscle drawing the cartilage downwards and partly because air is pushing the palate upwards. This stage is termed palatal instability (PI), and it may be reversed by deglutition which expresses the air from the oro-pharynx and re-establishes the stable relationship between the tongue and the palate. Most often the pattern is repeated within a few strides, but eventually the free border slips from beneath the epiglottis, and the loose palatal tissues obstruct breathing particularly during expiration. There is a marked overall reduction of ventilation. The obstructive effect persists until the horse swallows again to remove the air and to realign the larynx into its intra-narial position.

While the events which occur leading to DDSP are well recognized the causes are less clear. There are similarities between DDSP and human sleep apnoea – snoring – and it is likely that DDSP is also a multifactorial disorder. The lack of a specific cause for both conditions also explains the lack of a specific remedy and the disappointing levels of success reported for the therapies proposed to date.

Research has shown that PI and DDSP can be induced experimentally by inhibiting the function of specific intrinsic and extrinsic muscles of the region. Thus, current suggestions are that PI and DDSP may result from:

1. Weakness of the intrinsic musculature of the soft palate.
2. Abnormal relationships between the larynx and the hyoid apparatus.
3. Malfunction of the protrudor (genio-glossus) muscle of the tongue.

Clinical signs

The abrupt respiratory obstruction which accompanies DDSP usually causes a loud vibrant, predominantly expiratory noise but also precipitates a serious interference with the progress of the horse. Treadmill observations have established that a minority of horses experiencing DDSP do not produce an audible noise at the time of displacement.

In many cases the horse is unable to maintain its speed and may completely lose its stride rhythm as it makes gulping attempts to restore the larynx into the intra-narial position and to eliminate air from the oropharynx. Occasionally a horse will continue running noisily but with the severe handicap produced by partial asphyxiation. As soon as the normal anatomical configuration is restored, the horse is able to resume galloping and will not appear distressed thereafter.

An alternative expression of soft palate malfunction explains why some horses fade abruptly in races without producing abnormal breathing noises. Such horses have been seen to reduce speed during HSTE or OG endoscopy when the stage of PI is reached, but before DDSP has occurred.

DDSP generally occurs when a horse reaches the point of maximum exertion, typically in the later stages of a race or during a competitive gallop in training. Racehorses, including trotters and pacers, are mostly involved, but the dysfunction can occur in horses with other occupations, such as eventing.

Many young horses in the early stages of preparation for racing, or older horses returning to vigorous exercise after a period out of training, produce vibrant respiratory noises corresponding to PI, possibly leading to DDSP. Increased conditioning as the overall level of fitness rises generally leads to resolution.

Diagnosis

A history of relatively sudden onset of vibrant respiratory noises as a horse comes under pressure in vigorous exercise or in races is typical of DDSP. Information regarding the overall state of fitness of the patient, such as any recent exposure to upper respiratory tract infections, should be sought together with details of recent racing or competition performances.

Although a ridden exercise test should be included for cases of suspected DDSP it is unusual for a choking / gurgling episode to occur without the exertion of a race or competitive gallop.

Endoscopy at rest is generally accepted as being of limited value to diagnose DDSP although it can be helpful to identify alternative explanations for the airway obstruction. Intermittent DDSP during resting endoscopy is frequently observed in normal horses and should not be regarded as an indication of potential dynamic dysfunction during exercise.

However, persistent DDSP should be regarded as significant and is an indication for further investigations such as radiography or endoscopy *per os*.

Dynamic HSTE or OG endoscopy provide the definitive means to diagnose soft palate malfunction. DDSP is invariably preceded by a period of PI, and in some horses the disorder does not progress beyond the stage of PI within the limits imposed by an exercise gallop.

Treatment

Conservative measures

- Every attempt should be made to eliminate predisposing factors including unfitness, or, alternatively, to provide additional time or medication for recovery from respiratory tract infections.
- Aids to prevent mouth-breathing such as a drop or cross noseband should be tried, and a change of bit to a simple rubber snaffle.
- The position of the bit in the mouth may be modified by an Australian noseband.
- Tongue straps or tongue-ties are used to discourage caudal retraction of the tongue and may assist in the function of the genio-glossus muscle.
- The 'Cornell Collar' is a device mounted on a strap which aims to push the body of the thyroid rostrally and dorsally in relation to the basihyoid bone, acting in a non-surgical 'tie forward' capacity. Favourable results have been reported, but this equipment is not permitted by some racing authorities.

Surgical treatments

The results of surgical procedures performed to inhibit DDSP are varied and, of course, will depend on how rigorous the methods of diagnosis and selection have been at the outset.

1. Resection of the free border – staphylectomy.
   - This technique appears to be quite illogical inasmuch as the effect of the surgery is to increase the size of the intra-pharyngeal ostium so that the intra-narial configuration of the larynx would be expected to be less stable. However, there is agreement that a proportion of horses are improved by staphylectomy – 59% in one survey – and it has been

suggested that the improvement arises through a reduction in the bulk of the tissues available to obstruct the rima glottidis.
- Staphylectomy is performed under general anaesthesia and through a conventional laryngotomy incision at the crico-thyroid membrane.
- The major detractions of staphylectomy are the need for general anaesthesia and the possibility that the excessive removal of palatal tissue will precipitate a permanent dysphagia.

2. Section of the strap muscles – sterno-thyro-hyoideus myectomy.
   - There is no evidence that laryngeal retraction by the action of the sterno-thyroid and sterno-hyoid muscles contributes to DDSP, and yet resection of these muscles remains a popular remedy in some countries. The procedure can be performed under local analgesia and sedation but is more safely and more accurately carried out under general anaesthesia. The overall success rate of 60% which has been recorded after myectomy is not significantly different from that achieved by staphylectomy.
3. Interferences to the dorsal surface of the soft palate.
   - Scarification of the soft palate either by the trans-endoscopic application of laser cautery or by injection of sclerosis agents has been advocated as the means to 'tighten' the soft palate. Both have the advantage that they are performed with the patient standing and the recovery time to return to training is short. However, there are no published controlled results of either technique, and it also appears to be illogical to damage the intrinsic musculature which lies immediately ventral to the nasal mucosa.
4. Interferences to the ventral surface of the palatal arch.
   - These insult the oral mucosa and the underlying loose glandular tissue but do not extend deeper than the aponeurosis of the palate. The techniques include physical and chemical cautery, as well as partial thickness 'nip-and-tuck' (Ahern) resections. All require general anaesthesia to achieve their objective to increase tension in the part of the palate which is in contact with the tongue.
5. Laryngeal advancement – 'tie forward'.
   - 'Tie forward' surgery aims to move the larynx rostrally and dorsally in relation to the basi-hyoid bone and comprises the placement of sutures to mimic the action of the thyro-hyoid muscle. The result is that the body of the thyroid moves forwards by at least 4 cm. Published results suggest that this is the most successful technique currently in use with reported resolution in over 80% of cases.

Prognosis

A cautious prognosis must be given for all horses in which DDSP is diagnosed. The results of the surgical treatments outlined above are, at best, unpredictable.

5.22 EPIGLOTTAL ENTRAPMENT (EE)

Aetiopathogenesis

- In this condition the cartilage of the epiglottis becomes enveloped by a fold of glosso-epiglottic mucosa arising between the epiglottis itself and the base of the tongue and extending laterally as the aryepiglottic folds.
- The aetiology of EE is usually not known but involves a degree of stretching of the mucosa and it is not clear why some horses develop this condition, and why others are susceptible to axial deviation of the ary-epiglottal folds (see 5.26).
- The aetiology of EE is no mystery when the epiglottis is congenitally hypoplastic or where it is associated with a sub-epiglottic cyst. These possibilities should be checked by endoscopy and/or radiography before surgical correction is attempted.

Clinical signs

The signs associated with EE are highly variable and include:

- exercise intolerance with vibrant inspiratory and/or expiratory noises at exercise.
- intermittent gurgling from secondary DDSP.
- coughing after eating.

Some cases of EE are asymptomatic and are chance findings during routine endoscopy for other purposes.

Reports of the prevalence of EE in endoscopic surveys of athletic horses at rest suggest an incidence in the range of 0.75% to 3.3%.

Diagnosis

A definitive diagnosis of EE is achieved by endoscopy which shows that the epiglottis has lost its wrinkled cartilage border and that the superficial blood vessels are obscured by the entrapping mucosa (Figure 5.10).

Occasionally the mucosa overlying the epiglottis is ulcerated.

When persistent DDSP is present, the epiglottis cannot be seen at all, in which case a lateral radiograph of the pharynx is indicated to determine whether or not entrapment is present. On this projection the epiglottis should measure at least 7.0 cm from tip to hyoid articulation; an epiglottis with a length less than 5.5 cm is indicative of hypoplasia, and surgery is contra-indicated.

EE may be intermittent, and it is important, as a routine part of the endoscopic procedure, to stimulate a series of deglutition sequences in an attempt to provoke the condition.

It is very rare indeed that EE is diagnosed on the basis of HSTE or OG endoscopy during exercise when it has not already been seen during conventional endoscopy.

Treatment

The treatment options for epiglottal entrapment are:

(a) resection via ventral laryngofissure.
(b) axial division *per os*.
(c) axial division *per nasum*.
(d) transendoscopic diode laser resection.

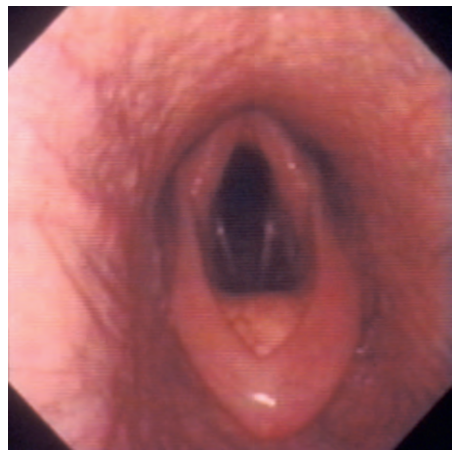


Figure 5.10 Epiglottal entrapment in a 7-year-old gelding. Note that the apex of the epiglottis is covered by a doubled layer of mucosa.

Methods (a) and (b) require general anaesthesia, but both (c) and (d) can be performed on the standing horse with the obvious advantage of minimal disruption of training and racing schedules.

Methods (b) and (c) utilize a hooked bistoury to make a longitudinal cut in the loose mucous membrane overlying the epiglottal cartilage. The procedure of axial section of the EE is performed under endoscopic surveillance – the endoscope is passed through one nostril while the bistoury is introduced through the other. The aryepiglottic mucosa is quite tough and care is required to prevent a disastrous palatal injury when the hook suddenly cuts free from the entrapment. Method (b) is safer and represents the technique of choice albeit with the risk of anaesthesia. A hybridized technique whereby axial section of the mucosa is performed via the mouth with the horse standing has been described, but desensitization of the oropharynx and the abolition of swallowing responses is essential.

Prognosis

All of the methods of treatment mentioned above yield a high rate of uncomplicated resolution of EE. The possibility of recurrence of entrapment is greatest with the axial section techniques, but even this is uncommon. Iatrogenic trauma to the epiglottal cartilage can provoke granulomas and other distortions which may compromise the relationship between the epiglottis and the soft palate. Chronic coughing associated with low-grade dysphagia is a rare but recognized complication of resection of the glosso-epiglottal mucosa.

5.23 SUB-EPIGLOTTAL CYSTS

Aetiopathogenesis

- Developmental cysts are occasionally recognized in the pharynx of horses and represent causes of dyspnoea and/or dysphagia.
- The most frequently encountered of these lesions is the sub-epiglottic cyst which is thought to be derived from the embryological remnants of the thyroglossal duct, a structure which runs from the level of the epiglottis to the anterior mediastinum.
- It is believed that sub-epiglottic cysts are present from birth although they may not be discovered until the horse is mature and commences training.
- The cysts are smooth-walled, sometimes multilobular, structures filled with straw-coloured slightly tenacious fluid. Acquired inversion cysts are less common but arise at the same location in horses of any age.
- Both arise from within the loose glosso-epiglottic mucosa lying between the base of the tongue and the epiglottis. It is unusual to find a sub-epiglottic cyst that is not located within an epiglottal entrapment.

Clinical signs

- The age and manner by which the cysts cause clinical signs is dependent on their size.
- Large cysts can be a cause of dysphagia and respiratory obstruction in foals. Such foals may present within a few days of birth with reflux of milk from the nares and will require an endoscopic examination for differentiation from foals with palatal clefts.
- Older horses with cysts may present with a variety of respiratory signs including nasal discharge from dysphagia and abnormal respiratory sounds at exercise.
- Horses with small sub-epiglottic cysts may present with a history of choking noises under exertion and will require differentiation from DDSP.

Diagnosis

- The diagnosis is easily established by endoscopy provided that the cystic lesions are available to be seen above the soft palate (see Figure 1.4).
- In some horses with this condition persistent DDSP is present, in which case it will be necessary to resort to other diagnostic procedures such as plain and contrast radiography or oral endoscopy. It is not uncommon for a cyst to occur concurrent with entrapment of the epiglottis presumably because the overlying mucosa has become stretched by the cyst.

Treatment

- The objective of treatment is to ablate the cyst by sharp excision which can be achieved per os using an ecraseur.
- Resection by conventional ventral laryngofissure is generally simpler and safer. The technique is similar to that used for the treatment of epiglottal entrapment but the placement of stay sutures on either side of the cyst is invaluable.

Prognosis

The cystic lesions can often be excised intact, and a favourable prognosis can be given. Recurrence is unlikely.

5.24 ARYTENOID CHONDROPATHY

Aetiopathogenesis

Arytenoid chondritis (chondropathy) consists of the development of suppuration within the matrix of one or both arytenoid cartilages. The mechanism of development of micro-abscesses with discharging tracts is not clear, but young male Thoroughbreds were originally felt to be most susceptible. However, horses of any breed and age can be afflicted. The condition appears to be more prevalent in the USA than Europe.

Clinical signs

The signs associated with arytenoid chondritis arise through a combination of airway obstruction, and, when the cartilages are severely diseased, through compromised glottic protection. Thus, stridorous noises are heard when the horse is exerted, and if there is bilateral involvement there may be airway obstruction at rest. Coughing may be evident at any stage.

Diagnosis

- The diagnosis of arytenoid chondritis is primarily made by endoscopy showing distortion of the affected cartilage(s) (Figure 5.11). As the micro-abscesses develop the cartilage thickens and shows axial displacement towards the midline and reduced motility.
- In the early stages, particularly when the left side is involved, casual endoscopy may suggest a diagnosis of RLN. As the condition advances, the distortion of the cartilage becomes more obvious, and granulomatous eruptions appear on the medial face of the corniculate process.
- Contact lesions may develop on the contralateral arytenoid cartilage.
- Lateral radiographs of the larynx usually show focal mineralization even in early cases.

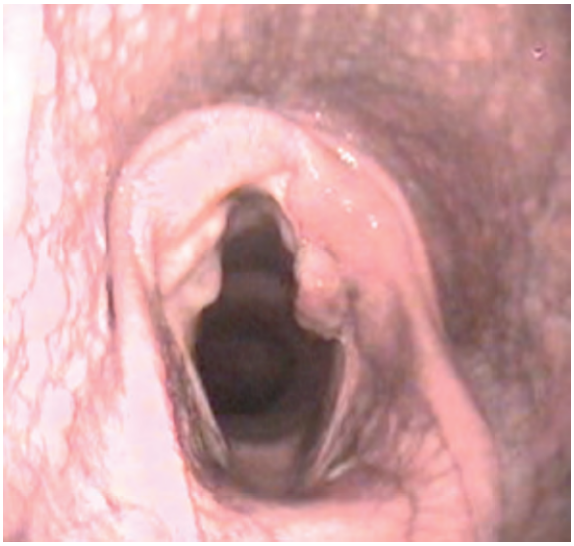


Figure 5.11 Bilateral arytenoid chondropathy in a 2-year-old colt. Note the distortion and swelling of both arytenoid cartilages as well as the axial displacement of the left side which could be mistaken for recurrent neuropathy.

Treatment

- The progress of arytenoid chondritis may be arrested in the early stages by the prolonged (i.e. 6 weeks) use of potentiated sulphonamide medication, but once the chronic stage has been reached, the only treatment option is arytenoidectomy.
- Total arytenoidectomy implies that the entire arytenoid cartilage, including the corniculate and muscular processes, is removed.
- In partial arytenoidectomy the muscular process and articular facet are left in situ, and in sub-total arytenoidectomy part or all of the corniculate process is also left in place.
- Partial arytenoidectomy is usually the preferred method to treat chondritis, and the surgery is performed through a conventional laryngotomy with a tracheotomy route to maintain anaesthesia. Most surgeons attempt to close the mucosal defect. Although it is generally difficult to lift the mucosa away from the underlying cartilage there will often be sufficient laxity in the ary-epiglottal mucosa to fashion a flap to move backwards over the defect.

Prognosis

Partial arytenoidectomy offers the best compromise to salvage a horse afflicted with laryngeal chondritis. The athletic possibilities for a horse afterwards are determined by how efficiently the diseased cartilage has been resected and how well the mucosa has healed

5.25 FOURTH BRANCHIAL ARCH DEFECTS (4-BAD)

Aetiopathogenesis

- This is a syndrome of irreparable congenital defects resulting from a failure of development of some or all of the derivatives of the fourth branchial arch.
- The structures which may be defective are the wings of the thyroid cartilage, the crico-thyroid articulation, the crico-thyroideus muscles and the crico-pharyngeal sphincter muscles.
- Any permutation of aplasia or hypoplasia of these structures may arise uni- or bilaterally. However, right unilateral defects are eight times more common than those on the left and four times that of bilateral defects.
- The absence of a firm bond between the wing of the thyroid and the cricoid cartilages deprives the larynx of a stable skeleton to facilitate the function of its intrinsic musculature. For example, in the face of such defects, the action of the crico-arytenoideus dorsalis muscle will be ineffective and the extent of arytenoid abduction is reduced. Clearly, this may convey a false impression of RLN on the affected side or sides.
- The absence of the crico-pharyngeus muscles has two obvious effects, firstly the inability to close the upper oesophageal sphincter which results in involuntary aerophagia; secondly, there is no means to anchor the palatal pillars into a position caudal to the apices of the corniculate cartilages resulting in rostral displacement of the palatal arch (RDPA).

Prevalence

The prevalence of 4-BAD in Thoroughbred horses is approximately 0.2% and the condition has been identified in other breeds such as the Hanovarian, warmbloods and the Haflinger. There is no current evidence that the syndrome is genetically transmitted.

Clinical signs

The presenting signs of horses with 4-BAD are very variable and reflect the severity of the absence of the structures involved. However, in order of frequency, the signs are:

- abnormal respiratory sounds at exercise.
- eructation.
- nasal discharge.
- coughing.
- recurrent colic.

Horses with less extensive defects may not be presented for investigation until they are mature. They may even begin to produce untoward respiratory noises having raced for a number of seasons without symptoms.

The involuntary aerophagia and eructation sometimes observed by owners may be confused with the noises produced by 'wind-suckers' (see 'Wind-sucking and crib-biting' in 5.29).

Diagnosis

- A complete evaluation of the extent of 4-BAD can only be made at MRI, exploratory surgery or post mortem, but the combined findings of palpation, endoscopy, ultrasonography and radiography are generally sufficient to justify a diagnosis.
- Palpation on its own is reliable to identify a defective thyroid cartilage – an unusually wide gap can be palpated between the caudal margin of the thyroid and the rostral edge of the cricoid whereas in the normal larynx the two structures overlap. A palpable abnormality will be present in almost all cases of 4-BAD.
- The two endoscopic features to alert the clinician to the possibility of 4-BAD are RDPA (see above) where the caudal pillars of the soft palate form a cowl which partly obscures the corniculate processes dorsally and may even leave the upper oesophagus open, and defective arytenoid motility (Figure 5.12).
- When the upper oesophageal sphincter is absent lateral radiographs will reveal a continuous column of air extending from the pharynx into the oesophagus. The RDPA is seen as a 'dew drop' intruding into this air column from the dorsal wall.

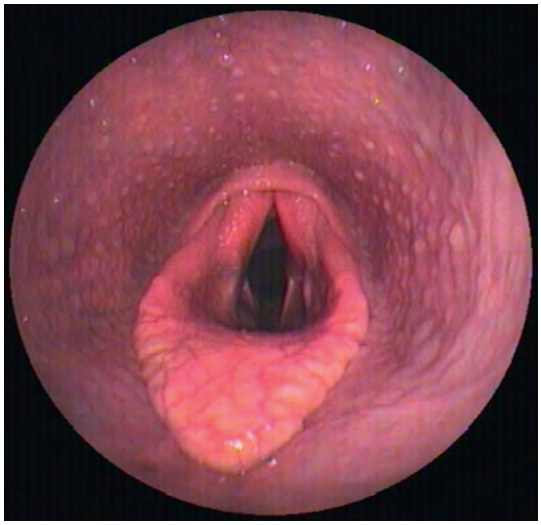


Figure 5.12 Fourth branchial arch defects. Note the rostral displacement of the palato-pharyngeal arch and the slight asymmetry of the rima glottidis.

Dynamic endoscopy is helpful to identify the structures which are impeding airflow. Axial deviation of the ary-epiglottal folds is a frequent finding in the less severe cases of 4-BAD.

Treatment

There are no effective means to reconstruct the absent structures. However, resection of the ary-epiglottal fold(s) may reduce the obstructive effect whenever ADAF has been confirmed.

Prognosis

Horses afflicted with 4-BAD are generally ineffective athletes, but the prognosis is directly related to the severity of the defects. Those horses with minor 4-BAD defects may even race effectively before beginning to produce abnormal respiratory noises. Conversely, severely deformed horses without an upper oesophageal sphincter may show repeated aerophagia leaving these horses susceptible to episodes of tympanitic colic which may be life-threatening.

5.26 AXIAL DEVIATION OF THE ARY-EPIGLOTTAL FOLDS (ADAF)

Aetiopathogenesis

The ary-epiglottal mucosal folds extend from the lateral margins of the epiglottis to the arytenoid cartilages. Dynamic collapse of one or both of these folds towards the midline is a relatively common observation during treadmill or OG endoscopy and almost always is associated with other forms of dynamic obstruction such as DDSP or ACC. ADAF may also arise on the right side of the larynx of horses which have been subjected to left-sided prosthetic laryngoplasty. The inversion of the soft tissues causes a major obstruction of the rima glottidis, but the aetiology is unknown beyond appearing to result from stretching of the mucosa itself.

Diagnosis

Those horses where ADAF occurs in isolation have a history of harsh inspiratory noises at exercise which become louder the further the exercise continues and the more intense it becomes. Confirmation of the diagnosis is by treadmill or OG endoscopy.

Treatment

Resection of the folds comprising a double layer of mucosa with minimal connective tissue in between can be achieved by conventional surgery under anaesthesia via a laryngotomy, or by diode laser resection through an endoscope passed per nasum in the standing horse. The latter is the treatment of choice.

5.27 OTHER CAUSES OF DYNAMIC AIRWAY COLLAPSE

Pharyngeal collapse

It is quite normal for the walls of the pharynx to collapse slightly during exercise and for the soft palate which makes up the floor of the nasopharynx to billow slightly upwards, but weakness of the muscular structures surrounding the airway at this level will produce a marked obstruction to air flow and reduced performance associated with abnormal inspiratory noises.

A diagnosis of pharyngeal collapse can be established only by dynamic endoscopy, and two forms of collapse are recognized. In lateral collapse the sides of the pharynx move towards the midline reducing the pharyngeal airway to a vertical slit. More common is collapse of the dorsal wall when the corniculate processes of the arytenoid cartilages become obscured by the roof of the pharynx as it moves downwards to obstruct the laryngeal additus.

The causes of pharyngeal collapse have not been determined, and neither protracted rest, corticosteroids, anti-inflammatory throat sprays, let alone any surgical remedy have been shown to be of the slightest benefit.

Ventro-axial luxation of the corniculate process of the arytenoid cartilage (VALCPA)

VALCPA comprises collapse of the dorsal wall of the rima glottidis during intense exercise whereby the left or, less frequently the right, corniculate process becomes displaced ventral to the contralateral cartilage. The cause has been suggested to be an excessively wide transverse arytenoid ligament. The majority of cases are diagnosed during dynamic endoscopy with a lesser number identified by endoscopy of the patient at rest. It is not uncommon for VALCPA to be identified concurrent with other dynamic airway obstructions such as ADAF and palatal malfunction. Afflicted horses are likely to show reduction of the cross-sectional area of the rima glottidis at exercise with associated performance-limitation and abnormal inspiratory noises. A poor outlook for athletic excellence is indicated whether the diagnosis is made at rest or during exercise because there is as yet, no effective remedy.

Epiglottal retroversion

Failure of the hyo-epiglotticus muscles leads to instability of the epiglottis during vigorous exercise such that the apex of the epiglottis turns backwards at each inspiration, causing the cartilage to obstruct the rima glottidis. Afflicted horses show performance-limitation with a progressive inspiratory noise which can become loud. Diagnosis is confirmed by dynamic treadmill or OG endoscopy. The cause of the loss of this muscle's function is unknown – it receives its innervation through the hypoglossal nerve and the nerve trunk lies well protected through most of its course. It is notable that the soft palate usually remains in a normal position in contact with the dorsal surface of the tongue and this observation provides evidence that the epiglottis has an insignificant role in stabilizing the palate.

5.28 OTHER CAUSES OF AIRWAY OBSTRUCTION IN HORSES

Choanal stenosis

Congenital narrowing of the airways at the level where the nasal meati meet the nasopharynx can be a highly subjective diagnosis. This site corresponds with the location of the bucco-nasal membrane in the developing fetus, but this should perforate and retract well before term in order that the foal can commence nose breathing immediately after birth. Persistence of the bucco-nasal membrane can occur as a membranous or bony septum, uni- or bi-laterally, to cause severe obstructions to nose-breathing with no air movement through the diseased side. Complete bilateral atresia is likely to result in asphyxiation within minutes of birth. There is no effective remedy for partial choanal stenosis, and afflicted horses show limited athletic capacity.

Pharyngeal lymphoid hyperplasia (PLH)

PLH consists of extensive follicles of lymphoid hyperplasia on the walls of the nasopharynx and pharyngeal recess. PLH, also known as 'follicular pharyngitis', has a 100% prevalence in young horses and the significance which should be attached to the finding is doubtful. There is little interference with the performance of those adolescent horses which have marked PLH, but some clinicians believe that the disturbance of laminar airflow over the protuberances causes adventitious noise and that the discomfort of engorged follicles provokes episodic gurgling through secondary DDSP.

Palatal cysts

Cysts, possibly of salivary origin, occasionally arise within the tissues of the soft palate and have been incriminated as a cause of abnormal respiratory noise in horses. They may not be easily identified by endoscopy, and contrast radiography of the oropharynx is helpful to outline them. Access for surgical dissection is difficult, but ventral laryngotomy offers the best option. Iatrogenic fenestration of the soft palate is a likely surgical complication when excision is attempted, and the prognosis for horses with these cysts becoming useful athletes is not good.

Palatal defects

Defects of the hard and soft palate may be congenital or iatrogenic (see 'Congenital and acquired palatal defects' in 1.3). Although the overwhelming majority of horses with palatal defects are presented with a history of dysphagia, the more subtle signs of long-standing clefts include a cough not invariably related to feeding and respiratory noise associated with palatal instability. Epiglottal entrapment is a frequent concurrent finding with long-standing congenital palatal defects.

Iatrogenic palatal defects arise through over-enthusiastic resection surgery for DDSP (see 'Treatment' in 5.21), and the most common sequel is chronic palatal instability leading to more severe signs of DDSP.

Pharyngeal cysts

Congenital cysts arising from the pharyngeal walls arise occasionally in horses, and the stage at which clinical signs are noted probably depends on their size. Diagnosis by endoscopy is invariably straightforward. The origin of these cysts is not known, but they are generally amenable to surgical excision.

Pharyngeal paralysis

Although guttural pouch mycosis is the most common cause of pharyngeal paralysis (hemiplegia) (see 'Clincal signs' in 5.6) this neuropathy can arise following heavy metal poisoning and in botulism. Although dynamic collapse of the pharyngeal walls and intractable DDSP would lead to partial asphyxiation during exercise, this is most unlikely to be a major presenting sign (see 'Pharyngeal paralysis' in 1.4).

Pharyngeal and laryngeal neoplasia

All tissues which are rich in lympho-reticular elements are susceptible to lymphosarcoma development, and the naso-pharynx and larynx are no exceptions. Other tumours at these locations are very rare. However, it should be noted that arytenoid chondropathies (5.24) frequently resemble tumours.

Congenital tracheal collapse

Congenital narrowing of the trachea through flattening of the cartilage rings is uncommon in horses. However, it is occasionally encountered in miniature Shetland ponies. Afflicted horses are presented with persistent coughing and adventitious respiratory sounds at exercise. There is no effective means to correct this deformity.

Acquired tracheal obstructions

The tracheal cartilage rings are vulnerable to fracture by kicks from other horses or by running against paddock rails. Inept tracheotomy intubation (see the next section) may also produce transection of tracheal rings comparable to fractures. The likely result of these injuries is narrowing of the airway in the form of a stricture or stenosis. Such lesions are usually palpable from the exterior, and the luminal distortion can be confirmed by a combination of endoscopy and radiography.

Only narrow stricture bands may be treated by tracheal resection and anastomosis, but even so the tension in the repaired incision is such that recurrence of the cicatrix is common. Longer segments of tracheal distortion may be treated by the application of an external prosthesis against which the collapsed cartilage rings are conformed. The prognosis for the successful repair of acquired tracheal stenoses is guarded.

Tracheotomy intubation

Tracheotomy intubation comprises the implantation of a tube directly into the trachea to provide an alternative airway when the upper respiratory tract is completely or partly obstructed or when it may become obstructed, for example after surgery. It can be used as the primary means to manage pharyngeal or laryngeal obstructions or when other methods to treat RLN or arytenoid chondritis have failed (see 'Treatment' in 5.20).

The surgery is performed through a ventral midline incision on the standing, possibly sedated, horse under local anaesthesia.

After clearing the overlying muscle and fascia, an incision is made into the lumen without transection of a complete tracheal cartilage ring. Thus, semi-circles of cartilage are removed from adjacent tracheal rings leaving a narrow bridge of cartilage on either side of the hole by which to maintain the structural stability of the trachea.

A self-retaining metallic tube is placed for racing purposes. A similar technique may be used for anaesthetic maintenance when the presence of an endotracheal tube passed *per os* is an encumbrance to the surgeon.

It should be noted that horses that have undergone tracheotomy intubation are not allowed for competition by some racing or equestrian authorities.

5.29 MISCELLANEOUS THROAT CONDITIONS OF HORSES

Wind-sucking and crib-biting

Wind-sucking and crib-biting are well recognized examples of equine stereotypic behaviour whereby horses grasp fixed objects between their incisor teeth, arch the neck and emit a characteristic noise. There is evidence that the behaviour commences as a means to stimulate saliva production to buffer gastric acidity especially around the time of weaning.

- Deglutition and the passage of air through the oesophagus to the stomach do not occur.
- The behaviour involves the contraction of the strap muscles of the neck to create a pressure gradient across the oesophageal wall so that air suddenly rushes from the pharynx into the upper oesophagus. As the muscles relax, the air returns to the pharynx.
- The role of the crib-biting component of the activity is that the grasping of fixed objects in the stable environment facilitates the arching of the neck and contraction of the strap muscles.
- The characteristic grunting sound associated with wind-sucking derives from the rush of air from pharynx to oesophagus.

The surgical methods used to treat wind-sucking all aim to inhibit the effectiveness of contraction of the ventral muscles of the neck, and Forssell's operation and its variants excise or denervate the sterno-hyoideus, sterno-thyroideus, omo-hyoideus and sterno-mandibularis muscles.

Salivary gland disorders

- Salivary gland disorders are uncommon in horses.
- The relatively superficial positions of the parotid gland and its duct renders them vulnerable to laceration wounds. Saliva is saturated with calcium salts, and any leakage of saliva to the skin surface can be differentiated from serum, for example, by the chalky deposits on the surrounding area.
- Fresh wounds to the parotid duct should be closed by simple anastomosis around a pre-placed catheter which is passed from the exterior into the buccal cleft through a stab wound before entering the duct via the parotid papilla.
- Wounds to the glandular tissues themselves are generally self-limiting but can produce considerable leakages of saliva for 2–3 weeks while the defects resolve.
- Chronic salivary fistulation is rare, but destruction of the glandular tissue offer a superior solution to complete excision of the injured gland.
- Stricture of the duct may follow trauma, and this will produce a distended painless fluid-filled structure corresponding to the course of the duct. The parotid gland can be destroyed by the introduction of a number of chemical agents such as ether, concentrated iodine solution or 10% formalin. The author's preference is formalin which is introduced into the gland by way of the isolated duct and approximately 30 mL is left in situ for 90 seconds before being flushed clear with normal saline solution. Salivary secretion should cease within 3 weeks.
- Salivary calculi invariably involve only the parotid system and generally come to obstruct the major duct close to the parotid papilla. The resultant swelling is similar to that which follows a stricture, but the calculus is usually palpable.
- Sometimes the sialolith can be delivered through the papilla using forceps, but otherwise a direct surgical incision onto the calculus is made.
- Seasonal sialadenitis is an idiopathic, usually bilateral, swelling of the parotid glands which is typically seen in grazing horses in spring/summer. This condition has no clinical significance other than the owner's concern regarding the physical appearance of the swollen glands.

Thyroid neoplasia

- Benign adenomas and C-cell tumours of the thyroid glands produce discrete mobile swellings on one or other side of the larynx.
- The lesions should not be confused with normal thyroid glands which are the size of a small hen's egg and are frequently suspended by a loose pedicle on either side of the cricoid cartilage.
- Thyroid tumours are not usually functional, and therefore their significance simply relates to the physical presence of the swelling which is likely to cause concern to the owners long before there is compression of the upper respiratory tract.
- These tumours are easily removed by surgery provided that care is taken with their vascular supply; a ventral midline approach is recommended.
- Malignant tumours of the thyroid glands are rare.

Parotid melanomas

Melanomas are common tumours of grey horses from middle age onwards. They are most commonly located in the perineum, but the parotid region is the site next most frequently involved. The parotid melanomas may not be the primary tumours, but secondary deposits within the salivary gland. The primary melanomas may be derived from ectopic melanosis in the lateral compartment of the adjacent auditory tube diverticulum.

- There are no effective treatments for parotid melanomas.
- Medical management using cimetidine has been reported as helpful but, in the author's hands, has been consistently ineffective.
- Any attempt to excise the secondary parotid lesions should be avoided.
- Thus, the significance of these tumours should be clarified with the potential purchaser of a grey horse with parotid swellings. Expansion of the untreated lesions will initially limit mobility at the upper neck, and spread to adjacent sites may cause dyspnoea or dysphagia.

Facial palsy

See Chapter 11.

In many instances facial paralysis is iatrogenic, and in most cases this insult is avoidable. The buccal branches are vulnerable during dental extractions, but the nerve tracts can be identified and retracted or bypassed. During general anaesthesia and recovery from it, the nerve trunk is particularly vulnerable to insult by unpadded head collars as it passes over the caudal border of the vertical ramus of the mandible.

Fortunately facial nerve palsies often show spontaneous complete or partial recovery in the months following injury, but the time taken for the improvement to occur depends on the distance between the site of trauma and the denervated muscle.

Horner's syndrome

See Chapter 11.

The two most likely causes of Horner's syndrome are injury to the cervical pre-ganglionic pathway for example by perivascular reaction to the vago-sympathetic trunk, and guttural pouch mycosis where the mycotic plaque infiltrates the nerve pathway in the region of

the cranial cervical ganglion. The level at which the hemifacial sweating commences provides a firm guide to the level of the injury. In cases of peri-vascular reaction the skin over much of the side of the neck may be persistently damp.

Jugular thrombophlebitis

See Chapter 7.

Nuchal fractures

Avulsion fractures of the nuchal crest of the occipital bone arise from direct trauma to the poll region. Afflicted horses show acute onset inability to raise the head from the ground, but their co-ordination is otherwise normal. Radiographs confirm the diagnosis. Avulsion fractures should be managed conservatively as a fibrous union will form within weeks, and a full range of mobility will return. In the early stages food and water should be offered from ground level.

Cervical injection infection and abscesses

Suppuration may develop in the segmental muscles on either side of the nuccal ligament through contaminated injections. The development of a hot painful swelling at the side of the neck in the days following an intramuscular injection make the diagnosis straightforward. Afflicted horses may be febrile and are likely to show restricted neck movement. Treatment based on first principles dictates that the abscess should be encouraged to drain, and ultrasonography can be helpful to establish the extent of free pus in the lesion and the optimal site for lancing. Chronic abscesses at this site are inclined to infiltrate between the fascial plains and may require aggressive surgery to expose and drain all of the infected tissue.

FURTHER READING

McGorum BC, Dixon PM, Robinson NE, Schumacher J (2007) Equine respiratory medicine and surgery. Saunders, Edinburgh

Rush BR, Mair TS (2004) Equine Respiratory Diseases. Blackwell, Oxford

Proceedings of the fifteenth Bain-Fallon Memorial Lectures: Equine head and hind limb medicine and surgery 1993 Australian Equine Veterinary Association, Artarmon, NSW, Australia

APPENDIX 1

Differential diagnostic list of causes of respiratory obstruction and/or abnormal respiratory noise in the horse

The following list itemizes the potential causes of obstructive dyspnoea on the basis of location in the upper respiratory tract.

Nostrils:

- Wry nose.
- Post-traumatic stenosis.
- Collapse resulting from facial palsy.
- Alar fold hypertrophy.

Nasal chambers:

- Deviation and deformity of midline nasal septum.
- Sinus empyema.
- Sinus cyst.
- Progressive ethmoidal haematoma (PEH).
- Polyp formation.
- Neoplasia.

Nasopharynx:

- Choanal atresia/stenosis.
- Palatal defects: congenital, iatrogenic.
- Lymphoid hyperplasia (PLH).
- Dynamic collapse of pharyngeal walls.
- Instability of soft palate.
- Dorsal displacement of soft palate (DDSP).
- Intra-palatal cysts.
- Pharyngeal cysts.
- Pharyngeal paralysis.
- Neoplasia.
- Extra-mural distortion by: strangles abscess, ATD distension, neoplasia.

Larynx:

- Epiglottal hypoplasia.
- Epiglottal entrapment (EE).
- Sub-epiglottic cyst.
- Epiglottitis, including foreign body-induced.
- Retroversion of epiglottis.
- Fourth branchial arch defects (4-BAD syndrome).
- Axial deviation of ary-epiglottal fold(s) (ADAF).
- Recurrent laryngeal neuropathy (RLN) idiopathic, iatrogenic and other acquired.
- Dynamic vocal cord collapse (VCC).
- Dynamic arytenoid cartilage collapse (ACC).
- Ventro-axial luxation of corniculate process(es) of arytenoid cartilages (VALCPA).
- Laryngeal granuloma.
- Arytenoid chondropathy.
- Neoplasia.

Trachea:

- Congenital collapse.
- Traumatic ring fracture.
- Post-traumatic, including iatrogenic, stenosis/stricture.

APPENDIX 2

Differential diagnosis of non-infectious nasal discharge in the horse

	Sinu-nasal conditions							
	Acute sinus empyema	**Chronic sinus empyema**	**2° dental sinus empyema**	**Ethmoidal haematoma**	**Sinus cyst**	**Conchal necrosis**	**Sinu-nasal neoplasia**	**ATD mycosis**
1. History (• = typical sign, x = occasional finding)								
Discharge								
Acute epistaxis				x			x	•
Recurrent epistaxis				•			•	
Exercise induced epistaxis								
Mucopurulent	•	x	x	x	•		x	
Purulent		•	•	x	x	•	x	
Malodorous		•	•	x	x	•	x	x
Ingesta								x
Unilateral	•	•	•	•	•	•	•	•
Bilateral								x
External swelling		•		x	•		•	
Coughing								
Respiratory obstruction		x		x	•		•	
2. Diagnostic procedures (a) (• = often positive, x = occasionally helpful)								
External observation								
Exaggerated expiratory effort								
Unequal airflow at nares		x		x	•		•	
Trial allergen-free management								
Facial distortion	x	•		x	•		•	
Palpation								x
Auscultation								
Percussion		x		x	•		•	
Oral examination			•			•		
Oesophageal manometry (*Ventigraph*)								
Exercise test								
Attempt pass stomach tube								

Continued

	ATD conditions			Dysphagia				1°pulmonary conditions		
	ATD empyema	ATD diverticulitis	Palatal defects	Pharyngeal paralysis	Laryngeal disorders	Oesophageal obstruction	Grass sickness	EIPH	COPD, small airway inflammation	Pleuro-pneumonia
								•	x	
								x		
								•	x	
	•	•	x	•	x	x			•	x
	x	x								•
										•
		x	•	•	•	•	•			
	•	•						x	x	
	x	x	•	•	•	•	•	•	•	•
	x									
	x	x	•	•	•	•	•	x	•	
	x			x	•					
									•	•
									•	
	x				•	•				
									•	•
										•
			x							
									•	
					•			•	•	
				•		•	•			

Continued

APPENDIX 2

(*Continued*)

	Sinu-nasal conditions							
	Acute sinus empyema	**Chronic sinus empyema**	**2° dental sinus empyema**	**Ethmoidal haematoma**	**Sinus cyst**	**Conchal necrosis**	**Sinu-nasal neoplasia**	**ATD mycosis**
3. Diagnostic procedures (b) (• = often positive, x = occasionally helpful)								
Endoscopy								
Nasal meati	•	•	x	•	•	•	•	
Direct sinus endoscopy	x	x	x	•	•	x	•	
Pharynx/larynx/ ATDs								•
Trachea including aspiration								
Radiography/ultrasonography								
Erect lateral sinu-nasal area	•	•	x	•	•	•	•	
30-degree oblique maxilla	x	x	•	x	x	x	x	
Ventrodorsal		•		•	•	x	•	
Pharynx/larynx/ ATD/oesophagus								x
Carotid angiography								•
Chest								
Exploratory surgery and biopsy		x			•	x	•	

	ATD conditions		Dysphagia					1°pulmonary conditions		
	ATD empyema	ATD diverticulitis	Palatal defects	Pharyngeal paralysis	Laryngeal disorders	Oesophageal obstruction	Grass sickness	EIPH	COPD, small airway inflammation	Pleuro-pneumonia
	•	•	•	•	•					
								•	•	•
	•	x			•	x	x			
			x	x	x	x	x	x	x	• + U
					x		•			

Chapter 6

Lower respiratory tract

Tim S. Mair, Bonnie R. Rush

CONTENTS

6.1 Diagnostic approach to lower respiratory tract diseases 111
6.2 Diagnostic features of the common lower respiratory tract diseases of the adult horse 117
6.3 Recurrent airway obstruction (RAO) 117
6.4 Acute obstructive pulmonary disease 121
6.5 Summer pasture-associated obstructive pulmonary disease (SPAOPD) 122
6.6 Inflammatory airway disease (IAD) 122
6.7 Exercise-induced pulmonary haemorrhage (EIPH) 123
6.8 Lungworm (*Dictyocaulus arnfieldi*) infection 124
6.9 *Parascaris equorum* infection 124
6.10 Inhaled foreign bodies 124
6.11 Pulmonary/pleural/mediastinal neoplasia 124
Primary lung tumours 124
Lung metastases 124
Clinical features of lung tumours 124
Primary pleural tumours 124
Pleural metastases 124
Mediastinal tumours 125
6.12 Pleuropneumonia 125
6.13 *Rhodococcus equi* pneumonia 126
6.14 Interstitial pneumonia/chronic interstitial inflammation 127
6.15 Acute interstitial pneumonia in foals 128
6.16 Fungal pneumonia 128
6.17 *Pneumocystis* pneumonia 128
6.18 Pulmonary oedema 128
6.19 Drowning/near drowning 129
6.20 Pulmonary congestion 129
6.21 Haemothorax 129
6.22 Pneumothorax 129
6.23 Ruptured diaphragm 130
6.24 Pleural transudates 130
6.25 Smoke inhalation 130
Further reading 131
Appendix 131

6.1 DIAGNOSTIC APPROACH TO LOWER RESPIRATORY TRACT DISEASES

The major clinical signs associated with respiratory diseases include *coughing, nasal discharge, dyspnoea* and *exercise intolerance*.

History

History and signalment can be important in aiding diagnosis of horses with respiratory diseases. Important points include:

- Age – many contagious diseases, including strangles and *Rhodococcus equi* infection, are most frequently seen in young horses, whereas allergic diseases such as recurrent airway obstruction (RAO) are usually seen in mature horses.
- Health of in-contact horses – similar clinical signs among in-contact horses suggests contagious disease.
- Environment – RAO is most likely in stabled horses or horses exposed to hay and/or straw. *Dictyocaulus arnfieldi* infection is most likely in horses grazing pasture contaminated by donkeys.
- Seasonality – seasonal disease in horses at grass is suggestive of summer pasture-associated obstructive pulmonary disease (SPAOPD).
- Stress – severe respiratory disease occurring in a recently stressed horse (such as after long distance travel or surgery) should arouse suspicion of pleuropneumonia.

Physical examination

The physical examination should be performed in a quiet environment with the horse relaxed and rested. Examination during and after exercise may be required subsequently.

Systemic and extra-thoracic signs that may occur with lower respiratory tract disease include:

© 2013 Elsevier Ltd
DOI: 10.1016/B978-0-7020-2801-4.00006-7

- Pyrexia.
- Weight loss.
- Elevated heart rate (tachycardia).
- Weak pulse.
- Dehydration.
- Signs of pain.
- Cyanosis.
- Peripheral subcutaneous oedema.
- Pectoral oedema.
- Subcutaneous emphysema.
- Hypertrophic osteopathy (Marie's disease).

Respiratory rate, pattern and character

The rate, depth, and pattern of breathing should be noted, as should any abnormal sounds associated with breathing. This is best done by distant observation with the horse in a quiet location. The breathing is most easily assessed by observing the movement of the costal arch whilst standing caudolaterally to the horse. Alternatively, the respiratory rate may be measured by feeling airflow with a hand placed close to the external nares.

- Normal respiration in the resting adult horse is slow (8–16 breaths/min) with minimal chest or abdominal wall movement. Ponies may have a slightly higher resting respiratory rate (up to 20 breaths/min). During quiet breathing, resting horses normally have only slight movements of the nostrils and costal arch. Increased depth of breathing (with or without an increased rate) is abnormal, and its character should be noted.
- Normal horses have biphasic inspiratory and expiratory phases. The end expiratory abdominal lift is often visible in normal horses, and does not necessarily indicate disease.
- Expiratory dyspnoea (Box 6.1) results in an exaggeration of the biphasic expiratory phase with increased incorporation of the abdominal muscles producing an obvious biphasic or double expiratory lift ('heave'), which is typical of small airway obstruction.
- Inspiratory dyspnoea associated with a stertorous or stridorous noise during inspiration (Box 6.1) is indicative of upper airway obstruction. Occasionally, inspiratory dyspnoea may occur with severe restrictive lung diseases (e.g. pneumonia, interstitial disease, pneumothorax, rib fracture).
- Combined inspiratory and expiratory dyspnoea, usually with tachypnoea, is suggestive of severe upper or lower airway obstruction, diffuse pulmonary disease or pleural disease.

Box 6.1 Characteristics of inspiratory and expiratory dyspnoea

Expiratory (obstruction of intrathoracic airways)

Prolonged and laboured expiration
Exaggerated expiratory contraction of the abdominal muscles
Heave line in chronic cases
Rhythmic pumping of the anus
Dilation of the nares throughout the respiratory cycle
Extension of the head and neck

Inspiratory (obstruction of extrathoracic airways, restrictive lung diseases, thoracic tumours)

Prolonged and laboured inspiration
Increased respiratory effort
Dilation of the nares throughout the respiratory cycle
Extension of the head and neck

Nasal discharge

- Unilateral nasal discharges usually originate from the nasal chamber and paranasal sinuses. Drainage from the guttural pouch will be reported by the owner as unilateral or predominately unilateral discharge.
- Bilateral discharges can arise from the upper or lower respiratory tracts.
- Serous nasal discharges are commonly seen in viral infections, although they frequently change to mucopurulent or purulent with secondary bacterial infection.
- The presence or absence of odour or food material in the discharge is also helpful in determining the cause.

Cough

- Coughs originating from upper airway infections are usually harsh, dry, hacking and non-productive.
- Coughs originating from the lower airway are usually deep, soft and productive. The intra-narial position of the equine larynx usually precludes the coughing up of sputum directly into the mouth, but the observation of swallowing after a cough indicates that the cough is productive.
- Short, painful coughs, associated with reluctance to move or to lie down, are typical of pleural disease and pain (pleurodynia).

Palpation

- Laryngeal palpation – see Chapter 5.
- The cervical trachea is palpated for abnormalities of structure. The space between the cricoid cartilage and the first tracheal cartilage ring (the cricotracheal ligament) is normally wide. Palpation of the cervical trachea can detect pain, swellings, deformities, developmental defects (e.g. dorso-ventral or lateral collapse), and misalignment of the free borders of tracheal rings.
- The bases of the jugular grooves are palpated for the presence of a mass which may indicate a mediastinal tumour that has extended rostrally through the thoracic inlet; these cases often have subcutaneous sternal / pectoral oedema as well, due to venous obstruction.
- Distension of the jugular veins or the presence of an abnormal jugular pulse indicates primary cardiac disease or compression of the heart/vessels by a mass or fluid.
- The chest wall should be palpated if there is a possibility of thoracic trauma (e.g. newborn foals with rib fractures).

Auscultation

Auscultation is performed on both sides of the chest and over the cervical trachea. Lung sounds will vary depending on the body condition and depth of breathing. In fat horses, airflow sounds may be difficult to appreciate; these sounds can be accentuated by using a *re-breathing bag* (place a large plastic bag over the nose and let the horse breathe into and out of the bag until the arterial carbon dioxide tension increases, thereby increasing the depth of respiration) (Figure 6.1) or by temporarily *occluding the nostrils*. The normal margins of the lung fields are level with the tuber coxae at the 18th rib, mid thorax at the 13th rib, shoulder at the 11th rib, and then curving down to the level of the elbow (Figure 6.2).

- Normal airflow sounds are most readily appreciated over the distal cervical trachea and at the area around the carina, and may be difficult to perceive at the lung periphery.
- Normal airflow sounds are slightly louder on the right side than the left and louder during inspiration than during expiration.
- Increased audibility of normal breath sounds occurs with hyperventilation and focally over areas of consolidated lung tissue.

Figure 6.1 Use of a re-breathing bag. The horse is forced to breathe into and out of a large bag secured over the nose. This results in increased depth of respiration, which accentuates normal and abnormal lung sounds.

Figure 6.3 Thoracic percussion. The fingers of one hand are sharply tapped onto the middle finger of the other hand, which is placed over the intercostal space.

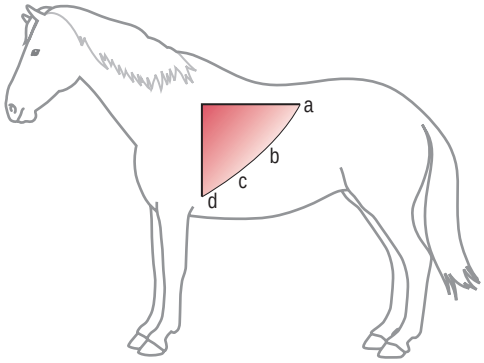


Figure 6.2 Normal area of auscultation of the lungs. (a) Tuber coxae at the 18th rib. (b) Mid thorax at the 13th rib. (c) Level with the shoulder at the 11th rib. (d) Point of the elbow.

- Reduced audibility of normal lung sounds is common in obese horses. Regional loss of lung sounds occurs when the pleural cavity contains air, fluid or abnormal tissue.
- A generalized increase in the intensity of airflow sounds is suggestive of lower airway disease such as RAO.
- A localized absence of sounds may indicate a pulmonary or pleural abscess/tumour.
- Absence of sounds in the ventral thorax (may be bilateral) suggests a pleural effusion.
- Absence of sounds in the dorsal thorax is suggestive of pneumothorax.
- It is not unusual to detect borborygmi referred from the abdomen during auscultation of a normal thorax.

Adventitious sounds (i.e. abnormal sounds) include *crackles* (rales), *wheezes* (rhonchi) and pleural friction sounds.

- Wheezes and crackles can sometimes be heard at the nares with the naked ear.
- Crackles are produced by the sudden 'popping' open of airways that were closed during expiration. They are most commonly heard during inspiration, and can be detected in RAO and acute obstructive pulmonary disease, and pulmonary oedema.
- Wheezes are musical notes produced by air flowing through narrowed airways. They occur in obstructive lower airway diseases including RAO and bronchopneumonia.
- Coarse crackles may be heard over the distal cervical trachea (tracheal rattle). These are 'bubbling and crackling' sounds caused by air bubbling through excessive mucus in the airways.
- The gliding movements of the visceral and parietal pleura are normally silent. Pleural friction sounds are crunching/creaking sounds that may be detected early in cases of pleuritis, due to the rubbing together of inflamed pleural surfaces. They are usually absent once a pleural effusion has formed.

Percussion

Percussion may be used to detect pleural and superficial pulmonary lesions. It is less sensitive than ultrasonography. A plexor and pleximeter may be used, or, alternatively, the fingers can be employed to sharply tap over the intercostal spaces (Figure 6.3). The area of percussion is similar to the area of auscultation, remembering that there is an area of cardiac dullness (larger on the left than the right) in the ventral thorax. The entire chest on both sides should be percussed working in parallel lines from dorsal to ventral and anterior to posterior.

- Normal lung tissue sounds resonant and hollow.
- Fluid or soft tissue sounds dull and flat.
- Pain on percussion is observed in early pleuritis.

Endoscopy

Endoscopy allows direct visualization of many parts of the respiratory tract, and the use of flexible fibreoptic or videoendoscopic equipment has become an essential component in the evaluation of respiratory tract diseases. The parts of the tract that are accessible to examination are determined by the equipment available. Although a narrow 1 or 1.2 m endoscope will permit examination of most parts of the upper respiratory tract in adult horses, it is unlikely to have sufficient length to allow examination of the bronchial tree – an instrument of 2 m or longer is needed for bronchoscopy. Fine paediatric endoscopes may

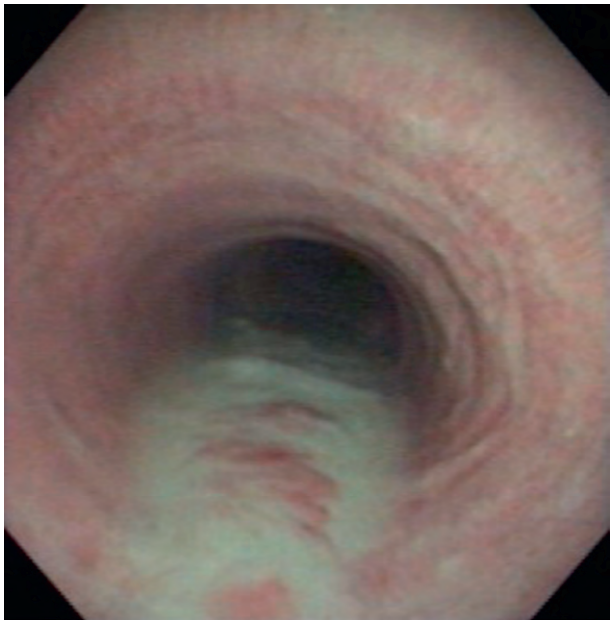


Figure 6.4 Endoscopic view of tracheal exudate accumulating in the distal cervical trachea.

Figure 6.5 Endoscopic view of the tracheal bifurcation (carina) of a horse affected by RAO showing exudate within the airways.

be required for foals. Adequate disinfection of the instrument between horses is essential to prevent cross-infection.

The horse is restrained as necessary. A nose twitch is usually sufficient, and this helps stabilize the head. Chemical sedation is sometimes required. It is helpful to have three people to perform the examination: one holds the horse and twitch; one stabilizes the endoscope at the nostril, and one has charge of the controls. For routine examinations, the endoscope is introduced through one nostril and passed along the ventral nasal meatus into the nasopharynx. In most horses, an endoscope can be introduced from the nasopharynx into the larynx without inducing significant coughing. However, in horses affected by lower airway diseases, this manoeuvre may induce a severe cough response. Coughing may be abolished by spraying a dilute lidocaine solution over the larynx (via a catheter passed through the accessary port of the endoscope). Sedation with xylazine, detomidine or romifidine may also diminish the cough response.

The trachea can be examined in its entirety if a sufficiently long endoscope is used (>1.8 m). Strictures of the tracheal lumen (congenital, iatrogenic or traumatic) can be observed, although radiography may yield more useful information. Discharges often accumulate at the thoracic inlet (Figure 6.4), and their nature (mucus, purulent material, blood) can be assessed and samples obtained by aspiration. A haemorrhagic discharge observed after exercise is suggestive of *exercise-induced pulmonary haemorrhage*. Foreign bodies such as brambles may lodge in the distal trachea/main bronchi and are an uncommon cause of chronic coughing – they can usually be identified and retrieved during endoscopy.

The bronchial tree can be examined as far as the length and diameter of the endoscope permits. The carina (Figure 6.5) normally presents as a sharp angle at the junction of the right and left principal bronchi. Thickening of the angle (owing to mucosal oedema and inflammation) and hyperaemia may be seen in chronic lower airway diseases. A unilateral purulent discharge draining from only one principal bronchus indicates a focal lung lesion on that side (e.g. focal pneumonia, pulmonary abscess or foreign body). Passage of the endoscope down the bronchial tree may induce significant coughing which renders the examination difficult. This reaction can be reduced by repeated infusions of small volumes of dilute lidocaine solution as the endoscope is advanced. The bronchial walls can be assessed for thickening, inflammation and collapse. Lungworms (*Dictyocaulus arnfieldi*), foreign bodies and endobronchial tumours may be identified.

Tracheal aspiration

Samples of lower airway secretions may be obtained for cytology or culture. Tracheal aspirates can be collected by endoscopy or by a transtracheal technique. In the former, the flexible endoscope is passed in the usual way via the nares into the trachea. When the tip has reached the distal trachea, a catheter is passed down the accessory port until it protrudes from the end. Sterile saline (10–15 mL) is injected through the catheter, which forms a pool at the thoracic inlet. The catheter tip is positioned into this pool and the sample is aspirated. Only a small proportion of the delivered fluid is likely to be retrieved. Aspirates from normal horses contain mainly ciliated epithelial cells and macrophages. Approximately 10% of the cells are neutrophils, although some stabled horses may have up to 40% neutrophils.

- An increase in numbers of neutrophils indicates inflammatory lower airway disease, such as RAO, bronchopneumonia, etc.
- Large numbers of eosinophils are seen in lungworm infections and ascarid migrations in young horses.

Samples collected in this way are suitable for bacterial culture, but culture results must be interpreted with caution since the endoscope is unavoidably contaminated by respiratory pathogens residing in the upper respiratory tract. Guarded aspiration catheters are available that will reduce the risk of contamination of samples obtained in this way. The endoscope must be thoroughly disinfected if samples are being obtained for microbiological culture (and to prevent cross-contamination between horses). Contamination of flexible endoscopes by *Pseudomonas* spp. is common. If the horse coughs frequently during endoscopic tracheal aspiration, there will be an increased risk of contamination by oropharyngeal organisms.

Samples obtained by transtracheal aspiration are collected aseptically and are suitable for both cytology and culture. An area over the

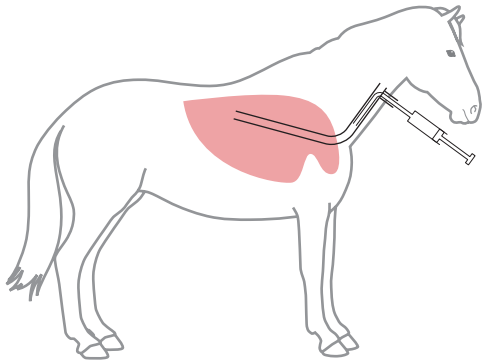


Figure 6.6 Transtracheal aspiration. A catheter is passed into the tracheal lumen through a cannula placed between two tracheal rings in the cervical trachea. Saline is infused and aspirated at the level of the distal cervical trachea.

lower third of the cervical trachea is clipped and prepared aseptically, and an over-the-needle cannula is inserted between two tracheal rings into the tracheal lumen. A catheter is then threaded through the cannula and passed to the level of the thoracic inlet where lavage with 20–30 mL of sterile saline is performed (Figure 6.6). In cases of lower airway or pulmonary infection, a Gram-stained smear of a sample obtained in this way may give an early indication of the types of bacteria involved before culture results are available. Both aerobic and anaerobic cultures should be performed in cases of pneumonia/lung abscess.

Several complications may arise following the transtracheal aspiration technique:

- Damage to the tracheal cartilage, with resultant chronic infection.
- Breaking of the catheter within the trachea. Most horses will cough up the damaged catheter within 30 minutes.
- Breaking of the catheter in a subcutaneous site as a result of initial misplacement of the cannula/needle. The catheter must be removed surgically in these cases.
- Local infection/cellulitis at the site of tracheal puncture owing to contamination of the site by bacteria from the lower airways.
- Laceration of a carotid artery resulting in rapid cervical swelling and dyspnoea.

Bronchoalveolar lavage (BAL)

BAL is used to obtain samples of lower airway secretions, primarily for cytology, from the small airways and alveoli. The technique may be performed through an endoscope (which is lodged in a bronchus prior to lavage) or blindly using a catheter passed *per nasum* into the bronchial tree.

Cytology of BAL fluid provides a more accurate reflection of the state of the small airways and alveoli than that of tracheal aspirates. BAL is ideally suited to the assessment of diffuse lower airway and alveolar diseases. When performed blindly, the segment of lung being lavaged is unknown, and the technique should, therefore, not be used if focal lung disease is suspected (including exercise-induced pulmonary haemorrhage (EIPH), pneumonia, lung abscess). Endoscopy permits more accurate placement of the lavage tube, but the precise location can still be difficult to assess.

Endoscopic BAL technique

A 120 cm (or longer) endoscope is passed in the usual way to the carina and then into a mainstem bronchus. It is advanced down the bronchial tree until it wedges in an airway (usually about a fourth to sixth generation bronchus if an endoscope with an external diameter

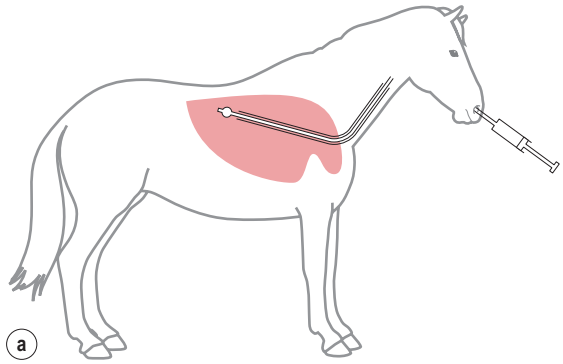


Figure 6.7 (a, b) Bronchoalveolar lavage. A catheter is passed into the distal bronchial tree, where saline is infused and aspirated.

of >8–10 mm is being used). Lavage is performed via the accessory channel of the endoscope with or without a catheter. Coughing can be severe as the endoscope passes down the bronchial tree. In most horses this abates after 10–20 seconds, but in horses with airway inflammation coughing can be persistent, in which case this can be reduced by infusing a small volume of dilute lidocaine solution (0.4%) at each bronchial division.

The volume and type of fluid used for BAL is a matter of personal preference. Most clinicians use sterile pre-warmed saline, infused in 50 mL aliquots to a total volume of about 300 mL. The retrieval rate for this fluid varies, but is usually in the region of 50–80%. Normal BAL samples will appear foamy due to the presence of surfactant, and this indicates a good sample. The samples should be pooled together prior to evaluation.

Blind BAL technique

Commercially available nasobronchial BAL tubes should be used for the purpose when the technique is performed blindly. The tubes usually have an inflatable cuff at the distal end. With the horse's head extended, the tube is passed in the same way as the endoscope through the nasopharynx, into the trachea until a cough reflex is elicited. Infusion of dilute lidocaine solution (as for the endoscopic technique) will alleviate the coughing, and permit the tube to be advanced down the bronchial tree (the tube most commonly enters the right dorsocaudal lung). When the tube becomes lodged and will not advance any further, the cuff is gently inflated with 5–10 mL of air, lavage fluid is instilled and the sample is recovered by manual or automated aspiration (as for the endoscopic technique) (Figure 6.7a,b). Excessive

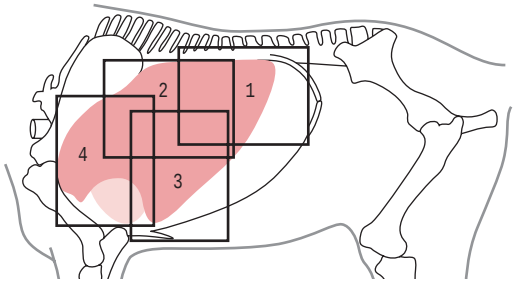


Figure 6.8 Thoracic radiography in the adult horse. A series of overlapping radiographic fields are required to cover the entire thorax.

Figure 6.9 Ultrasonogram of a typical pleural effusion. There is a hypoechoic area within the pleural cavity (fluid) containing the pericardiodiaphragmatic ligament, and consolidated lung deep to the fluid.

negative pressure for aspiration (manual or automated) will collapse small airways, limiting the recovery volume, and may result in blood contamination of the sample.

Complications of BAL are minimal. A mild neutrophilic inflammatory response in the lavaged lung segment is expected. Occasionally, mild pyrexia can be detected for about 24 hours after the procedure.

Thoracic radiography

In adult horses, only lateral views are possible. Four overlapping fields are often required to cover the entire thorax in adult horses (Figure 6.8). An air gap is used between the patient and the film to avoid the use of a grid. Lesions that can be detected include pulmonary consolidation/pneumonia, lung abscesses, advanced EIPH lesions, lung infiltrates such as neoplasia, granulomas and fibrosis, and pleural effusions.

Diagnostic ultrasound

Ultrasonography is well suited to the investigation of pleural diseases, especially pleural effusions, where the technique can provide some clues as to the nature of the fluid (i.e. transudate or exudate), as well as the presence of adhesions and loculations. Consolidated lung, pulmonary abscesses and pulmonary masses may also be imaged if these lesions are adjacent to the pleural surface. Ultrasound waves are not transmitted through aerated lung, so the technique is of little value in airway disease or focal lung diseases where the diseased area lies deep to the lung surface. The ribs can also be imaged by ultrasound.

Either linear array, curvilinear or sector scanners can be used. Superficial structures may be examined using a high-frequency probe (7.5–10 MHz), but for deeper structures (>15 cm) a lower frequency probe should be used. The intrathoracic structures are imaged by placing the probe in the intercostal spaces and scanning both sides of the chest in a systematic fashion. The normal inflated lung has a highly echogenic surface. The reverberation artefact caused by the echogenic interface of pleura and aerated lung obscures the visualization of deeper structures. The pleural surfaces glide smoothly against each other as the lungs inflate and deflate. The two pleural surfaces normally appear as one echogenic line. Ribs are echogenic at their surface, smoothly marginated, convexly curved and cast a shadow. In the caudal thorax, the diaphragm may be visualized as a thin, smooth, echogenic line between the aerated lung and the cranial border of the liver.

Commonly identified abnormalities include:

- Pleural effusion (Figure 6.9) – separation of the parietal and visceral pleura by a band of hypoechoic material (fluid) deep to the intercostal muscles and superficial to the lungs. The echogenicity of the fluid varies depending on its nature (anechoic fluid represents fluid of low cellularity/protein content, whereas echogenic fluid indicates fluid with high cellularity/protein concentration). In the standing horse, free fluid will gravitate to the ventral thorax.
- Pleuritis – a roughened texture of the pleural surface causes narrow streaks of reverberation artifacts, commonly referred to as 'comet tails' (Figure 6.10). Comet tails can sometimes be identified in normal lungs, especially ventrally at the end of expiration. In chronic pleuritis, adhesions between the visceral and parietal pleura may restrict the normal gliding motion.
- Pleural effusion with fibrin/fibrin tags – linear echogenic structures that extend from the pleural surface into a region of effusion. These bands may float or wave within the pleural fluid.
- The pericardiodiaphragmatic ligament can be imaged in the ventral thorax in horses with pleural effusion (Figure 6.9). This linear soft-tissue structure is a normal structure that should not be mistaken as a fibrin tag. It is attached at either end and will float or wave during the respiratory cycle.
- Atelectasis and consolidation – consolidated lung appears similar to liver ('hepatized') with tubular, hypoechoic branching vascular structures.

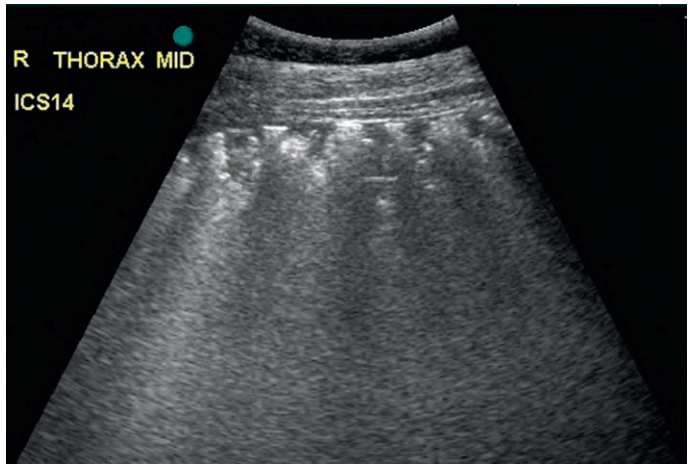


Figure 6.10 Ultrasonogram showing irregular visceral pleural surface due to consolidation and 'comet tails' passing into the lung tissue.

Figure 6.11 Thoracocentesis and drainage of a pleural effusion.

- Pulmonary mass – if a mass contacts the pleura it will be visible ultrasonographically, but not if it lies deep to aerated lung.
- Pnemothorax – this will cause a reverberation artefact without the normal gliding appearance of the pleura.

Thoracocentesis

Thoracocentesis allows the collection of samples of pleural fluid for analysis (including cytology and bacteriology); it can also be used in the treatment of pleuritis by allowing the drainage of large amounts of fluid. The precise site at which the chest is entered will vary depending on the amount of effusion; ultrasound guidance can be helpful. The usual site is either the right side at the 7th intercostal space, or the left side at the 7th or 8th spaces in the ventral third of the chest. Care must be taken to avoid the lateral thoracic vein, the liver, and the heart. The area is clipped and local anaesthetic infiltrated under the skin and into the intercostal muscles down to the pleura. A stab incision in the skin allows the introduction of a cannula (such as IV cannula, teat cannula or metal bitch urinary catheter) which is pushed into the pleural cavity along the cranial border of the rib (Figure 6.11). Fluid will normally drain by gravity, but care must be taken to avoid introduction of air into the cavity. Normal horses have a small quantity of clear, watery and pale straw-coloured fluid. The nucleated cell count of the fluid is low ($<4 \times 10^9$/L) with approximately 70% neutrophils, 20% large mononuclear cells, and small numbers of lymphocytes and eosinophils. The total protein content is normally less than 30 g/L. Effusions are classified as transudates, modified transudates, exudates or chylous.

- Transudates have normal characteristics and may be found in some early neoplastic diseases, congestive heart failure, hypoproteinaemia, etc.
- Modified transudates are similar but with added cells (e.g. mesothelial cells, neutrophils or neoplastic cells) or protein. They are usually associated with neoplasia.
- Exudates have high cell counts ($>10 \times 10^9$/L) and high protein concentrations (>30 g/L). The majority of the cells are neutrophils. Exudates are usually thick and cloudy, and are seen in pleuritis/pleuropneumonia, and sometimes with neoplasia.
- Chylous effusions are rare; they are milky white in appearance, and contain large numbers of lymphocytes.

Pleuroscopy (thoracoscopy)

Direct endoscopic examination of the pleural cavity is occasionally helpful in the evaluation of selected cases of pleuropneumonia, thoracic abscesses, pericarditis and thoracic neoplasia. It may also be helpful in aiding biopsy of pulmonary and pleural masses. Either rigid endoscopes (such as arthroscopes or laparoscopes) or flexible endoscopes may be used.

Haematology

Haematological alterations in lower respiratory tract diseases are often minimal or non-specific. Hyperfibrinogenaemia, hyperglobulinaemia, hypoalbuminaemia and mild anaemia may be observed in infectious, inflammatory and neoplastic diseases.

Other diagnostic tests

Other diagnostic techniques that may be usefully employed in some situations include lung biopsy, pulmonary function tests (such as intrapleural pressure measurement) and arterial blood gas analysis.

6.2 DIAGNOSTIC FEATURES OF THE COMMON LOWER RESPIRATORY TRACT DISEASES OF THE ADULT HORSE

See Table 6.1.

6.3 RECURRENT AIRWAY OBSTRUCTION (RAO)

RAO is also known as chronic obstructive pulmonary disease (COPD), chronic small airway disease (SAD), chronic pulmonary disease (CPD), broken wind, heaves, chronic alveolar emphysema.

This is a common condition characterized by laboured breathing that results from obstruction of small airways (bronchioles). The disease is reversible by dust control in the environment or by the use of bronchodilators. However, in the very late stages, some horses may develop irreversible damage including emphysema, and rarely cor pulmonale. The term inflammatory airway disease (IAD) is reserved for the condition in which there is lower airway inflammation with no clinical evidence of laboured breathing. RAO is seen in horses that are stabled and/or fed hay; a similar condition can occur is some horses that are pastured, and this syndrome has been called summer-pasture-associated obstructive pulmonary disease (SPAOPD).

RAO occurs in all breeds of horses, ponies and donkeys. The incidence tends to increase with age – usually horses older than 4 years are affected. In performance horses, low-grade RAO may cause few overt clinical signs other than *exercise intolerance* and *poor performance*. There is also a possible association with exercise-induced pulmonary haemorrhage (EIPH).

Aetiology

RAO is strongly associated with stabling and feeding hay. In most cases, a *pulmonary hypersensitivity* associated with inhalation of organic dusts, primarily *hay and straw dusts*, occurs; this results in inflammation of the small airways. Hypersensitivity to fungal and thermophilic actinomycete spores (major component of hay and straw dust, especially from 'heated' bales) is involved in most cases. Spores of *Faenia rectivirgula* (formerly known as *Micropolyspora faeni*), *Aspergillus fumigatus*, etc., have been implicated. Recently it has also been suggested that RAO may result from non-specific inflammatory responses to inhaled

Table 6.1 Diagnostic features of lower respiratory tract disease

Disease	Diagnostic features
RAO/SPAOPD/chronic lower airway inflammation	Endoscopy – lower airway exudate/bronchial inflammation Tracheal aspirate – neutrophilia BAL – neutrophilia
EIPH	Endoscopy – blood in airways Tracheal aspirate/BAL – red blood cells and haemosiderophages Radiography – opacity in dorsocaudal lung (advanced cases)
Lungworm	Endoscopy – larvae in bronchial tree Tracheal aspirate – eosinophilia/larvae BAL – eosinophilia
Neoplasia	Endoscopy – usually normal; rarely endobronchial mass Tracheal aspirate/BAL – usually normal; rarely neoplastic cells Radiography – pulmonary mass/pleural effusion Ultrasound – pleural effusion/mass Thoracocentesis – cytology may show neoplastic cells Biopsy – diagnostic Pleuroscopy – may show pleural mass
Pleuritis/pleuropneumonia	Endoscopy – lower airway exudate Tracheal aspirate – neutrophilia; Gram stain/culture BAL – may be normal or neutrophilia Radiography – pulmonary consolidation/pleural effusion Ultrasound – pleural effusion Thoracocentesis – exudate; culture
Focal pneumonia/lung abscess	Endoscopy – lower airway exudate Tracheal aspirate – neutrophilia; Gram stain/culture BAL – may be normal Radiography – pulmonary consolidation Haematology – anaemia, hypoalbuminaemia, hyperfibrinogenaemia
Tracheal stenosis	Endoscopy – tracheal narrowing Radiography – tracheal narrowing
Tracheobronchial foreign body	Endoscopy – foreign body Tracheal aspirate – neutrophilia Radiography – may be focal pulmonary consolidation
Chronic interstitial lung disease	Endoscopy – normal Tracheal aspirate/BAL – neutrophilia/lymphocytosis Radiography – miliary pulmonary infiltrate Lung biopsy – diagnostic

pro-inflammatory agents, including moulds, endotoxin, particulates and noxious gases which are present in the breathing zone of stabled horses.

Pathogenesis

Inhaled fungal and actinomycete spores (and other possible allergens) deposit in the bronchioles where specific hypersensitivity reactions are initiated.

Horses with RAO exhibit a modified type 2 cytokine response characterized by the production of interleukin-4 (IL-4) and IL-13 mRNA, but not IL-5 mRNA in BAL cells. Interferon-gamma mRNA is also elevated, suggesting a mixed response. Non-antigen-specific inflammatory responses are also probably involved. Inhalation of endotoxins (that may be present in hay and straw dusts) may result in lower airway inflammation, although the concentrations of endotoxin in the air of most stables is relatively low. Moulds can also cause a non-specific inflammatory response (i.e. not due to an allergic mechanism). Noxious gases (such as ammonia, hydrogen sulphide and methane) may also cause airway inflammation, especially in horses with RAO that also have non-specific airway hyperresponsiveness.

The net results of these reactions are:

- Spasm of small airways (bronchospasm due to smooth muscle contraction).
- Mucus hypersecretion.
- Inflammatory bronchiolitis (Figure 6.12).

These reactions result in *small airway obstruction*; airflow is impeded, especially during expiration. Most of these reactions are reversible if exposure to the offending allergens/pollutants is eliminated. In long-standing cases, some structural changes to the bronchioles may occur, e.g. metaplasia of the epithelium, hyperplasia of mucus-secreting goblet cells. In severe cases, chronic airflow obstruction results in alveolar overinflation and eventually areas of emphysema. In rare cases, secondary infection by opportunist bacteria may occur.

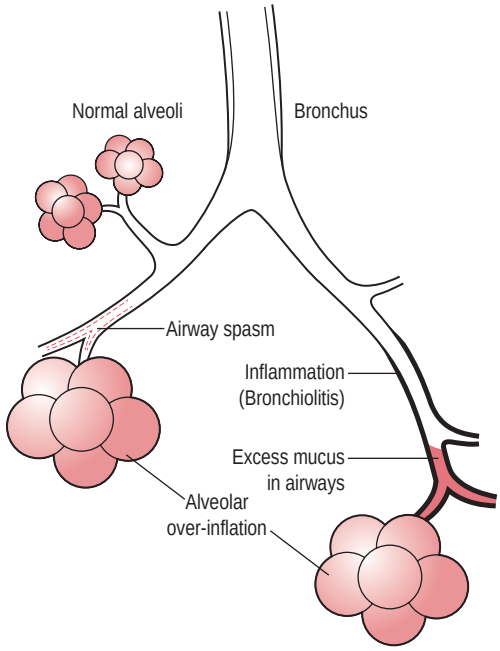


Figure 6.12 Pathological changes associated with RAO.

Epidemiology

The average age of onset of RAO is 9 years of age. Approximately 12% of mature horses have some degree of allergen-induced lower airway disease, and over 50% of horses that present for evaluation of respiratory disease are diagnosed with heaves. There is no breed or gender predilection, however, there does appear to be a possible heritable component to the aetiology of this condition.

Clinical signs

The onset of disease is frequently insidious. Alternatively, the disease may follow an acute respiratory infection.

The severity of the disease is extremely variable, ranging from subclinical to mild to severe. If left untreated, severity tends to worsen. The clinical signs may be continuous or intermittent/recurrent (seasonal). The signs abate and recur depending on the environmental challenge. RAO is associated with exposure to hay and straw, and is therefore most common when horses are stabled during the winter. However, some affected horses will demonstrate clinical disease all year round. When in remission, clinical signs may be completely absent.

Subclinical disease

This is most likely to be recognized in performance horses demonstrating *exercise intolerance* with few or no other overt clinical signs. It is associated with persistent low-grade airway inflammation and mucus accumulation, and affected horses may demonstrate occasional coughing (especially at the start of exercise).

Mild clinical disease

- There is usually a *chronic cough* that is occasional and sporadic; it is frequently noticed at the start of exercise or whilst feeding.
- Normal respiratory rate and normal respiratory character.
- There may be a slight serous/mucoid bilateral *nasal discharge* (especially after exercise).
- *Exercise intolerance* (performance horses).
- Appetite and general demeanor are normal.

Severe clinical disease

- Chronic cough that may occur at any time, but is most marked if the horse is exerted and during feeding.
- Increased respiratory effort with expiratory dyspnoea. Expiration becomes more markedly biphasic, with use of the abdominal muscles; an 'abdominal lift' occurs at the end of expiration resulting in a double expiratory effort. In longstanding cases, hypertrophy of the external oblique abdominal muscle leads to the development of a heave line. Expiratory airflow at the nostrils becomes biphasic.
- Resting respiratory rate may be normal or mildly elevated (16–20/min).
- There is usually a bilateral nasal discharge, but this is not invariably present and may be worse after exercise or when the head is lowered. In some horses, the discharge may appear to be predominantly unilateral. The nature of the discharge varies from serous to mucoid to mucopurulent.
- Exercise intolerance is marked in performance horses, and the recovery period after exercise is prolonged.
- Severe cases may develop weight-loss due to the difficulty of eating with dyspnoea, and the increased caloric needs to maintain the work of breathing necessary for adequate gas exchange.

Diagnosis

1. History and clinical signs – horses > 7 years of age, chronic cough >3 months' duration, normal appetite, no pyrexia, double expiratory effort, dilated nostrils.
2. Increased sensitivity of laryngeal cough reflex.
3. Auscultation:
   - fluid sounds over distal cervical trachea.
   - bronchial sounds audible over larger than normal area.
   - crackles.
   - wheezing.
   - crackling sounds audible to naked ear at nostrils.

   Subtle changes in lung sounds may be enhanced by the use of a re-breathing bag.
4. Endoscopy:
   - accumulation of variable quantities of mucopurulent discharge in the trachea (especially at the thoracic inlet) and bronchi.
   - congestion and thickening of the airway mucosa of the distal trachea, carina and mainstem bronchi.
   - 'blunting' of the carina due to swelling/oedema of the mucosa.
   - in severe cases, there may be observable collapse of the trachea and bronchi during expiration and coughing.
5. Tracheal aspirate: neutrophilia.
6. Bronchoalveolar lavage: variable cytological change – usually increase in neutrophils >25% (normal <5%).
7. Thoracic radiography: normal or mild interstitial reticular pattern.
8. Intradermal allergen tests/serum precipitins (mould species): non-specific reactions are common, and these tests are unreliable.
9. Inhalation challenge test: expose horse to mouldy hay/straw and observe for worsening of clinical signs, or changes in tracheal aspirate/BAL cytology.
10. Pulmonary function tests, e.g. intrapleural pressure measurement, arterial blood gas analysis: non-specific changes in severe clinical disease, but may be no changes in milder disease.
    - Intrapleural pressure: maximum ΔPpl – normal <6 mmHg.
    - Arterial oxygen: PaO_2 – normal 90–110 mmHg.

11. Response to change in management: manage from pasture with no access to hay or straw.
12. Response to drug therapy: corticosteroids (provided respiratory infection is unlikely) and bronchodilators.

Differential diagnosis

1. Inflammatory airway disease.
2. Bronchopneumonia.
3. Viral infections.
4. Neoplasia.
5. Lungworm.
6. Interstitial lung diseases.

Treatment

Environmental control is essential for the long-term management of horses with RAO. The reversal of clinical signs with strict environmental changes may start within a few days, but is likely to take 4–6 weeks to be fully effective. Drug therapy may be required in severely affected horses and in order to achieve short-term improvement, but they are unlikely to be beneficial in the long term if the horse remains in the allergen challenged environment.

To relieve clinical signs

1. Environmental control ('dust-free' management)
   - Manage from pasture with no access to hay or straw.
   - Minimize dust levels in stable:
     - Bedding: shavings/shredded paper/peat/synthetic bedding/rubber mats/sand, etc.
     - Maintain clean, dry bed (avoid deep litter).
     - Ventilation: ensure adequate ventilation rates.
     - Ensure horse is in a separate airspace (or else all horses in the same airspace must be managed under similar dust-free conditions).
     - Avoid overhead lofts.
     - Keep muck heap as far away from the stable as possible.
     - Horse should be out of the stable when it is mucked out (and for 30 minutes afterwards).
     - Feeding: If possible, feed an alternative to hay, e.g. 'complete diet' nuts, silage, vacuum-packed hays.
       If hay is to be fed, it should be good quality, and steamed or thoroughly soaked prior to feeding. Soaking may decrease the nutritional content of the hay. Soaked hay should be fed in small quantities so it doesn't dry out before it is consumed.
       Consider use of dust extractor in the box.
2. Corticosteroids. (see Table 6.2). Corticosteroids are the most potent anti-inflammatory agents available for the treatment of RAO. They reduce inflammation, decrease smooth muscle contraction (by inhibiting inflammatory mediators), potentiate the bronchodilatory effects of catecholamines, and reduce mucus production. They are usually for short-term use only, and can be administered orally, systemically or by inhalation.

Systemic and oral corticosteroid therapy

1. Systemic corticosteroid preparations are inexpensive and easy to administer, however, therapeutic drug concentrations are capable of inducing adverse effects in horses, and conservative dosing regimens are recommended.
2. Dexamethasone improves the clinical signs of RAO within hours of administration and the maximal response is obtained within a week. It is the most commonly used corticosteroid in the treatment of RAO. After cessation of treatment, some therapeutic benefit can be detected for up to a week later. Administration of dexamethasone produces marked suppression of endogenous cortisol production, which persists approximately 3 days after discontinuation of drug.
3. Triamcinolone acetonide relieves airway obstruction for up to four weeks, but being a long-acting corticosteroid it is more likely to cause side effects such as laminitis. Adrenal suppression resulting from a single dose persists for approximately 5 weeks. For this reason it is only recommended in severe cases.
4. Oral prednisolone is frequently used as an anti-inflammatory agent in horses because of its ease of administration, minimal cost, and reduced risk of laminitis (perceived). However, its efficacy in controlling RAO is uncertain.

Table 6.2 Drugs used to treat recurrent airway obstruction (RAO) in horses

Drug	Dosage
Corticosteroids	
Dexamethasone	0.04–0.1 mg/kg q 24 hours iv (Some clinicians keep horses on dexamethasone long-term, e.g. for 5–6 months, with no apparent side-effects when dosed every 48–72 hrs.)
Triamcinolone acetonide	20–40 mg im
Beclomethasone diproprionate	3500 µg q 12 hours in MDI
Fluticasone propionate	2000 µg q 12 hours in MDI
Bronchdilators	
Clenbuterol	0.8–3.2 µg/kg per os q 12 hours 0.8 µg/kg iv
Fenoterol	1–2 mg/kg q 1 hour in MDI
Albuterol	0.8–2.0 µg/kg q 1 hour in MDI
Pirbuterol	1.3 µg/kg q 7 hours in MDI
Salmeterol	0.5–1.0 µg/kg q 6 hours in MDI
Ipratropium bromide	0.4–1.0 µg/kg q 6 hours in MDI

Inhaled corticosteroids

1. Inhalation therapy allows the maximal concentration of the drug at the effector sites and reduces the total body dose, minimizing the inpact on other body systems. Inhaled corticosteroids are therefore preferred when prolonged therapy is required. Inhaled drugs can be administered using metered dose inhalers (MDI) designed for use in human asthmatic patients. A number of inhalation administration devices are available for use in horses. There are 3 aerosolized corticosteroid preparations available in MDI formulation for administration to horses via the Equine AeroMask™ or Equine Haler™: beclomethasone dipropionate, fluticasone propionate, and flunisolide. The relative potency of these surface-active corticosteroids is fluticasone > beclomethasone > flunisolide = triamcinolone.
2. Beclomethasone administered twice a day reduces pulmonary inflammation, improves parameters of pulmonary function, and improves ventilation of horses with RAO. Clinical signs and pulmonary function begin to improve within 24 hours.
3. Fluticasone propionate is a newer, potent corticosteroid that is effective in treating RAO. It is administered twice a day. Due to

its low oral bioavailability (<2%) and extensive first-pass metabolism (99%), fluticasone has the least potential for adverse systemic effects. It is highly lipophilic, and consequently, has the longest pulmonary residence time. However, it is expensive.

4. Bronchodilators (see Table 6.2). Bronchodilators relieve obstruction caused by airway smooth muscle contraction. They have rapid, but short-lived action, and only offer symptomatic relief of RAO. They are typically combined with environmental control and corticosteroid therapy. In horses with significant airway obstruction, bronchodilator therapy can be used as rescue therapy and will aid the delivery of inhaled corticosteroids to the small airways.
5. Beta$_2$ agonists and parasympatholytic medications are the most popular and effective bronchodilators for use in horses with RAO. Aminophylline and theophylline (phosphodiesterase inhibitors) are alternative systemic bronchodilators for horses with airway obstruction, however, the therapeutic index is relatively narrow and the magnitude of bronchodilation is less than that of the β_2 agonists. The phosphodiesterase inhibitors delay fatigue of the muscles of respiration, and may be valuable in horses with severe airway obstruction with impending respiratory failure due to fatigue.
6. Aerosolized albuterol sulfate (beta$_2$-agonist) improves pulmonary function by 70% within 5 minutes of administration. Unfortunately, the beneficial effect lasts for only 1–3 hours, which necessitates addition of a long-acting bronchodilator for horses with severe airway obstruction. Despite the short duration of effect, albuterol provides rescue therapy for horses during a severe episode of airway obstruction. Albuterol can be administered every 15 minutes for up to 2 hours to sequentially dilate small airways in horses with extreme bronchoconstriction. In addition, administration of albuterol improves the pulmonary distribution of aerosolized medications, and speeds mucociliary clearance. Terbutaline sulphate is another β_2-adrenoceptor agonist that may be effective when administered by nebulization (0.02 mg/kg in 4 mL saline), but has negligible oral bioavailability. Fenterol, pirbuterol and salmeterol may also be administered by inhalation.
7. Clenbuterol provides long-acting, β_2 stimulated bronchodilation for horses with moderate to severe RAO. Clenbuterol administration produces moderate improvement in objective parameters of pulmonary function, which may be difficult to appreciate or detect by clinical observation. The label recommendations outline an incremental dosage schedule based on the response to therapy. In addition to bronchodilator activity, clenbuterol is a mucokinetic agent which acts through increased ciliary beat frequency to speed mucociliary clearance. It can cause side effects (sweating and tachycardia) especially when administered intravenously or at higher oral dose rates.
8. Down-regulation of β_2 receptors has been documented in horses after 12 days of administration of clenbuterol. Administration of corticosteroids accelerates recovery from down-regulation in horses treated with clenbuterol alone and prevents clenbuterol-induced desensitization when administered concurrently.
9. Atropine provides rapid and powerful bronchodilation and can serve as a rescue medication in horses during a severe crises of airway obstruction; however, the abbreviated duration of action (0.5–2.0 hours) and adverse effects of systemic administration (ileus, CNS toxicity, tachycardia, increased viscosity of mucus secretion, impaired mucociliary clearance) limit its use to a single rescue dose for horses with severe airway obstruction. Ipratropium bromide is a synthetic, anticholinergic compound that is administered via inhalation. Ipratropium produces bronchodilation, inhibits cough and protects against bronchoconstrictive stimuli. Ipratropium improves pulmonary function by 50% within 30 minutes and the duration of effect is approximately 4–6 hours in severely affected horses. Due to its quaternary ammonium structure, absorption of ipratropium from the respiratory and gastrointestinal tract is negligible. Consequently, ipratropium produces minimal systemic effects, and ipratropium does not inhibit gastrointestinal motility, dry respiratory secretions, nor inhibit mucociliary clearance.
10. Mucolytics, expectorants and mucokinetic agents. Mucolytics are drugs that loosen secretions and decrease their viscosity. Expectorants increase the volume of secretions. Mucokinetic agent implies a drug that aids the clearance of respiratory secretions, and this is the preferred term. There is little clinical evidence to demonstrate the efficacy of mucokinetic agents in treating RAO, but they are commonly used. Drugs that can be used for this effect include clenbuterol, oral iodides, nebulized acetylcysteine and dembrexine. Increased airway hydration using intravenous saline (20 L/day) or saline nebulization has also been proposed to reduce viscosity and facilitate clearance of exudate from the lower respiratory tract.
11. Antioxidants. Oxidative stress may contribute to airway inflammation in RAO. For this reason dietary antioxidant supplements, such as vitamin C have been recommended.

To prevent recurrence of disease

1. Maintain strict 'dust-free' conditions.
2. Sodium cromoglycate. This drug acts by stabilizing mast cells and other inflammatory cells. It is administered by nebulization (80 mg/horse every 24 hours). The horse should be free from signs when the drug is given. The efficacy of the drug is questionable.

Prognosis

Lung changes of RAO are mostly reversible until structural damage (emphysema) occurs. Most cases have a favourable prognosis provided that exposure to allergens can be kept to a minimum.

Prevention

Maintain good stable hygiene, especially during the course of any infectious respiratory disease.

6.4 ACUTE OBSTRUCTIVE PULMONARY DISEASE

Acute airway obstruction may occur as an acute exacerbation of RAO or SPAOPD of similar aetiology and pathogenesis.

Clinical signs

Usually seen within 20 to 30 minutes of stabling:

- Marked dyspnoea – mainly expiratory with forced abdominal efforts.
- Tachypnoea – up to 40/min.
- Sweating.
- Anxious facial expression.
- Dilated nostrils.
- Protrusion of the anus with each expiratory effort.
- Paroxysmal coughing.
- Congestion of the mucous membranes.
- Tachycardia – up to 80–90/min.
- Normal to mildly elevated rectal temperature.

Diagnosis

1. Clinical signs.
2. Auscultation:
   - harsh lung sounds with widespread crackles and wheezing.
   - crackles audible at the nostril to the naked ear.

Treatment

- Turn out into fresh air immediately.
- Bronchodllators, e.g. clenbuterol (preferably administered IV).
- Corticosteroids, e.g. dexamethasone or betamethasone (preferably administered IV).
- Consider prophylactic antibiotic cover (especially if corticosteroids are used).
- In extreme cases, atropine (up to 5 mg IV) will provide effective and fast bronchodilation. (NB. the use of atropine in horses can result in ileus and abdominal pain.)

6.5 SUMMER PASTURE-ASSOCIATED OBSTRUCTIVE PULMONARY DISEASE (SPAOPD)

The clinical signs and pathophysiology of SPAOPD are similar to RAO, but it occurs in grazing horses at grass in the summer. It occurs sporadically thoughout the Europe and USA, but is especially common in southeastern USA.

Aetiology

The aetiology is uncertain, but probably involves a pulmonary hypersensitivity to environmental allergens including pollens. Anectdotal reports in the UK suggest an association with oilseed rape pollen, but this has not been confirmed by immunological methods or challenge tests.

Clinical signs

Signs are similar to RAO: expiratory dyspnoea and exercise intolerance. Coughing is not usually as marked as with RAO. Sometimes there is anorexia and weight loss.

Treatment

Drug therapy is as for RAO. In addition, the horse should be stabled and fed a diet of high-quality hay. Some horses with SPAOPD will also be allergic to indoor allergens, including mouldy hay and straw.

6.6 INFLAMMATORY AIRWAY DISEASE (IAD)

Lower airway inflammation associated with mild clinical signs, but often causing exercise intolerance and poor performance in athletic horses (especially racehorses). The terms inflammatory airway disease (IAD) and small airway inflammatory disease (SAID) have been used to describe this syndrome. Some cases follow an initial viral infection ('chronic post-viral coughing'). Initial viral infection may be subclinical (especially equine herpesvirus (EHV) 1 and 4 infections).

Aetiology and pathogenesis

The aetiology may vary from case to case, and IAD probably represents the common manifestation of a number of different aetiological and pathological pathways. One or more of the following may be involved in individual cases:

- Chronic bacterial bronchitis/bronchiolitis, e.g. *Streptococcus zooepidemicus, S. pneumoniae, Pasteurella species, Bordetella bronchiseptica*.
- Mycoplasma infections – *Mycoplasma felis* and *Mycoplasma equirhinis*.
- Recurrent or persistent viral infection.
- Non-infectious causes – airborne dusts and allergens, noxious gases.

The net result of these conditions is airway inflammation, mucus cell hyperplasia and airway hyperresponsiveness. In some cases a hypersensitivity reaction similar to RAO may be involved. There are probably a number of different sub-categories of IAD reflecting the different aetiologies, and the prevalence of the different sub-categories is likely to vary in different horse populations. The relationship between IAD and RAO is unclear. IAD usually affects younger horses than RAO and the clinical signs are generally less severe (exercise intolerance only).

Clinical signs

- Pyrexia.
- Chronic cough (especially during exercise or eating).
- Mucopurulent nasal discharge (variable).
- Exercise intolerance and poor performance in performance horses.
- Delayed recovery from exercise.
- Normal respiratory rate and character at rest.

Diagnosis

- Clinical signs.
- Endoscopy: increased quantities of tracheal mucus/exudate.
- Tracheal aspiration/BAL:
  - cytology usually shows increased numbers of neutrophils. In some cases there will be increased numbers of mast cells or eosinophils in BAL.
  - culture to identify potential pathogens.
- On the basis of airway cytology and microbiology, it is possible to classify cases into two groups:
  1. Bacterial IAD – associated with culture of a known bacterial or mycoplasmal pathogen in numbers greater than 10^2 colony-forming units/mL of tracheal aspirate, plus neutrophilic inflammation.
  2. Non-bacterial IAD: (i) mixed inflammation – high total nucleated cell count with neutrophilia (>5% of total BAL cells, or >20% of total tracheal aspirate cells), (ii) increased metachromatic cells (mast cells >2% of total BAL cells), (iii) eosinophilic inflammation (eosinophils >5% of total BAL cells).

Treatment

- 'Dust-free' management as for RAO.
- Bronchodilators may help to reduce minor degrees of small airway obstruction. Clenbuterol is the most commonly used agent. Aerosolized albuterol can be administered immediately pre-exercise to reduce coughing and bronchoconstriction during work.
- Antibiotics – indicated in cases with positive bacterial cultures of pathogens. Penicillin G and ceftiofur sodium have good efficacy against most bacterial respiratory pathogens. Oxytetracycline is also effective in many cases.
- Mucolytics.
- Antiinflammatory treatments for non-bacterial IAD: i. Mixed inflammatory reaction: low-dose natural human interferon-alpha reduces exudate in the respiratory tract and lowers total cell counts in BAL fluid. Interferon-alpha (IFN-alpha) is a

proximal mediator of immunomodulation and antiviral activity. ii. Eosinophilic inflammatory reaction: aerosolized corticosteroid administration is recommended for horses with eosinophilic BAL fluid. Eosinophilic pneumonitis responds slowly and incompletely to immunosuppressive therapy. iii. Metachromatic cell reaction: Once or twice daily administration of aerosolized corticosteroids provides safe and effective control of aiway inflammation in most performance horses.

6.7 EXERCISE-INDUCED PULMONARY HAEMORRHAGE (EIPH)

Exercise-induced pulmonary haemorrhage (EIPH) is a disorder of horses running at high speed (mainly racehorses) that results in haemorrhage into the lungs, usually in the dorsocaudal area of the diaphragmatic lobe. Almost all thoroughbred racehorses have haemosiderophages (macrophages containing haemosiderin) in BAL fluid samples, indicating that some degree of EIPH is almost universal in these animals. The disease occurs throughout the world.

Epistaxis (i.e. the presence of blood at the nostrils) occurs in up to 5% of racehorses after galloping. This blood usually originates from the lungs as a result of EIPH. However, in most racehorses EIPH occurs without any evidence of epistaxis. Endoscopic evidence of blood in the trachea is seen in up to 75% of horses examined after racing. Horses that have experienced one episode of EIPH are likely to have further episodes. The condition has also been identified (at a lower rate) in three-day event horses, polo ponies, barrel racing horses, and pulling horses, but has not been observed in horses performing low intensity endurance events.

Aetiology and pathogenesis

A number of different theories have been proposed:

- Stress failure of pulmonary capillaries. Pulmonary capillaries have extremely thin walls to allow rapid exchange of respiratory gases across them. Mechanical failure of the walls of the capillaries may occur when the pressure inside them rises to high levels during exercise.
- Haemorrhage occurs from areas of lung neovascularization, where there has been proliferation of the bronchial arterial system as a response to low-grade lower airway disease, e.g. RAO.
- Haemorrhage occurs in areas of lung subjected to high-pressure swings and stress during exercise.
- Haemorrhage occurs in the lung secondary to partial asphyxia due to an upper airway obstruction such as laryngeal hemiplegia.
- Haemostatic abnormalities.
- Changes in blood viscosity.

It is likely that the pathogenesis of EIPH in individual horses involves several processes, including pulmonary hypertension, low alveolar pressure, and changes in lung structure that all combine to cause stress failure of pulmonary capillaries. The characteristic location of lesions of EIPH is the caudodorsal lung fields. Regardless of the cause, rupture of pulmonary capillaries and subsequent haemorrhage into the airways causes inflammation and damage (fibrosis) to the interstitium in the affected area.

Clinical signs

Most horses are affected by EIPH with no obvious overt clinical signs. In a small number of cases, there may be epistaxis after racing. Occasionally, a massive fatal pulmonary haemorrhage may occur.

The effect of EIPH on racing performance is variable, and in most it is probably minimal. In some cases, there may be reduced exercise tolerance, sometimes to the point where the horse pulls up during a race. In these cases, the affected horse may show respiratory distress, with an anxious facial expression and some coughing. Repeated swallowing may be observed as the blood is cleared from the trachea into the pharynx.

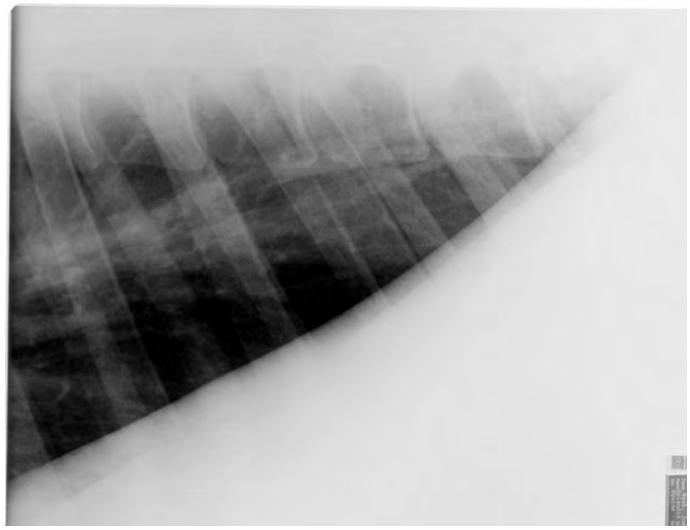


Figure 6.13 Lateral thoracic radiograph of the dorsocaudal lung of a horse with recent history of EIPH. There is a diffuse increase in radiopacity in the dorsocaudal tip of the lung fields.

Some horses may continue to bleed into the lung for several days or even weeks after racing. These horses tend to be dull and lethargic but non-pyrexic. Haematology may show a progressive anaemia.

Diagnosis

- Endoscopic examination of the trachea 30–120 minutes after racing or strenuous exercise. Blood can be seen in the large airways in variable amounts from a few specks to a continuous stream. The degree of observed blood can be graded from 0 (none) to 4 (multiple coalescing streams of blood covering >90% of the tracheal surface).
- Tracheal aspirate/BAL cytology: red blood cells or haemosiderophages may be seen using a specific stain for haemosiderin (Pearl's Prussian Blue).
- Radiography: opacity in dorsocaudal lung field (must be performed within 7 to 10 days of galloping to identify a recent haemorrhage) (Figure 6.13).

Treatment

Treatment of EIPH is mainly empirical. A large number of prophylactic treatments have been proposed, but their efficacies are largely unproven.

1. Any underlying small airway disease (RAO, viral infections, etc.) should be treated.
2. A dust-free environment should be available.
3. Horses that have suffered haemorrhage that is believed to have affected performance should be rested to allow healing of damaged capillaries and lung tissue.
4. Antibiotic cover may be indicated to prevent secondary infection of affected areas.

The following prophylactic treatments have been employed (use of these will be dictated by the relevant racing authorities):

- Frusemide (furosemide). The mechanism by which frusemide prevents or reduces EIPH is uncertain, but it will attenuate the increase in pulmonary artery and pulmonary capillary pressures that occur with exercise.

- Flair nasal strip. To reduce intrathoracic pressure associated with resistance created by unsupported soft-tissue structures of the rostral nasal passage.
- Oestrogens.
- Disodium cromoglycate.
- Clenbuterol.

6.8 LUNGWORM (*DICTYOCAULUS ARNFIELDI*) INFECTION

See also Chapter 19.

Donkeys are the natural host of *D. arnfieldi*, and commonly carry asymptomatic infections. Horses usually become infected by grazing pasture contaminated by donkeys. Eggs passed out in donkey faeces can develop rapidly into infective larvae which can survive 6–7 weeks on pasture under suitable conditions (damp/shade). The larvae cannot overwinter.

Horses become infected by ingesting larvae, which migrate to the lungs. In most horses, larval development becomes arrested and egg-laying adults do not arise. The disease is usually recognized in late summer/autumn.

Clinical signs

Chronic coughing (often paroxysmal in nature) is the most prominent clinical sign of lungworm infection. Many horses will have an obstructive breathing pattern with a prolonged expiratory phase of respiration. Clinical signs may be indistinguishable from heaves (RAO).

Diagnosis

- History and clinical signs.
- Faecal examination for presence of larvae (Baerman technique). This is usually negative since most infections are non-patent.
- Endoscopy may reveal larvae in the trachea or mainstem bronchi.
- Tracheal aspirate/BAL: pronounced eosinophilia. In some cases, larvae may be recovered by tracheal aspirates or BAL.
- Response to treatment.

Treatment

Ivermectin (0.2 mg/kg) by mouth. There may be a temporary worsening of clinical signs for a few days after treatment, followed by a rapid improvement.

6.9 *PARASCARIS EQUORUM* INFECTION

See also Chapter 19.

This is an unusual cause of coughing in foals, weanlings and yearlings, and is associated with the migration of larvae through the lungs, resulting in mild eosinophilic pneumonia.

Clinical signs

Signs are cough, serous nasal discharge, inappetence and weight loss.

Diagnosis

- Clinical signs and history. Absence of fever may help to distinguish *P. equorum* infection from bacterial infections.
- Tracheal aspirate/BAL: eosinophilia.

Treatment

Ivermectin, moxidectin or fenbendazole by mouth. Use of moxidectin is not recommended for foals older than 6 months of age.

6.10 INHALED FOREIGN BODIES

This is an uncommon cause of chronic coughing. The foreign body may lodge in the pharynx/larynx or in the tracheobronchial tree.

Various types of foreign bodies may be involved. In the lower airways they usually involve twigs or brambles (thorns act as barbs which prevent the bramble from being coughed up).

Bronchial foreign bodies often enter the right mainstem bronchus, and may cause secondary pneumonia or lung abscess, or may penetrate the pleura leading to pleuritis.

Clinical signs

- Sudden onset of coughing – often paroxysmal.
- Pharyngeal foreign bodies may cause dysphagia and painful swallowing (differentiate from oesophageal obstruction).
- Tracheobronchial foreign bodies result in halitosis (malodorous breath) and purulent nasal discharge.

Diagnosis

- Endoscopy.
- Radiography.

Treatment

Surgical removal under general anaesthesia, or snare, or standing (under sedation) using an endoscopic biopsy instrument.

6.11 PULMONARY/PLEURAL/MEDIASTINAL NEOPLASIA

Lower airway neoplasia is uncommon. Diagnosis depends on ultrasonography, radiography, biopsy, pleural fluid cytology.

Primary lung tumours

These are rare, e.g. myoblastoma (granular cell tumour), bronchial and alveolar carcinomas, haemangiosarcoma.

Lung metastases

Metastases are uncommon, e.g. lymphosarcoma, various carcinomas (e.g. renal), melanosarcoma.

Clinical features of lung tumours

Lung tumours usually present as chronic progressive dyspnoea with weight loss. There may be a chronic cough and intermittent/persistent pyrexia. There may be an associated pleural effusion (with worsening dyspnoea). Clinical signs associated with lung metastases often relate more to the primary site of tumour formation than to thoracic involvement.

Primary pleural tumours

These are generally mesotheliomas.

Pleural metastases

Examples are gastric squamous cell carcinoma, other carcinomas.

Pleural tumours usually present as dyspnoea (may have apparent sudden onset) with pleural effusion and weight loss.

Pleural fluid cytology may be diagnostic in pleural neoplasia and pulmonary neoplasia with pleural effusion (neoplastic cells).

Mediastinal tumours

Lymphoma (lymphosarcoma) can affect horses of all ages. Usually multicentric, and commonly involves the mediastinal lymph nodes. These may become large enough to act as a space occupying lesion compressing the lungs, trachea, oesophagus and major blood vessels, resulting in dyspnoea, dysphagia and distension of the jugular veins.

A mass may be palpable at the base of the jugular groove (extension of neoplastic lymphoid tissue through the thoracic inlet and involvement of the deep caudal cervical lymph nodes) – this may be accessible to biopsy.

Many affected horses have pleural effusion and ventral thoracic oedema. Pleural fluid cytology may be diagnostic – large numbers of lymphoid cells and lymphoblasts showing variation in size and shape. (N.B. Normal pleural fluid cytology does not exclude lymphoma or other neoplasia.)

6.12 PLEUROPNEUMONIA

This is more common in the USA than in Europe.

The following may occur alone or in combination:

- Pneumonia.
- Pulmonary abscess.
- Pleuritis (pleurisy).

Aetiology and pathogenesis

1. Stress, e.g. long-distance travel ('transit fever'), surgery, strenuous exercise.
2. Secondary bacterial infection after viral respiratory disease.
3. Corticosteroid therapy or Cushing's disease.
4. Bacteraemia, e.g. strangles.
5. Inhalation pneumonia secondary to dysphagia:
   - oesophageal obstruction (choke).
   - pharyngeal paralysis.
   - structural abnormalities.
   - grass sickness.
   - iatrogenic.
   - inhaled foreign bodies.
6. Direct thoracic trauma and chest penetrations.
7. Pulmonary emboli and infarction, e.g. secondary to jugular phlebitis and thrombosis.

Long-distance transportation (>500 miles) is the most common predisposing factor, and is associated with prolonged head elevation, which prevents the postural drainage of respiratory secretions and facilitates the aspiration of upper respiratory tract bacteria into the lower airways. In addition, long-distance travel reduces pulmonary macrophage function, and alters systemic lymphocyte function.

Pathology

The pathology is variable. Usually there are discrete areas of pneumonia / abscesses. Occasionally the pneumonia is generalized and involves the entire lung. Pleuritis with pleural effusion is usually bilateral. In chronic cases, there may be extensive fibrin formation within the pleural cavity, with adhesion formation and pleural abscessation. Abscess/granulation tissue may localize cranial to the heart forming a *cranial thoracic mass*.

Three phases are recognized in the pathogenesis of pleuropneumonia:

1. Exudative phase – initial colonization of lung tissue (typically the ventral aspects of the cranial lung lobes and the cranial aspects of the caudal lung lobes). Extension of the inflammatory response to the visceral pleura results in the production of a protein-rich inflammatory exudate.
2. Fibrinopurulent phase – direct extension of bacterial infection into the pleural cavity results in the production of large volumes of septic fibrinopurulent exudate in the pleural cavity.
3. Organization phase – organization of fibrin into fibrous tissue and a thick 'pleural peel'.

Organisms

A variety of organisms may be involved: *Streptococcus* spp., *Staphylococcus* spp., *Klebsiella* spp., *Pasteurella* spp., *Escherichia coli*, *Bordetella bronchiseptica*, *Nocardia* spp., *Mycoplasma felis*.

Anaerobic bacteria are common, e.g. *Bacteroides* spp.

Clinical signs

- Initially fever and pain (reluctance to move; abduction of the elbows) must be differentiated from more common causes of acute pain, e.g. colic, laminitis, rhabdomyolysis.
- Later, recurrent/persistent fever, inappetence, tachypnoea and dyspnoea (inspiratory and expiratory), cough, weight loss, pectoral and sternal oedema – must be differentiated from thoracic neoplasia, interstitial lung diseases (tuberculosis, granulomatous pneumonia.).
- Pleurodynia (thoracic pain) causes anxious facial expression, stilted gait, reluctance to turn or to lie down, a soft suppressed cough, and expiratory grunting.
- Nasal discharge is variable – occasionally blood-tinged. May be malodorous breath with anaerobic infections or tissue necrosis. Large volume, malodorous bloody discharge carries a poor prognosis for survival.
- In cases with a cranial thoracic mass, there may be pointing of a foreleg, distension of jugular veins and tachycardia.

Diagnosis

May be difficult in the early stages.

1. Clinical signs.
2. Thoracic auscultation – may be normal in the early stages:
   - pleural friction sounds.
   - ventral dulling of lung sounds with heart sounds audible over a larger than normal area, indicative of pleural effusion.
   - localized areas of reduced lung sounds, indicative of pulmonary consolidation/pneumonia/abscessation.
   - adventitious lung sounds (crackling/wheezing/pleural friction rubs).
3. Thoracic percussion:
   - pain response by horse (especially in early stages of pleuritis).
   - reduced resonance ventrally, indicative of pleural effusion.
4. Radiography: to assess pulmonary parenchymal disease and mediastinal structures.
   - consolidated lung.
   - pleural effusion – fluid line.
5. Haematology – variable:
   - leucocytosis, neutrophilia and hyperfibrinogenaemia may occur, but haematology may be normal initially; in the acute stages there may be leucopenia.
6. Thoracocentesis – cytology and bacterial culture (including anaerobic culture).
7. Tracheal aspiration/BAL – cytology and culture.
8. Ultrasonography: to assess pleural space and peripheral pulmonary parenchyma.

- consolidated lung and abscesses (adjacent to the pleural surface only).
- assess presence, quantity and nature of pleural effusion.
- fibrin, adhesions, loculated fluid, cranial thoracic mass.

Treatment

- Appropriate systemically administered antibiotics (preferably identified by bacteriological cultures) for a prolonged period.
- Analgesics – non-steroidal anti-inflammatory medication.
- Supportive nursing care.
- Drainage of pleural fluid – continuous drainage via indwelling thoracic drain with one-way valve, or repeated drainage by thoracocentesis.
- Pleural lavage may be useful in selected cases to aid removal of tenacious exudate, etc.
- Intrapleural antibiotics, e.g. metronidazole, amikacin.
- Drainage of localized pleural/cranial thoracic abscess (using ultrasound guidance).
- Thoracotomy – standing thoracotomy is sometimes needed for horses with large quantities of thickened, organized fibrinopurulent debris or empyema that is walled off in pockets, especially in cases where such material cannot be removed by conventional pleural drainage or lavage. It is important to establish that the patient can breathe comfortably with a unilateral pneumothorax, prior to performing a thoracotomy.

Complications

Complications can include cranial thoracic masses, bronchopleural fistulae, pericarditis, laminitis, pleural adhesions, and pneumothorax.

6.13 *RHODOCOCCUS EQUI* PNEUMONIA

Rhodococcus equi is a cause of pneumonia of foals, especially in the 1–4 month age group. *Rhodococcus equi* is a Gram-positive, soil saprophytic, facultative intracellular bacterium that proliferates in alveolar macrophages. The organism is found throughout the world. The pathogenicity of this bacterium may be dependent upon the presence of an 85–90-kilobase plasmid. The organism survives in the soil, and the disease tends to become endemic on certain farms, with regular recurrent outbreaks of disease. Mares can also be the source of *R. equi* for their foals, since the organism can survive and multiply in the intestinal tract; it can frequently be isolated from faeces. Infection may occur by inhalation or ingestion, and the prevalence of disease increases with dusty environments and dry weather.

Two clinical forms of *R. equi* pneumonia are recognized. The subacute form is characterized by a diffuse miliary pyogranulomatous pneumonia with a short clinical course usually resulting in death. The chronic form is characterized by solitary or multifocal pyogranuloma, and has a prolonged clinical disease of weeks to months.

Clinical signs

- *Subacute form*. Acute onset of fever, tachypnoea and dyspnoea. Coughing and nasal discharge are variable. Often die within a few days of onset of clinical signs. Post-mortem examination reveals lung abscesses and diffuse pneumonia.
- *Chronic form*. Fever, depression, lethargy, dyspnoea and tachypnoea, weight loss and unthriftiness. Coughing and nasal discharge are variable.
- *Intestinal form* is sometimes recognized in older foals. Weight loss, failure to thrive, diarrhoea, fever and recurrent colic associated with granulomatous enterocolitis/typhlitis. Abscessation of the mesenteric lymph nodes and peritonitis may occur.

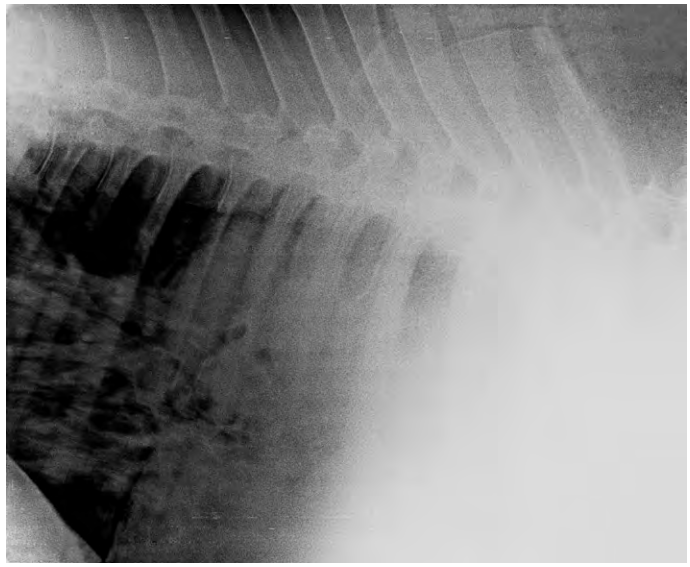


Figure 6.14 Lateral thoracic radiograph of a foal affected by *Rhodococcus equi* pneumonia showing multiple nodular pulmonary opacities.

- Polysynovitis. This may occur as a result of an immune-complex mediated disease. Commonly affected joints include the tarsocrural, femoropatellar and carpal joints. Signs include joint effusion with or without lameness.
- Dissemination of the infection can result in osteomyelitis or septic arthritis. Osteomyelitis of the vertebrae can cause ataxia.

Diagnosis

- Clinical signs. History of disease on the premises.
- Haematology: neutrophilia, hyperfibrinogenaemia.
- Transtracheal aspirate culture (negative in 30–60% of cases). Polymerase chain reaction (PCR) may be more sensitive than culture. Cytology shows many degenerate neutrophils and Gram-positive, intracellular coccobacilli.
- Thoracic radiography: perihilar nodular lung lesions (may be cavitary) and lymphadenopathy. Some cases have pleural effusion (Figure 6.14).
- Thoracic ultrasonography can be used as a rapid and sensitive way of detecting peripheral pulmonary consolidation and abscessation (Figure 6.15).

Treatment

The combination of erythromycin and rifampin has historically been the treatment of choice for *R. equi* infections in foals. These antimicrobials may be bacteriostatic, but their activity is synergistic, and the combination has markedly improved survival of foals with *R. equi* pneumonia. The duration of antimicrobial therapy using this combination of medications typically ranges from 4 to 9 weeks. Adverse reactions are relatively common in foals treated with the erythromycin/rifampin combination. Diarrhoea, idiosyncratic hyperthermia, tachypnoea, anorexia, bruxism, and salivation can occur with erythromycin administration, and antimicrobial resistance of *R. equi* to erythromycin/rifampin has been reported.

Azithromycin is a newer generation macrolide with greater bioavailability than erythromycin, and achieves higher drug concentrations. Azithromycin is administered orally, once daily until clinical signs stabilize, followed by every other day until resolution of disease. The principle advantage of azithromycin-rifampin over erythromycin-rifampin is the convenience of once-per-day dosing.

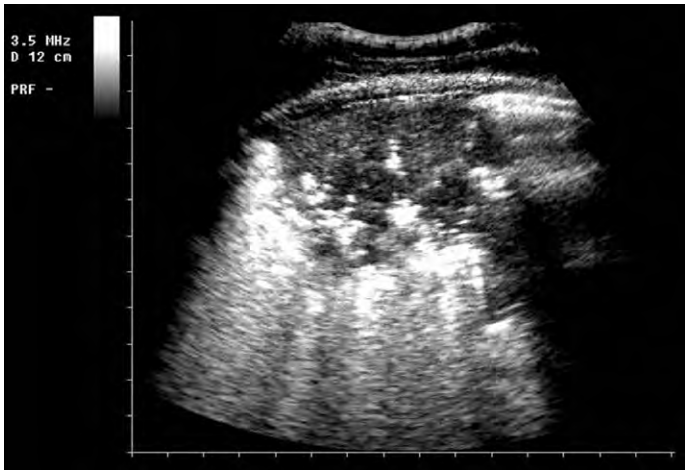


Figure 6.15 Thoracic ultrasonogram of a foal with *Rhodococcus equi* pneumonia showing irregular consolidation of the lung surface and deeper tissue.

Clarithromycin is an alternative macrolide with the most favourable minimum inhibitory concentration against *R. equi* isolates obtained from pneumonic foals. In foals with *R. equi* pneumonia, the combination of clarithromycin and rifampin is superior to erythromycin-rifampin and azithromycin-rifampin, particularly in foals with severe disease. Foals treated with clarithromycin-rifampin have improved short-term and long-term survival rates and fewer febrile days than foals treated with erythromycin-rifampin or azithromycin-rifampin. Life-threatening, antibiotic-induced enterocolitis, due to *Clostridium difficile,* has been observed in the dams of nursing foals treated with erythromycin, azithromicin, and clarithromycin. The mechanism is suspected to result from coprophagic behaviour by the mare, which leads to ingestion of sufficient active macrolide to perturb the intestinal flora of the mare. Dams of foals being treated for *R. equi* should be closely observed for signs of fever, toxemia, anorexia, depression, and diarrhoea during the treatment period.

In addition to antibiotic therapy, general nursing care, excellent nutrition, and provision of adequate ventilation are important to recovery. As a result of the stress associated with respiratory difficulty and repeated treatments, antiulcer medication may be indicated.

Prognosis

The prognosis is fair. Approximately 80% recover with erythromycin and rifampicin treatment, with good prognosis for athletic soundness.

Prevention

- Transfusion of hyperimmune plasma (1–2 L administered to the foal in the first few days of life and again during the third week of life).
- Screening of at-risk foals via weekly ultrasonographic examinations or bi-weekly CBC and fibrinogen measurements allow for early detection and intervention.
- Vaccination of dam – questionable efficacy.
- Foaling at pasture, decreased crowding.

6.14 INTERSTITIAL PNEUMONIA/CHRONIC INTERSTITIAL INFLAMMATION

Chronic interstitial disease involving mycobacterial infections (especially mycobacteria of the *avium–intracellulare* group) are occasionally recognized. Other chronic interstitial pneumonias and inflammatory/fibrotic diseases of unknown aetiology also occur. Recently, a progressive fibrotic interstitial lung condition, termed equine multinodular pulmonary fibrosis, has been described; infection with Equine Herpesvirus-5 (EHV-5) appears to be involved with the pathogenesis of this disease. Non-mycobacterial granulomatous interstitial pneumonia may occur as part of a generalized granulomatous disease of unknown aetiology. Eosinophilic interstitial pneumonitis is also occasionally identified. These interstitial diseases can eventually result in *fibrosing alveolitis*. Environmental exposure to coal dust/silica, etc., can result in *pneumoconiosis*.

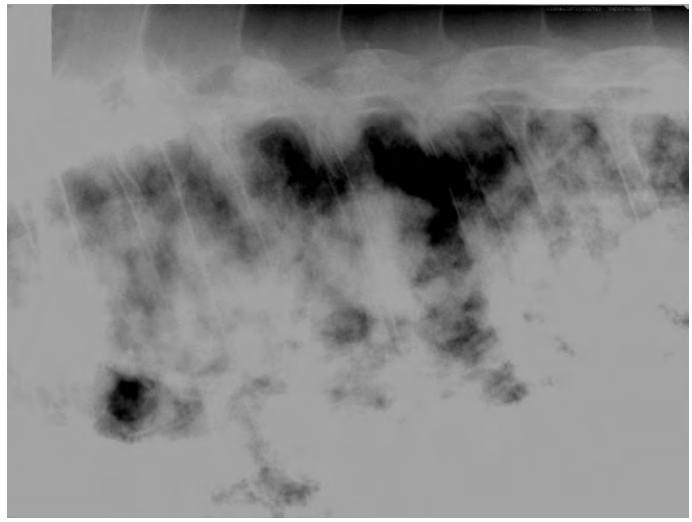


Figure 6.16 Lateral thoracic radiograph of an adult horse with interstitial pulmonary fibrosis showing a diffuse nodular radiopaque pulmonary infiltrate.

Clinical signs

- Chronic, progressive dyspnoea, with or without coughing.
- Weight loss.
- Persistent or recurrent pyrexia.

Diagnosis

- History and clinical signs. Lack of response to standard therapy for lower airway diseases.
- Auscultation – harsh lung sounds with variable wheezing and crackling.
- Leukocytosis and hyperfibrinogenaemia.
- Thoracic radiography – interstitial infiltrate (may appear nodular or miliary) (Figure 6.16).
- Bronchoalveolar lavage – variable cytology. Smear and culture for Mycobacteria. Particulate material within macrophages may be seen in cases of pneumoconiosis. Inclusion bodies in alveolar macrophages and positive results of PCR for EHV-5 may be demonstrated in cases of equine multinodular pulmonary fibrosis.
- Inclusion bodies in alveolar macrophages and positive results of PCR for EHV-5 may be demonstrated in cases of equine multinodular pulmonary fibrosis.

Treatment

There is no treatment. Mycobacterial infections are potential public health risks. Long-term antiinflammatory drugs or corticosteroids may be beneficial in chronic interstitial inflammatory diseases, especially if given early in the course of the disease prior to the development of pulmonary fibrosis. Acyclovir or valacyclovir may be helpful in early cases of EHV-5 infection (equine multinodular pulmonary fibrosis).

6.15 ACUTE INTERSTITIAL PNEUMONIA IN FOALS

See also Chapter 20.

Acute interstitial pneumonia is occasionally identified in foals (from a few days to several months of age) as a cause of severe respiratory distress. The underlying aetiology is often multifactorial (including bacterial, viral, fungal and toxic agents), and heat stress may be involved in some cases. The prognosis is generally poor, with a death rate of >50%; death is usually due to respiratory failure.

Clinical signs include:

- Acute onset tachypnoea.
- Dyspnoea with marked abdominal component to expiration.
- Cyanosis.
- Pyrexia.

Clinicopathological abnormalities typically include elevated plasma fibrinogen and neutrophilia. Arterial blood gas analysis shows hypoxia and hypercapnia. A diffuse interstitial or bronchointerstitial infiltrate is seen on thoracic radiography.

Treatment should include broad spectrum antibiotics, anti-inflammatory drugs, corticosteroids, bronchodilators (systemic and aerosolized) and supplemental oxygen therapy.

Post-mortem examination reveals lungs that are congested and heavy, and that fail to collapse. On histological examination, proteinaceous fluid, fibrin and cellular debris are found in the distal airways and alveoli. If a foal survives, it may suffer relapses through the first 12 months of life. After the first year, affected foals typically make a full recovery.

6.16 FUNGAL PNEUMONIA

See also Chapter 19.

Pathogenic fungi include *Coccidioides immitis, Histoplasma capsulatum* and *Cryptococcus neoformans*. Pulmonary infections by these fungi are usually only recognized in specific geographical locations.

Fungal pneumonia is relatively uncommon in horses and is typically associated with large colon disease with mucosal disruption and profound neutropenia (colitis, colon torsion). Opportunist pulmonary infections by other fungi may occur in immunocompromised horses. These include *Aspergillus* species, *Candida* species, *Mucor* species and *Rhizopus* species.

Diagnosis

- Tracheal aspirate – fungal elements identified by cytology. N.B. Fungal hyphae and spores are commonly found in tracheal aspirates from normal horses; to be significant, these elements must be present in very large numbers.
- Marked leukocytosis and hyperfibrinogenaemia.
- Thoracic radiographs – fluffy white appearance of coalescing nodules in the caudodorsal lung fields. (Figure 6.17)
- Lung biopsy is necessary for definitive diagnosis.

Treatment

Successful treatment is rarely documented, which may reflect rare antemortem diagnosis and/or poor response to therapy. Specific antifungal therapy for treatment of mycotic pneumonia is dependent on the isolate.

Amphotericin-B is an appropriate therapeutic choice for aspergillosis, but may be cost prohibitive for many owners. Side-effects include polyuria/polydypsia (4th week of treatment), intermittent fever (first two weeks), and lethargy (after every treatment). Urinalysis and serum biochemical profile should be obtained weekly to detect evidence of renal or hepatic dysfunction.

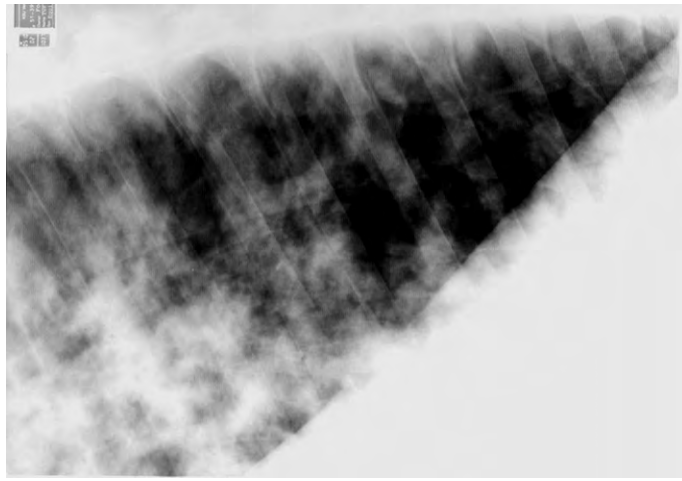


Figure 6.17 Lateral radiograph of a horse with fungal pneumonia showing coalescing radiopaque pulmonary nodules.

Ketoconazole and oral iodides are less expensive, but in general, are less likely to resolve fungal pneumonia. Successful treatment has been described with ketoconazole initially, followed by aerosolized enilconazole (1.2 mg/kg in saline via ultrasonic nebulization, BID) for long-term treatment. Itraconazole may be an appropriate antifungal therapy for horses, based on the sensitivity pattern of pulmonary isolates. However, bioavailability appears poor in the tablet form, but liquid formulation may be sufficient to provide therapeutic efficacy. Alternatively, the oral bioavailability of fluconazole is sufficient to maintain the plasma concentrations above the mean inhibitory concentration reported for fungal pathogens in horses.

6.17 *PNEUMOCYSTIS* PNEUMONIA

Pneumocystis carinii is a rare cause of pneumonia in foals. It is generally associated with immunocompromise and carries a poor prognosis. It should be suspected in foals with non-responsive pneumonia. It is a common cause of pneumonia and death in Arabian foals affected by combined immunodeficiency syndrome. It has also been associated with prior corticosteroid treatment and with *Rhodococcus equi* infection of foals.

The typical clinical presentation of pneumocystis pneumonia is acute onset of severe respiratory distress and cyanosis, often following a milder chronic bacterial pneumonia that had been unresponsive to treatment. Thoracic radiographs typically show a diffuse bronchointerstitial pattern. Diagnosis can be achieved by cytological examination of BAL fluid.

Treatment of pneumocystis pneumonia with trimethoprim/sulfamethoxazole has demonstrated variable success in foals. Dapsone, a sulfone antimicrobial, may be useful as an adjunctive treatment to traditional administration of trimethoprime-sulfamethoxazole.

6.18 PULMONARY OEDEMA

Pulmonary oedema is rarely recognized as a primary problem. It may be seen as a secondary feature or in association with a number of other diseases:

Figure 6.18 Pulmonary oedema – typical white frothy nasal discharge.

- Upper airway obstruction.
- Septicaemia/septic shock.
- Congestive heart failure.
- Ruptured chordae tendinae.
- Pneumonia.
- Overhydration.
- Smoke inhalation.
- Acute alveolitis.

Anaphylaxis may occur following injection or oral administration of specific medications. A similar reaction may arise following the ingestion of certain foods (especially proteins – soya and wheat origin) or inhalation of organic dusts. Immediate immune reactions usually result in subcutaneous oedema and skin plaques, but may cause pulmonary oedema. Accidental intravenous injection of procaine penicillin may precipitate pulmonary oedema, clinical signs of which are observed several hours after recovery from the acute neurologic reaction.

Clinical signs

The clinical signs will reflect the primary disease as well as the lung disease. Skin of affected horses is often cold and clammy. Horses are weak and ataxic, with a subnormal temperature. The pulse is weak and rapid. Respirations are rapid and shallow. Fluid (may be frothy and blood-tinged) discharges from the nose, and may drool from the mouth. In severe cases, there is marked dyspnoea, collapse and death (Figure 6.18).

Diagnosis

- Clinical signs and evidence of predisposing disease.
- Auscultation – fine crackles and sometimes wheezing.
- Thoracic radiographs – alveolization with air bronchograms.

Treatment

Treatment should be aimed at eliminating the underlying cause as well as treating the pulmonary oedema.

- Diuretics (frusemide IV or IM).
- Corticosteroids if allergic mechanism is involved.
- Broad-spectrum antibiotic cover, especially if corticosteroids are used.
- Non-steroidal anti-inflammatory medications.
- Nasal insufflation of oxygen.

6.19 DROWNING/NEAR DROWNING

Rare, but can occur in horses undertaking swimming exercise. Fresh water inhalation leads to atelectasis and hypoxaemia. Salt water inhalation may lead to severe pulmonary oedema and dehydration. Pneumonia may follow aspiration.

Treatment is as for pulmonary oedema. In addition, bronchodilators may be beneficial.

6.20 PULMONARY CONGESTION

Commonly occurs in recumbent horses (e.g. during general anaesthesia, or neuromuscular diseases – tetanus, botulism, etc.). This is rarely severe enough to compromise pulmonary function, but if prolonged may predispose to secondary infection.

Occasionally, severe pulmonary congestion may arise during strenuous exercise, especially in unfit horses. This may also involve a degree of EIPH and pulmonary oedema. The horse shows rapid, heaving respirations, with the head held down; haemorrhagic froth may appear at the mouth and nostrils.

Treatment

- Rest in quiet environment.
- Antibiotic cover.
- Diuretics.

6.21 HAEMOTHORAX

Uncommon. May occur when there is damage to either visceral or parietal pleural vessels:

- Trauma to chest wall and fractured ribs.
- Erosion of vessels by pulmonary abscess/neoplasm.
- Haemangiosarcoma involving pleura.
- Severe EIPH.
- Bleeding disorders.

The clinical signs resemble those of pleural effusion. Diagnosis is based on thoracocentesis. Treatment is aimed at treating the underlying cause.

6.22 PNEUMOTHORAX

This is uncommon and is usually related to trauma.

- Penetrating wounds to the thorax.
- Puncture or rupture of the trachea.
- Rupture of the oesophagus.
- Broncho-pleural fistula secondary to pleuropneumonia.

Open pneumothorax results when a wound allows air to enter and leave the pleural cavity. *Closed pneumothorax* involves trapping air within the chest. *Tension pneumothorax* occurs when a flap of tissue acts as a valve allowing air to enter but not leave the thorax.

Clinical signs

- Tachypnoea and dyspnoea.
- Sometimes cyanosis.

Diagnosis

- Clinical signs and evidence of cause.
- Auscultation – absence of normal lung sounds dorsally.

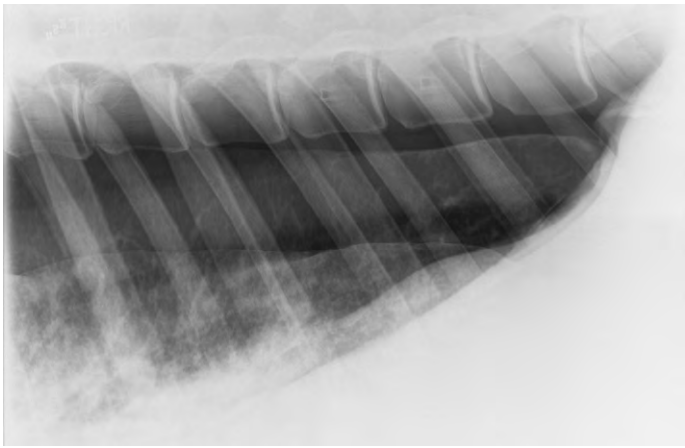


Figure 6.19 Lateral thoracic radiograph of a horse with pneumothorax. The lung has collapsed ventrally and the dorsal chest cavity is radiolucent.

- Percussion – increased resonance dorsally.
- Thoracic radiography (Figure 6.19).
- Ultrasonography.

Treatment

The underlying cause should be treated where possible. Any open wound should be cleaned and sealed. Plastic food wrap works good for sealing wounds that can't be surgically closed.

A chest drain with a one-way valve (e.g. Heimlich valve) should be introduced into the dorsal thorax to drain off the free air.

6.23 RUPTURED DIAPHRAGM

This is rare. Acquired diaphragmatic defects are commoner than congenital defects. They are usually associated with chest trauma, parturition or strenuous exercise.

Clinical signs

Variable (depending on the extent of the defect; which viscera enter the chest; and how much compromise to lung function occurs). Intestine passing through the defect may obstruct and/or strangulate, causing colic.

- Acute to chronic colic.
- Dyspnoea.
- In long-standing cases, a pleural effusion may form.

Diagnosis

- Auscultation:
  muffling of heart/lung sounds.
  borborygmi in chest. (N.B. Some referred intestinal sounds are normally heard within the chest.)
- Radiography (possibly with barium contrast).
- Rectal examination – 'empty' abdomen.

Treatment

Treatment is by surgical repair via laparotomy (see Chapter 2).

6.24 PLEURAL TRANSUDATES

Transudative pleural effusions are rare and arise as a consequence of a disease process that does not affect the pleural cavity directly. They are sometimes detected in early thoracic neoplasia.

- Hypoproteinaemia, e.g. malabsorption, protein-losing enteropathy, protein-losing nephropathy. These more commonly result in ventral subcutaneous oedema, but may result in pleural and/or peritoneal effusions.
- Increased capillary hydrostatic pressure – congestive heart failure, space-occupying lesion in the thorax.

Clinical signs

The clinical signs relate simply to the presence of a pleural effusion. Other signs referable to the primary disease problem are likely to be more obvious.

- Laboured breathing (inspiratory and expiratory dyspnoea) with flared nostrils.
- Reluctance to move or to lie down.

Diagnosis

- Auscultation – absence of lung sounds ventrally.
- Percussion – dullness in ventral chest.
- Radiography.
- Ultrasonography.
- Thoracocentesis – transudates are typically straw-coloured fluids with low total nucleated cell count ($<10 \times 10^9$/L), low total protein (<30 g/L) and low specific gravity (<1.017).

Treatment

Treat the primary disease.

N.B. Transudative effusions may occur in early neoplastic chest diseases and ruptured diaphragm. It is important to ascertain the cause of the effusion in cases where a pleural transudate is identified.

6.25 SMOKE INHALATION

Damage to the respiratory tract occurs because of the effects of heat as well as the chemical effects of smoke. Most damage arises within the upper respiratory tract, involving inflammation and oedema of the mucosa. Pulmonary damage and oedema may occur in severe cases. Small airways may become obstructed by inflammatory cells, necrotic epithelial cells and carbonaceous material. Secondary infection and pneumonia may occur.

Clinical signs

The clinical signs depend on the amount and type of smoke inhalation.

- Depression/ataxia/coma.
- Fever (38.8–39.9°C, 102–104°F) for up to 7 days.
- Dyspnoea.
- Cough.

Treatment

- Provide ventilatory support as necessary (humidified oxygen by nasal tube).
- Treat shock/toxaemia as necessary.

- Tracheostomy may be necessary if there is upper airway obstruction.
- Antibiotics (broad spectrum).
- Non-steroidal anti-inflammatory drugs.
- Corticosteroids are recommend by some clinicians, but controversial due to the risk of facilitating secondary infection.
- Provide 'dust-free' environment.
- Mucolytics and/or bronchodilators as necessary.

FURTHER READING

Mair TS (1994) Differential diagnosis and treatment of acute onset coughing in the horse. Veterinary Record suppl In Practice 16:154–162

McGorum B (1994) Differential diagnosis of chronic coughing in the horse. Veterinary Record suppl In Practice 16:55–60

McGorum BC, Dixon PM, Robinson NE, Schumacher J (2007) Equine respiratory medicine and surgery. Saunders, Edinburgh

Robertson JT, Reed SM (1991) Respiratory disease. Medicine and surgery. Veterinary Clinics of North America: Equine Practice 7(1) W.B. Saunders, Philadelphia

Rush BR, Mair TS (2004) Equine respiratory diseases. Blackwell, Oxford

Sweeney CR, Smith JA (1990) Diseases of the respiratory system. In: Smith BP (ed) Large animal internal medicine, ch 29. C.V. Mosby Co, St Louis

APPENDIX

Differential diagnoses of conditions presenting with respiratory signs

1. Acute/sudden onset coughing; viral, bacterial and parasitic infections

1. Contagious diseases, e.g.
   EHV-1 and-4.
   Equine influenza.
   Equine viral arteritis.
   Streptococcus equi (strangles).
   Streptococcus zooepidemicus.
   Dictyocaulus arnfieldi.
2. Non-contagious e.g. bacterial pneumonia/pleuropneumonia.
   Acute obstructive pulmonary disease.
   Foreign body.
   Acute dysphagia and food aspiration.
   Oesophageal obstruction.
   Smoke inhalation.
   Trauma to larynx, trachea or chest.
   Pulmonary haemorrhage.
   Pulmonary oedema.

2. Chronic coughing

RAO.
Chronic lower airway inflammation.
SPAOPD.
Chronic bacterial pneumonia/pleuropneumonia.
Pulmonary abscess.
Dictyocaulus arnfieldi infection.
Parascaris equorum larval migration.
Chronic dysphagia and food aspiration.
Chronic lesions of pharynx, larynx and trachea, e.g.
- retropharyngeal abscess.
- arytenoid chondritis.
- epiglottic entrapment.
- subepiglottic cyst.
- pharyngeal paralysis.
- tracheal stenosis/collapse.

Fungal pneumonias.
Congestive heart failure.
Tumours/polyps in airway.
Thoracic neoplasia.
Chronic interstitial lung diseases.

3. Bilateral purulent nasal discharge

Secondary bacterial infections (in viral infections).
Streptococcus equi (strangles).
Streptococcus zooepidemicus.
Bacterial pneumonia/pleuropneumonia/pulmonary abscess.
RAO.
Guttural pouch empyema/chondroids/mycosis.
Pharyngeal/retropharyngeal abscess.
Foreign body (nasopharyngeal/tracheobronchial).
Conidiobolomycosis (in endemic areas).

4. Unilateral purulent nasal discharge

Paranasal sinusitis/dental infection/sinus cyst/neoplasia.
Mycotic rhinitis.
Nasal foreign body.
Nasal tumour/polyp/cyst.
Guttural pouch empyema/chondroids/mycosis.

5. Nasal return of food

Oesophageal obstruction (choke).
Cleft palate.
Streptococcus equi (strangles).
Guttural pouch mycosis/diverticulitis/empyema.
Retropharyngeal abscess.
Grass sickness.
Gastric dilation.
Small intestinal obstruction.
Proximal enteritis (duodenitis/proximal jejunitis).
Myopathies.
Botulism.
Lead poisoning.
Gastroduodenal ulceration.
Megaoesophagus.

6. Epistaxis

EIPH.
Guttural pouch mycosis.
Progressive ethmoidal haematoma.
Trauma.
Nasal/paranasal sinus tumour.
Foreign bodies.
Clotting/bleeding disorders, e.g.
- thrombocytopenia.
- purpura haemorrhagica.
- disseminated intravascular coagulation.
- warfarin toxicity.

Conidiobolomycosis.

7. Dyspnoea in adult horse

RAO/acute obstructive pulmonary disease.
Pneumonia/pleuropneumonia/pulmonary abscess.
Retropharyngeal abscess.
Streptococcus equi (strangles).
Paranasal sinus disease.
Nasal/nasopharyngeal tumour/cyst.
Pneumothorax.
Diaphragmatic hernia.
Thoracic neoplasia.
Interstitial pulmonary disease.
Cardiac disease.
Shock.
Anaemia.
Pain.
Tracheal collapse.
Acute laryngeal conditions.
Anhydrosis.

8. Malodorous nasal discharge

Sinusitis (usually secondary to dental disease)/sinus neoplasia.
Foreign body.
Ethmoidal haematoma.
Nasal tumour.
Pneumonia/pulmonary abscess.
Mycotic rhinitis.
Guttural pouch mycosis.

Chapter 7

Cardiovascular system

Colin C. Schwarzwald

CONTENTS

7.1 Overview of heart disease in the horse 133
7.2 Evaluation of cardiovascular function 133
Signalment 133
Medical history 133
Physical examination 134
Ancillary diagnostics 138
7.3 Structural heart disease 142
Congenital malformations 142
Acquired valvular heart disease 143
Pericardial disease 147
Myocardial disease 147
7.4 Heart failure 148
7.5 Cardiac rhythm disturbances 149
Physiological arrhythmias 150
Pathological arrhythmias 151
7.6 Vascular disease 156
Jugular thrombophlebitis 156
Aorto-iliac thrombosis 157
Vascular ruptures 158
Further reading 158

7.1 OVERVIEW OF HEART DISEASE IN THE HORSE

Congenital and acquired cardiac disorders of the horse include valvular, myocardial, pericardial, and vascular diseases. Compared to other species, primary heart disease is relatively rare in horses. If present, it is often asymptomatic or leads to unspecific clinical signs such as exercise intolerance and poor performance. Occasionally, horses present with signs of advanced heart disease and congestive heart failure at rest.

The cardiovascular system can also be affected by other diseases such as colic, diarrhoea, endotoxaemia, severe respiratory diseases, or electrolyte and acid–base imbalances. The equine clinician should be familiar with the most common primary and secondary cardiovascular conditions, the underlying pathophysiological mechanisms, the potential haemodynamic and electrophysiological consequences, the clinical relevance, the treatment options, and the prognosis in regard to life and to future athletic use.

7.2 EVALUATION OF CARDIOVASCULAR FUNCTION

A cardiovascular examination includes the inquiry of a complete signalment and history, a thorough physical examination, and careful auscultation of heart and lungs. Electrocardiography, echocardiography, thoracic radiography, clinical laboratory tests, exercise testing, and other, more advanced, diagnostics may follow as necessary.

Signalment

- Generally (and with exceptions), congenital defects are present from birth and are diagnosed in foals and young horses (<3–5 years). Acquired diseases usually develop with advancing age and are more likely diagnosed in middle-aged and old horses.
- Congenital defects may be more common in certain breeds (e.g. Arabians).
- Some diseases may depend on the sex of the horse (e.g. aorto-cardiac fistulas in stallions).

Medical history

- The current and prospective use of the horse, the current exercise capacity, and the actual and expected level of performance are important for assessment of the clinical relevance and prognosis of cardiac disease.
- Previous health issues may be a cause or a result of cardiovascular disease and should be carefully reviewed.

© 2013 Elsevier Ltd
DOI: 10.1016/B978-0-7020-2801-4.00007-9

- Presenting complaints are important indicators of the severity and the clinical relevance of cardiovascular disease.
  - Heart murmurs and cardiac arrhythmias are common in athletic horses and must be evaluated in relation to the medical history and physical examination findings; they may not be clinically relevant in the absence of other clinical signs consistent with heart disease.
  - Exercise intolerance and poor performance are common, but very unspecific complaints that can be associated with cardiac disease and early heart failure. Other causes of poor performance such as respiratory, orthopaedic, neurological, or metabolic disorders also have to be considered. Generally, normal performance in a top-class athlete likely precludes the presence of severe cardiac disease and heart failure.
  - Cough, nasal discharge, and increased respiratory effort may be signs of advanced heart failure or respiratory disease.
  - Poor growth and exercise intolerance in foals (often associated with apparent signs of respiratory disease) may be a result of cardiac disease.
  - Horses with severe systemic disease such as patients with gastrointestinal disease, endotoxaemia, septicaemia, or severe acid–base and electrolyte disturbances are likely to suffer from some degree of circulatory compromise. Assessment of the volume status and cardiovascular function is therefore of great importance in all severely ill patients to choose the appropriate protocols for treatment and monitoring.

Physical examination

The physical examination aims to detect signs of poor circulatory function, heart disease, or heart failure, as well as diseases of the respiratory tract and of other organ systems.

Inspection and palpation

The body surface temperature, the turgor of the skin, the colour of the mucous membranes, the capillary refill time, the jugular vein filling, and the rate and quality of peripheral arterial pulsations provide important information on volume status, peripheral vascular perfusion, blood pressure, and vascular reactivity. Signs of clinically manifest heart failure may be detected through careful inspection and palpation.

- An increase in body surface temperature may indicate fever secondary to an infectious or inflammatory process (e.g. endocarditis or pericarditis); conversely, a cool or cold body surface may be due to poor cardiac output, hypotension, intense peripheral vasoconstriction, and circulatory collapse.
- Reduced skin turgor (>1 second) may indicate dehydration.
- The mucous membranes may be pale in patients with anaemia or intense peripheral vasoconstriction. Injected (dark red) mucous membranes are seen in association with septicaemia or endotoxaemia. Grey mucous membranes may indicate circulatory collapse and poor peripheral perfusion. Cyanosis (bluish discoloration due to severe hypoxia) is a rare finding in horses with cardiac disease.
- The capillary refill time (CRT) is prolonged (>2 seconds) during hypotension and with hypovolaemia, poor cardiac output, or intense peripheral vasoconstriction; conversely, it may be shortened (<1 second) when there is vasodilation.
- Jugular pulsations may be observed in healthy horses in the ventral one third of the neck and are more pronounced in excited horses or in horses with very high sympathetic tone. Abnormal jugular pulses may be observed with arrhythmias, with diseases of the tricuspid valve, with pericardial diseases, and with right ventricular or biventricular failure.
- Jugular vein distension can be caused by obstruction to flow (e.g. jugular vein thrombosis, intrathoracic mass), pericardial disease, increased intrathoracic pressure (e.g. pleural effusion), or heart failure. However, many horses have significant heart disease without jugular vein distension. A lack of appropriate jugular vein filling is consistent with severe hypovolaemia or venous occlusion.
- Heart rate and rhythm as well as altered haemodynamic states can be identified by palpating the facial artery pulse. The arterial pulse can be described as normal, hypokinetic (weak), hyperkinetic (bounding or 'water-hammer' pulse), or variable. Irregularity and variable pulse quality often result from cardiac arrhythmia.
  - It should be noted that palpation of a peripheral pulse wave only allows estimation of pulse pressure (difference between systolic and diastolic arterial blood pressure). However, it neither provides an accurate estimate of absolute systolic and diastolic blood pressures nor corresponds to blood flow or tissue perfusion.
- Peripheral oedema may indicate right ventricular or biventricular failure, vascular occlusion, severe hypoproteinaemia, vascular disease, or impaired lymphatic drainage. The latter causes should be ruled out even if there is evidence of heart disease.
- Nasal discharge, rapid breathing, increased depth of breathing, or increased respiratory effort at rest or following exercise may indicate cardiovascular or respiratory disease. Cough is relatively uncommon in horses with left-heart failure and more likely indicates respiratory disease.

Auscultation

Both cardiac and respiratory disease can result in non-specific clinical signs, including exercise intolerance and poor performance. Careful auscultation of the heart and lungs (including rebreathing examination) is therefore a crucial part of the physical examination.

The diagnostic sensitivity of cardiac auscultation is fairly high and many serious cardiac disorders, including most congenital malformations, valvular diseases, and persistent cardiac arrhythmias, can be detected by physical examination and careful auscultation. However, the differentiation between physiological (clinically irrelevant) and pathological (clinically relevant) cardiac murmurs and arrhythmias can be difficult and the correct diagnosis of the underlying cardiac problem largely depends on the clinician's knowledge and experience. Primary pericardial and myocardial diseases may be difficult to detect by cardiac auscultation.

- Normal cardiac auscultation in a horse with good exercise tolerance practically precludes clinically relevant heart disease.
- Most sustained or recurrent cardiac arrhythmias and most clinically relevant heart murmurs are easily discovered through cardiac auscultation.
- Crackles or wheezes on thoracic auscultation may indicate left-heart failure or respiratory disease.

Auscultation should be performed in a quiet environment, using a high-quality stethoscope.

Knowledge of the areas for auscultation (Figure 7.1) and appreciation of normal heart sounds in the horse (Figure 7.2) are important prerequisites. Also, the examiner must be familiar with the causes and clinical features of murmurs and arrhythmias in the horse (Tables 7.1 and 7.2) and needs to be able to precisely describe the auscultatory findings.

Heart sounds are produced by oscillations of cardio-haemic structures that occur during the cardiac cycle. Their intensity and character depend on loading conditions, rate and force of cardiac contraction, and chamber stiffness. In normal horses, two, three or four heart sounds may be identified on auscultation (Figure 7.2 and Table 7.2).

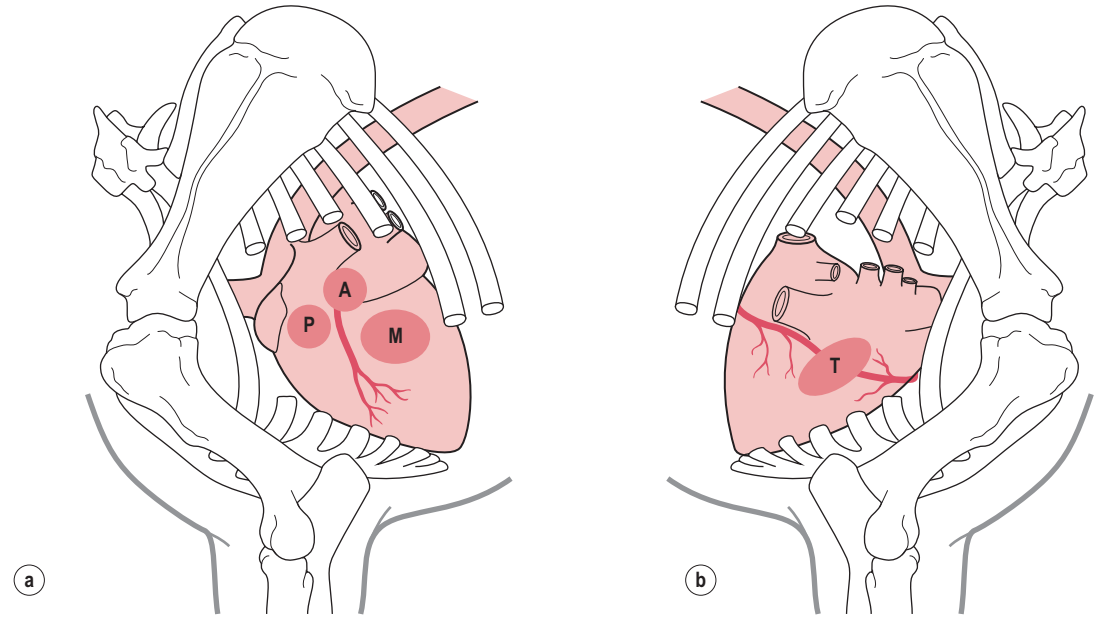


Figure 7.1 Cardiac auscultation areas in the horse from the left (a) and the right (b) side of the thorax. Note the size and anatomical position of the heart within the thorax. The cardiac apex (*apical area*) is usually located slightly above the level of the olecranon and can be identified by palpation of the heart beat. The cardiac base (*basilar area*) is located more cranially and at the level of the scapulohumeral joint. The shaded areas represent the respective valve areas (P = pulmonic vale; A = aortic valve; M = mitral valve; T = tricuspid valve).

Published in Reef VB (2003) Cardiovascular system. In: Orsini JA & Divers TJ (eds) Manual of equine emergencies, 2nd edn. Saunders, reprinted with permission of Elsevier Ltd.

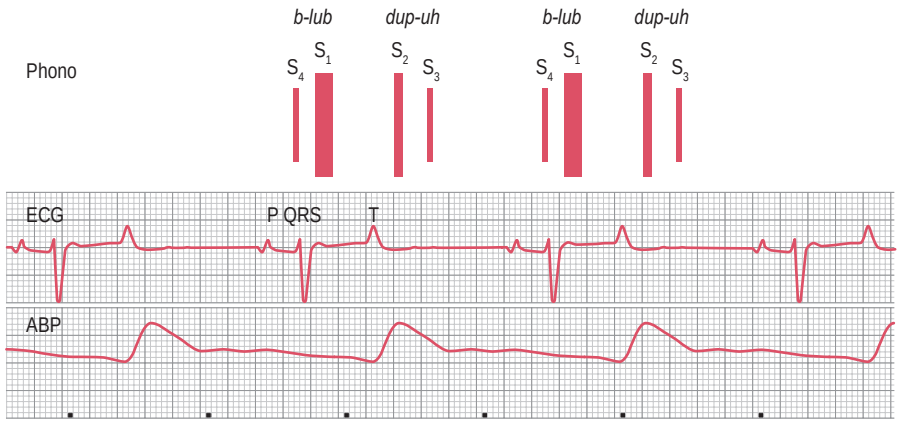


Figure 7.2 Schematic diagram of the normal heart sounds (Phono) in relation to a surface electrocardiogram (ECG) and an arterial blood pressure tracing (ABP) recorded from the transverse facial artery. S_1 = first (systolic) heart sound; S_2 = second (diastolic) heart sound; S_3 = third heart sound; S_4 = fourth (atrial) heart sound. '*B-lub dup-uh*' describes the sounds heard on auscultation. Note the timing of the heart sounds, the (peripheral) pulse pressure wave, and the ECG in relation to each other.

Published in Schwarzwald CC, Bonagura JD, Muir WW (2009) The cardiovascular system. In: Muir WW (ed) Equine anesthesia – monitoring and emergency therapy, 2nd edn. Saunders, p 67, reprinted with permission of Elsevier Ltd.

- The atrial sound (S4) is caused by atrial contraction at the end of diastole. It is heard loudest over the heart base and is audible (more or less pronounced) in most normal horses. Variation in the electrical activation of the atria and AV node often leads to gradual changes in the S4–S1 interval.
- The systolic heart sound (S1) is associated with ventricular contraction and occurs immediately after closure of the atrioventricular (mitral, tricuspid) valves. It is loudest over the apical area and can be heard in all normal horses. Variations in the intensity and quality of S1 can be caused by changes in electrical activation patterns, after prolonged diastolic periods, and with arrhythmias.
- The diastolic heart sound (S2) is associated with the beginning of ventricular relaxation and occurs immediately after closure of the semilunar (aortic, pulmonic) valves. It is loudest over the base of the heart. The second heart sound may be soft or absent after premature beats, when the stroke volume is very small. Close splitting of S2 may occur in normal horses due to asynchronous closure of the aortic and pulmonic valves. Splitting may vary with heart rate and respiration. Splitting of S2 with a loud, accentuated pulmonic component or occurrence of a single loud, pounding S2 may indicate the presence of increased blood pressures in the pulmonary circulation (pulmonary hypertension).

Table 7.1 Auscultation of cardiac arrhythmias

Rhythm	Typical heart rate	Heart sounds*	Auscultatory features
Sinus rhythms	26–48/min	S4-1-2 (3)	Rate and rhythm depend on autonomic tone
Sinus arrest/block†	<26/min	S4-1-2 (3)	Irregular, long pauses
Sinus bradycardia†	<26/min	S4-1-2 (3)	Generally regular
Sinus arrhythmia†	26–48/min	S4-1-2 (3)	Irregular, cyclic change in heart rate, usually variable interval between S_4-S_1, often associated with second-degree AV block
Sinus tachycardia	>48/min	S4-1-2 (3)	Typically regular
Premature complexes			
Supraventricular (atrial) Ventricular	— —	Premature S1-2	Irregular rhythm with single extra beats; intensity of S_1 may be louder or softer than normal
Atrial tachyarrhythmias			
Atrial tachycardia	AR: variable‡ VR: >30/min	Sl-2 (3)	Ventricular regularity/rate depend on AV conduction; S_4 inconsistent or absent; variable intensity S_1
Atrial flutter Atrial fibrillation	AR: 200–300/min‡ AR: 250–500/min‡ VR: >30/min	Sl-2 (3)	Ventricular response is irregular; ventricular rate depends on autonomic tone but is often normal (30–54/min); heart rates consistently above 60/min suggest underlying heart disease or heart failure; S_4 is absent; variable intensity S_1
Ectopic junctional and ventricular rhythms			
Escape rhythm – junctional Escape rhythm – ventricular Accelerated idioventricular rhythm (slow VT) Ventricular tachycardia (VT)	>25/min <25/min 60–80/min >80/min	S1-2 (3)	Ventricular rate usually regular during ectopic rhythm; rate depends on mechanism and autonomic tone; inconsistent S_4; variable intensity and split heart sounds
Atrioventricular block			
First degree AV block Second degree AV block†	<48/min	S4-1-2 (3) S4/S1-2 (3)	Heart rate variable; cyclical arrhythmia, variable S_4-S_1 interval; some variation in heart sounds; isolated S_4 without following S_1-S_2 in second-degree AV block
Complete (third degree) AV block	AR: 28–60/min VR: <25/min	S4//S1-2 (3)	Ventricular escape rhythm, usually regular; independent atrial (S_4) sounds; variable intensity heart sounds

*( ) = may be evident; †usually physiological; ‡exact rate limits are not established in horses; AR = atrial rate; VR = ventricular rate; AV = atrio-ventricular; VT = ventricular tachycardia.

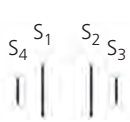
Published in Schwarzwald CC, Bonagura JD, Muir WW (2009) The cardiovascular system. In: Muir WW (ed) Equine anesthesia – monitoring and emergency therapy, 2nd edn, Saunders, p 66, reprinted with permission of Elsevier Ltd.

- The diastolic filling sound (S3) is caused by rapid inflow and sudden deceleration of blood in the ventricles during early diastole. It is characterized by a low intensity and low frequency and is best heard over the apical area of the heart. The third heart sound may only be heard in a localized area. It may summate with S4 at high heart rates. The third heart sound is considered a normal finding in horses, especially in young and athletic horses. However, S3 may become more pronounced and loud with ventricular dilation and elevated filling pressures (heart failure) and may then be used as a marker of severity of cardiac disease.

The examiner needs to be familiar with the auscultatory findings in the normal horse.

- The normal resting heart rate generally ranges between 26 and 48/min (Table 7.1). Heart rate in the horse can change rapidly and dramatically, depending on autonomic tone and the level of physical activity. Anxiety and exercise may cause sudden increases in heart rate.
- With a normal, regular rhythm, the intensity of heart sounds is steady. Accentuated sounds may be heard with excitement and high sympathetic activity.
- The resting normal horse with high vagal tone may exhibit physiologic bradyarrhythmias including atrioventricular block, sinus arrhythmia, and sinus block/arrest (Table 7.1). These arrhythmias are characterized by a slow heart rate (often less than 30/minute) and occasional pauses ('dropped beats') that may occur at more or less regular intervals (e.g. one pause after every 4 normal beats). Reducing vagal tone, e.g. by turning the horse quickly in a tight circle or by examining the horse immediately after exercise usually causes the arrhythmia to abate. If the arrhythmia persists or if the auscultatory findings suggest another arrhythmia, an electrocardiogram should be obtained.

Table 7.2 Identification of heart sounds and common cardiac murmurs

Auscultatory finding	Timing°		Point of maximal intensity (valve area)*
Normal Heart Sounds			
First heart sound (S_1)	Onset S		Left apex (mitral valve)
Second heart sound (S_2)	End S		Left base (aortic valve)
Pulmonic component	End S		Left base (pulmonic valve)
Third heart sound (S_3)	Early D		Left apex (mitral valve)
Fourth (atrial) sound (S_4)	Late D		Ventricular inlet or base (Left)
Functional Murmurs[†]			
Systolic ejection murmur	S		Left base (Aortic/pulmonary valves)
Early (proto-)diastolic	D		Ventricular inlets (Left/Right)[‡]
Late diastolic (presystolic)	D		Ventricular inlets (Left/Right)[‡]
Valvular Regurgitation[§]			
Mitral regurgitation	S		Left apex (mitral valve)
Tricuspid regurgitation	S		Right hemithorax (tricuspid valve)
Aortic regurgitation	D		Left base (aortic valve)
Pulmonary insufficiency	D		Left base (pulmonic valve)
Ventricular Septal Defect[‖]	S		Right sternal border/left cardiac base
Patent Ductus Arteriosus	S + D		Dorsal left base over pulmonary artery

°S – systole, the interval between S_1 and S_2; D – diastole, the interval between S_2 and S_1.

*Only typical features are considered; 'apex' refers to the ventral part of the heart, at the point of the palpable cardiac impulse (apical beat); 'base' refers to the craniodorsal part of the heart over the outlet valves (aortic, pulmonic), where the second heart sound is most intense.

[†]Functional murmurs are typically soft, localized, and labile.

[‡]Ventricular inlets refer to the parts of the thorax overlying the ventricular inflow tracts. These include the areas just dorsal to the mitral and tricuspid valve areas and extend ventrally to the apical regions of the ventricles.

[§]Murmurs of AV valve insufficiency are generally heard over the affected valve, project prominently toward the respective ventricular apex, and also radiate dorsally, following the regurgitant jet into the atrium. Late systolic murmurs, which may be related to valve prolapse, have been identified with mitral or tricuspid valve insufficiency. The murmur of aortic insufficiency may not always be holodiastolic. Pulmonary insufficiency is often silent.

[‖]Defects in the RV outlet septum (subpulmonic VSDs, rare) or increased flow across the pulmonic valve (relative pulmonic stenosis) can cause left-basilar systolic murmurs. Flow across very large nonrestrictive septal defects can be relatively soft.

From Schwarzwald CC, Bonagura JD, Muir WW (2009) The cardiovascular system. In: Muir WW (ed) Equine anesthesia – monitoring and emergency therapy, 2nd edn, Saunders, p 65.

- Heart murmurs are commonly heard in many normal horses. Most of them are so called functional (flow) murmurs (Table 7.2). Functional murmurs are very common in foals, trained athletes (athletic murmur), and in horses with high sympathetic nervous system activity. They can also be associated with fever and are often heard in anemic horses.
  - Typically, functional murmurs are soft (grade 1–3/6), localized, and labile. They are very dependent on physiological state and can be altered by changing the heart rate (e.g. they may become more prominent at higher heart rates).
  - The most common physiologic murmur is the systolic ejection murmur which begins after the first heart sound and ends before the second heart sound and is heard best at the left cardiac base over the aortic and pulmonic valves.
  - Early to mid diastolic murmurs extend from S2 to S3 and are associated with rapid filling of the ventricles. They are quite common, especially in athletic horses, and may be musical (squeaky) in character.
- Trivial or mild regurgitation across right-sided (most common) or left-sided (less common) cardiac valves can be identified in some horses by Doppler echocardiography. This is considered a normal finding of no clinical relevance provided that the regurgitation is of short duration and no other findings indicate the presence of cardiac disease. Many (up to 30%) trained equine athletes have audible murmurs of tricuspid and/or mitral regurgitation that are not associated with poor performance or signs of heart disease (by physical examination and echocardiography) and are not considered clinically relevant. Clinically negligible aortic and pulmonary insufficiencies are often silent.
- The continuous murmur of patent ductus arteriosus is normally present in full-term foals for up to 4 days post parturition.

Furthermore, normal foals can present with soft, left-basilar functional murmurs that are heard during systole and that can persist up to 30–60 days after birth.

Auscultation should include assessment of (1) heart rate, (2) intensity and character of the heart sounds, (3) heart rhythm, and (4) murmurs or 'extra sounds'.

- Heart rate:
  - An increase in heart rate (tachycardia) can be due to exercise, excitement, stress and anxiety, pain, fever, hypovolaemia and hypotension, anaemia, shock, metabolic derangements, electrolyte imbalances, drug effects (e.g. atropine, catecholamines), or primary heart disease (e.g. tachyarrhythmias, heart failure).
  - A decrease in heart rate (bradycardia) may result from an increase in vagal tone, drug effects (e.g. alpha-2 agonists, anaesthetics, digitalis glycosides), hypothermia, electrolyte imbalances (e.g. severe hyperkalaemia), or primary disturbances of impulse formation or impulse conduction in the heart.
  - Unexplained, persistent tachycardia or bradycardia should be assessed by means of electrocardiography to rule out rhythm disturbances other than sinus tachycardia and sinus bradycardia, respectively (Table 7.1).
- Heart sounds:
  - Accentuated sounds may be heard with volume overload or high sympathetic activity.
  - Varying intensity of normal heart sounds can occur due to cardiac rhythm disturbances leading to irregular ventricular filling and stroke volume.
  - Muffled heart sounds can indicate pericarditis and pericardial effusion.
- Rhythm disturbances:
  - With some exceptions (e.g. slow ventricular tachycardia or some ventricular conduction disturbances), cardiac rhythm disturbances are generally easily detected by careful auscultation. Pathological rhythm disturbances need to be differentiated from physiological dysrhythmias (Table 7.1).
- Heart murmurs:
  - The presence of a cardiac murmur is an essential finding that leads one to suspect potential acquired valvular disease or a congenital cardiac malformation. Pathological murmurs need to be differentiated from physiological murmurs (Table 7.2).
  - Heart murmurs should be characterized based on timing and duration, grade, point of maximal intensity, radiation, and quality.
    1. Timing and duration. Murmurs can be systolic (from S1 to S2), diastolic (from S2 to S1), or continuous (Figure 7.2, Table 7.2). Depending on the timing of appearance they are called early, mid, or late systolic and diastolic, respectively. Murmurs that are heard throughout systole and diastolic are referred to as holosystolic and holodiastolic.
    2. Grade. Grade 1/6 murmurs are very quiet, are only heard over a very localized area after careful auscultation in quiet environment, and may be inconsistent. Grade 2/6 murmurs are quiet, but are heard consistently over the point of maximal intensity. Grade 3/6 murmurs are moderately loud, are heard immediately and consistently, and have a small area of radiation. Grade 4/6 murmurs are loud, radiate over a wider area, but are not associated with a palpable thrill. Grade 5/6 murmurs are very loud, radiate over a widespread area, and are associated with a palpable thrill. Grade 6/6 murmurs are extremely loud and are also heard with the stethoscope held just above the skin surface.
    3. Point of maximal intensity (PMI). Apical murmurs are heard best at the location of the thoracic wall cardiac impulse (apical beat), approximately at or slightly above the level of the elbow (ventral region of the left ventricular inlet). Basilar murmurs are heard best over the area above the elbow and slightly more cranial, below the triceps muscle (region of the ventricular outflow tracts, semilunar valves, and great vessels). The PMI can also be described in relation to the pulmonic, aortic, mitral, and tricuspid valve areas (Figure 7.1).
    4. Radiation. Murmurs can radiate dorsally or ventrally, cranially or caudally, and to the left or right in relation to the PMI.
    5. Quality. The quality of the murmurs can be characterized based on the frequency of the sound as high pitch, low pitch, or mixed pitch. Furthermore, murmurs can be described as harsh, coarse, rumbling, muscial, squeaky, honking, or blowing.
  - Most murmurs can be distinguished through careful auscultation (Table 7.2), including evaluation of the effect of changing heart rate on the heart murmurs (e.g. by jogging or turning the horse in a tight circle). The assessment of the clinical relevance of an organic heart murmur may require ancillary tests (see below).
  - Causes of pathological murmurs include incompetent cardiac valves, septal defects, and vascular lesions. Valvular stenosis is very rare in horses and can be disregarded for most instances.
  - Significant myocardial disease may be associated with both arrhythmias and cardiac murmurs, especially in advanced cases in which ventricular dilatation causes secondary insufficiency of the mitral or tricuspid valves.
- Besides the heart sounds and classical murmurs, 'extra sounds' are sometimes detected.
  - Systolic clicks can occasionally by heard over the left apex and may indicate mitral valve disease or prolapse (especially when associated with a mid- to end-systolic murmur of mitral regurgitation).
  - Ventricular knocks are loud ventricular filling sounds (correlating to S3) heard with constrictive pericardial disease.
  - Pericardial friction rub typically is a triphasic creaking or scratching sound (systole, early diastole, late diastole) indicating pericarditis.

Ancillary diagnostics

Electrocardiography

Electrocardiography is the method of choice for assessment of cardiac rhythm disturbances.

- A resting electrocardiogram (ECG) recorded over a few minutes or less may reveal frequent or persistent rhythm disturbances, while infrequent or episodic disturbances may go undetected.
- Telemetric ECG recorders can be used in exercising horses and allow detection of exercise-induced arrhythmias.
- Ambulatory (Holter) ECG equipment is used for rhythm monitoring over a prolonged period of time (up to 24 hours or longer) and allows detection of infrequent or episodic arrhythmias.
- Generally, a normal ECG does not preclude heart disease in the horse, and the ECG cannot be used to assess cardiac dimensions or mechanical function of the myocardium.

The electrocardiogram is a tracing of the average electric potential generated by the heart muscle recorded throughout the different phases of the cardiac activation process and graphed in terms of

voltage (displayed on the y axis) and time (displayed along the x axis) (Figure 7.2).

The normal ECG complex waveforms – the P wave, the QRS complex, and the T wave – must be understood to diagnose arrhythmias and detect other electrical abnormalities.

- The P wave is generated by atrial depolarization, originating from the sino-atrial node.
- The PQ interval (often also referred to as PR interval) represents the time for conduction across the atrio-ventricular node and the His-Purkinje system.
- The QRS complex represents the electrical activity during ventricular excitation (depolarization).
- Repolarization of the ventricles begins at the end of the QRS complex and extends to the end of the T wave.
- The QT interval represents the total electrical activation-repolarization time.
- The amplitude and duration of these waveforms depend on many factors, including body mass and age of the horse, lead examined, size of the cardiac chambers, and mode of electrical activation.

A systematic approach to ECG analysis should be undertaken.

- The base–apex lead is the preferred lead for monitoring cardiac rhythm in horses. To obtain a base–apex recording, the left arm (LA) electrode is placed left over the cardiac apex (left chest, just caudal to the olecranon), the right arm (RA) electrode is placed over the right jugular furrow, and 'lead I' is selected on the electrocardiograph. The left leg (LL) electrode can be placed anywhere on the horse; for convenience, it may be placed next to the LA electrode on the left side of the chest.
- A paper speed of 25 mm/sec and voltage calibration of 1 cm/mV are commonly used for standard ECGs. These settings may need to be adjusted to obtain a good quality tracing.
- Evaluation of the electrocardiogram includes:
  1. Determination of heart rate.
     - Ventricular rate (count QRS-T complexes)
     - Atrial rate (count P waves)
     - The normal resting heart rate in adult horses ranges between 26 and 48 beats/min. Small-breed horses and ponies may have slightly higher resting heart rates (up to 54 beats/min).
     - In normal horses, atrial rates should equal ventricular rates unless second-degree AV block is present (see below).
     - Based on the heart rate, arrhythmias can be categorized as tachyarrhythmias (elevated heart rate) or bradyarrhythmias (slow heart rate).
  2. Assessment of cardiac rhythm.
     - Regular rhythm. RR intervals do not vary significantly or change abruptly.
     - Regularly irregular rhythm. Patterns of repeating cycles can be identified (e.g. second-degree AV block with 4 normal beats followed by 1 blocked beat).
     - Irregularly irregular rhythm. Chaotic rhythm without any pattern (i.e. RR intervals vary abruptly and in a random fashion; e.g. atrial fibrillation with irregular atrio-ventricular impulse conduction or multiform ventricular tachycardia).
  3. Assessment of waves and complexes.
     - Identify and compare normal and abnormal complexes. Even the most severe, persistent arrhythmias may be interspersed with normal P-QRS-T complexes. Identification of these complexes may greatly facilitate the assessment of abnormal complexes and is often a prerequisite for a correct rhythm diagnosis.
     - Assess the morphology, variations, and associations of waves and complexes.

P wave. The normal P wave is positive (when using a standard base-apex lead) and can be notched or bifid; however, single-peaked, biphasic (negative/positive), and polyphasic P waves may be encountered. The P wave morphology can change in a cyclic manner during sinus arrhythmia in horses (wandering pacemaker). During normal sinus rhythm, every P wave should be followed by a QRS complex. Second-degree AV block is seen frequently in healthy horses, in which case some of the P waves are not followed by a QRS complex.

PQ interval. Physiological variation in the PQ interval can be observed in many horses. Values that persistently exceed 450–500 ms are considered abnormal in horses of larger breeds and indicate disturbed atrio-ventricular impulse conduction (i.e. first-degree AV block). The PQ interval is shorter in smaller horses and ponies, with an upper limit of approximately 300–350 ms.

QRS complex. In normal horses, all QRS complexes are morphologically identical and are preceded by a normal P wave. When using a standard base-apex lead, normal QRS complexes appear negative.

The normal duration of the QRS complex is 80–140 ms in large-breed horses and 60–120 ms in smaller horses. Abnormal conformation (i.e. duration, orientation) of the QRS complex indicates ventricular conduction disturbance (in which case the P wave precedes the QRS complex, indicating a supraventricular activation pathway) or activation of the ventricles from an ectopic ventricular focus (in which case a preceding P wave cannot be identified).

T wave. The T wave morphology is variable in horses. Using a standard base–apex lead, the T wave is usually biphasic (negative/positive). Physiological changes in morphology or size occur during exercise (e.g. large, positive T waves). Enlargement of the T wave may also develop with myocardial hypoxia or hyperkalaemia (see below). Furthermore, altered T waves can be seen following abnormal QRS complexes (secondary T wave changes after abnormal ventricular activation).

QT interval. The upper limits for the QT interval at resting heart rates are approximately 560–600 ms in adult horses. The QT interval shortens at higher heart rates and strongly depends on changes in autonomic tone. Prolongation of the QT interval indicates delayed ventricular repolarization and can predispose to ventricular arrhythmias. It may be caused by a variety of drugs (e.g. quinidine, procainamide, flecainide, macrolide antibiotics).

- Determine the site of abnormal impulse formation:

Supraventricular arrhythmias originate above the AV node. They are characterized by a normal QRS-T complex that is preceded by a P wave or by flutter/fibrillation waves (see below).

Junctional arrhythmias originate in the AV node or bundle of His. They are characterized by a normal or near-normal QRS-T morphology. P waves may be identified, but are not consistently associated with the QRS complexes (AV dissociation; see below).

Ventricular arrhythmias originate below the bundle of His. They are characterized by an abnormal (bizarre) QRS-T morphology (i.e. wide, large complexes that are oriented differently compared to normal complexes). P waves may be identified between the abnormal complexes, but

are not consistently associated with the QRS complexes (AV dissociation; see below).

Echocardiography

Echocardiography is the technique of choice for evaluating horses with suspected cardiac disease. It allows assessing the size and mechanical function of the four cardiac chambers, visualizing valvular function, identifying specific cardiac lesions, and detecting abnormal blood flow. The severity of cardiac disease, its haemodynamic consequences, and its progression can be assessed.

The most important indications for echocardiography in horses include:

- Evaluation of heart murmurs. Identification of the source of the murmur, differentiation between physiological and pathological murmurs, assessment of the effects on heart size and function, and assessment of the clinical relevance of pathological murmurs.
  - A normal echocardiogram in a horse with a cardiac murmur is a favourable finding. Conversely, identification of significant cardiomegaly or abnormal ventricular function may indicate considerable prognostic risk.
- Evaluation of cardiac arrhythmias. Detection of underlying structural or functional cardiac disease (e.g. mitral regurgitation and left atrial dilation leading to atrial fibrillation).
- Evaluation of suspected congenital defects. Evaluation of heart murmurs, unexplained cyanosis, dysrhythmias, or signs of heart failure in neonates.
- Evaluation of congestive heart failure. Identification of the cause of heart failure, assessment of severity of underlying disease, monitoring of progression and response to treatment.
- Evaluation of severe respiratory disease: Diagnosis of pulmonary hypertension or Cor pulmonale, detection of patent foramen ovale in foals with respiratory disease.
- Detection or rule-out of:
  - Cardiac disease in horses with exercise intolerance or poor performance.
  - Pericardial disease in horses with muffled heart sounds, pericardial friction rubs, and/or distended jugular veins.
  - Endocarditis in horses with fever of unknown origin (± heart murmur).
  - Cardiac disease in horses with unexplained collapse or episodic weakness.

A systematic approach using standardized image planes (identified by means of specific intracardiac landmarks) is important for accurate measurements of cardiac dimensions, comparison of studies over time, and comparison of studies among different examiners.

- Two-dimensional echocardiography allows evaluation of the size of the cardiac chambers and the large vessels (aorta, pulmonary artery), the structure and thickness of the chamber walls, the structure and function of the heart valves, the myocardial function, and the anatomical relationship between the cardiac chambers and the large vessels.
- M-mode echocardiography shows the cardiac structures on the cursor line displayed over time. The motion of the heart and heart valves can be recorded with a very high temporal resolution. M-mode is routinely used for measurement of the left ventricular diameter, left ventricular free wall, and interventricular septal thickness in systole and diastole. The fractional shortening, an indicator of left ventricular systolic function, can be calculated.
- Doppler echocardiography is used to estimate blood flow velocities using the shift in ultrasound frequency which occurs after the ultrasound waves have been reflected by moving red blood cells (Doppler principle).
  - Color flow Doppler is routinely used to screen for disturbances of normal blood flow in a specified area of interest (e.g. around a valve or in areas with congenital defects). Direction of blood flow, its velocity, and its flow characteristics (e.g. turbulent flow vs. laminar flow) can be assessed using a colour-coded display superimposed to a 2D image (for location) or M-mode recording (for timing). Traditionally, flow directed toward the transducer is coded red and flow away from the transducer is coded blue. Turbulent flow may be coloured green.
  - In pulsed-wave (PW) Doppler and continuous-wave (CW) Doppler modes, blood flow velocity is displayed (on the y-axis) versus time (on the x-axis). Pressure gradients between two chambers (e.g. between the left and right ventricle in the presence of a VSD, or between the right atrium and the right ventricle in the presence of tricuspid regurgitation) can be estimated based on the velocity of blood flowing between the two chambers measured by Doppler echocardiography. Estimation of pressure gradients is a very useful tool for evaluation of hemodynamic consequences of cardiac disease. For example, Doppler echocardiography can be used for assessment of haemodynamic significance of ventricular septal defects (pressure difference between right and left ventricle), or for detection of pulmonary hypertension (if tricuspid regurgitation or pulmonic insufficiency are present).

Figure 7.3 shows echocardiograms obtained from the right side of the chest.

Exercise testing

- Evaluation during and after exercise may be useful in asymptomatic horses or in horses with unspecific or subtle clinical signs (e.g. exercise intolerance) when cardiac disease is suspected.
- Horses can be lunged, ridden, or exercised on a high-speed treadmill. The level of exercise should be chosen individually according to the horses' condition and training status.
- Heart rate monitors or telemetric ECG units can be used allowing heart rate and rhythm to be monitored before, during, and after exercise. If such equipment is not readily available, heart rate and rhythm can be obtained by auscultation immediately after exercise. Post-exercise recovery should be monitored every 5 min for at least 20 min or until heart rate and respiratory rates are normal.
- As a general rule, the heart rates in fit horses range between 60 and 80 beats/min during walking, 80–120 beats/min during trotting, 120–150 beats/min at a moderate canter, 150–180 beats/min at a gallop, and up to 200–240 beats/min at maximal effort. The heart rate in fit horses falls quickly in the first minute after exercise, should be below 100 beats/min after 4–5 min (usually after 1–2 min), and reaches baseline values within 15–30 min of strenuous exercise. Recovery times are strongly influenced by the fitness of the horse, the level of exercise, and environmental factors. Cardiovascular or respiratory dysfunction can increase heart rates during exercise and may prolong recovery times.
- Respiratory rate should not exceed heart rates at any time during exercise and should return to baseline values within 10–20 min after exercise.
- Transient sinus arrhythmia and second-degree AV block are considered normal findings in the immediate post-exercise period. Occasional, isolated supraventricular or ventricular ectopic beats can be seen in many normal horses during and

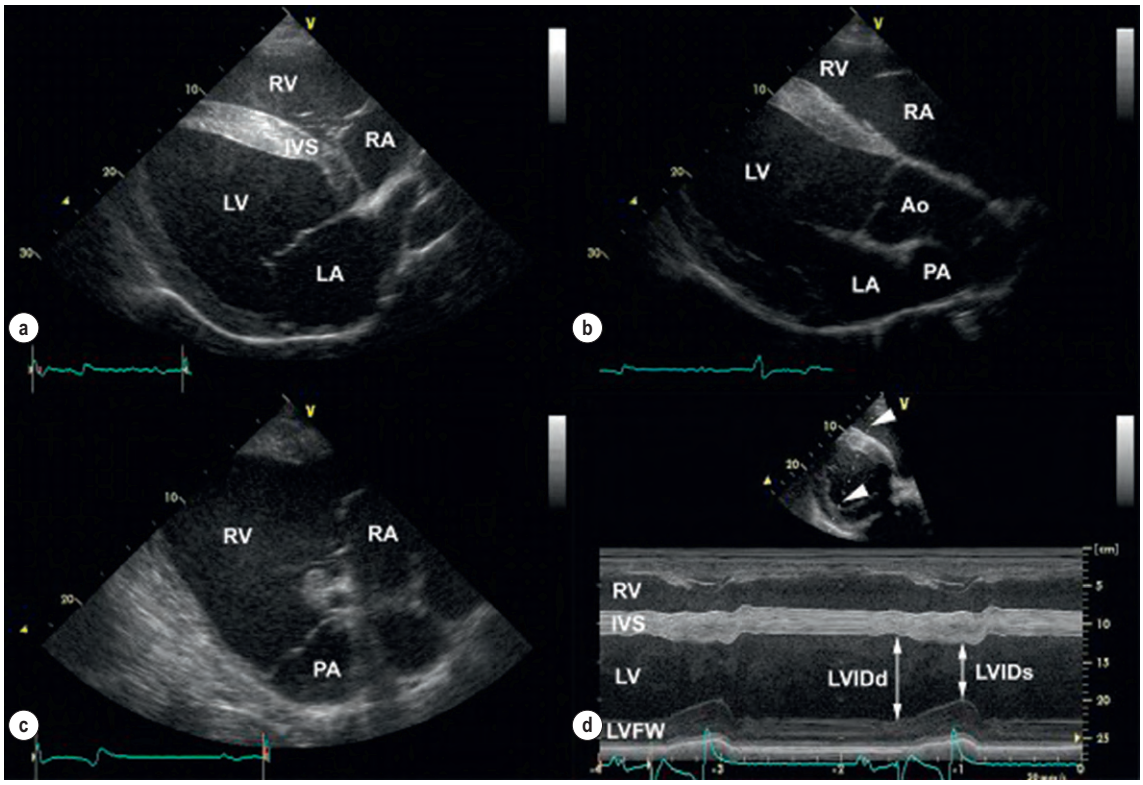


Figure 7.3 Two-dimensional echocardiograms obtained from the right side of the chest. An ECG is recorded simultaneously for timing. (a) Four-chamber view. (b) Left-ventricular outflow tract view. (c) Right-ventricular inflow and outflow tract view. These images, recorded in a right-parasternal long-axis view, allow subjective assessment of cardiac structures and myocardial function and measurement of selected cardiac dimensions. (d) M-mode echocardiogram of the normal left ventricle performed in a right-parasternal short-axis view at the level of the chordae tendineae. The myocardial wall motion (y-axis) along the cursor line (arrowheads) is displayed over time (x-axis). An ECG is recorded simultaneously for timing. This view allows subjective assessment of right- and left-ventricular dimensions and left-ventricular systolic function, measurement of the left-ventricular internal dimensions in systole and diastole, and calculation of the left-ventricular fractional shortening (% change in the internal dimension). The latter provides an index of systolic left-ventricular function. LV = left ventricle; LA, left atrium; RV, right ventricle; RA, right atrium; IVS, interventricular septum; Ao = aorta; PA = pulmonary artery; LVFW = left-ventricular free wall; LVIDd = left-ventricular internal diameter at end-diastole; LVIDs = left-ventricular internal diameter at peak-systole.
Published in Schwarzwald CC, Bonagura JD, Muir WW (2009) The cardiovascular system. In: Muir WW (ed) Equine anesthesia – monitoring and emergency therapy, 2nd edn, Saunders, p 73, reprinted with permission of Elsevier Ltd.

after exercise and are usually not of a concern. However, persistent or more severe cardiac rhythm disturbances indicate cardiac disease and warrant further investigation.

Clinical laboratory tests

- Complete blood count and plasma fibrinogen concentration serve to identify anaemia and signs of inflammation that may indicate inflammatory or infectious cardiovascular disease (e.g. endocarditis, pericarditis, thrombophlebitis).
- Serum electrolyte concentrations aid in the assessment of cardiac arrhythmias that can be caused by or associated with electrolyte disturbances (e.g. hypo- or hyperkalaemia, hypomagnesaemia). Assessment of fractional excretion of electrolytes in the urine may provide additional information on electrolyte homeostasis.
- Indices of renal function (blood urea nitrogen and creatinine concentrations) are important for assessment of volume status and detection of impaired renal perfusion in patients with compromised cardiovascular function.
- Elevated serum concentrations of unspecific muscle enzymes (creatine kinase [CK], aspartate transaminase [AST]) and cardiac-specific enzymes (cardiac troponin T [cTnT], cardiac troponin I [cTnI]) allow diagnosis of myocardial cell injury.
- Blood lactate concentration as well as arterial and venous blood gas analyses, respectively, serve to evaluate arterial oxygenation and pulmonary function, acid-base status, and – with limitations – oxygen delivery and oxygen utilization in the tissues.
- Arterial blood gas analyses may also be useful in the diagnosis of intracardiac shunts. A normal partial pressure of oxygen in arterial blood (p_aO_2) should be >65 mmHg in foals 24 hours *post natum* and >90 mmHg in older foals and in adults. Hypoxaemia can be caused by right-to-left shunting across a cardiac or vascular defect, pulmonary hypoperfusion, or respiratory disease. Intranasal supplementation of oxygen will not increase the p_aO_2 in horses with shunts, but may improve arterial oxygenation in horses with respiratory disease.
- Pericardial effusates should be collected and submitted for cytological examination, bacterial cultures, and antibiotic sensitivity testing.
- Blood cultures are recommended in cases of thrombophlebitis or suspected endocarditis to diagnose bacteraemia, identify the aetiologic agents, and determine the antibiotic sensitivity pattern.
- Monitoring of serum or plasma drug concentrations are indicated when certain drugs (e.g. quinidine, digoxin) are used to treat patients with heart disease.

Arterial blood pressure

- Arterial blood pressure can be measured invasively (by means of arterial catheterization) or non-invasively (using a variety of auscultatory, Doppler, or oscillometric techniques).
- Blood pressure monitoring is a routine procedure during general anaesthesia in horses. Furthermore, blood pressure measurement is commonly used for assessment of cardiovascular function in critically ill neonates.
- Arterial blood pressure monitoring includes measurement of systolic, diastolic, and mean pressures.
- Arterial pressure depends on the interplay between cardiac output and vascular resistance. Therefore, arterial pressure is not a reliable index of blood flow if vascular resistance (vascular tone) is abnormal or is changing over time.
- Normal reported values for indirect arterial systolic and diastolic pressures are 111.8 ± 13.3 (mean ± SD) and 67.7 ± 13.8, respectively.
- The systolic pressure is generated by the left ventricle and is affected by stroke volume, aortic/arterial compliance, and the previous diastolic blood pressure.
- Arterial pulse pressure, the difference between systolic and diastolic pressures, is highly dependent on stroke volume and the peripheral arteriolar resistance, which determines the run-off of diastolic pressure. The pulse pressure is the primary determinant of the intensity of the palpable peripheral arterial pulse.
- Ventricular failure reduces pulse pressure (hypokinetic, weak pulse), whereas increased stroke volume and abnormal diastolic run-off due to aortic insufficiency or generalized vasodilation widens the pulse pressure (hyperkinetic, bounding, or water-hammer type pulse).
- Diastolic and mean pressures are better estimates of perfusion pressure than systolic pressures.

Radiography

- The diagnostic value of radiography in horses with cardiovascular disease is limited.
- While gross cardiac enlargement can be detected on thoracic radiographs, echocardiography is better suited for accurate measurement of chamber size, assessment of chamber function, and detection of structural cardiac or vascular lesions.
- Thoracic radiographs are primarily used to assess pulmonary structures and can be used in the differential diagnosis of respiratory disease and for detection of cardiogenic pulmonary congestion or oedema, respectively.
- Angiographic methods (i.e. contrast radiography and contrast computed tomography) and in the future also cardiac magnetic resonance imaging may be used to assess heart size and heart function and to diagnose congenital heart disease in foals. However, the application of these methods is generally limited to research or to cases where echocardiography is inconclusive or unavailable.

Cardiac catheterization

- Cardiac catheterization serves to measure intracardiac pressures, cardiac output, and vascular resistance and to assess oxygenation of blood in the various cardiac chambers.
- It can be used to assess central haemodynamics as well as systolic and diastolic cardiac function. Furthermore, the results may demonstrate shunting of blood between the systemic and the pulmonic circulation that may occur in association with congenital cardiac defects.
- Cardiac catheterization is rarely used for routine diagnosis of cardiovascular disease in horses and has largely been replaced by Doppler echocardiography for clinical purposes. Its use is generally limited to research applications.
- Occasionally, cardiac catheterization is performed in critically ill foals and in selected cases for cardiovascular monitoring during anaesthesia.

7.3 STRUCTURAL HEART DISEASE

Congenital malformations

- Approximately 3% of all horses presented for a cardiovascular workup are diagnosed with congenital heart disease.
- Congenital heart disease should be considered when a foal, weanling, or young horse is identified with a prominent cardiac murmur, cyanosis, or signs of congestive heart failure.
  - Ventricular septal defects (VSD) are most frequently seen. They are usually located in the membranous part of the ventricular septum, just below the aortic and the tricuspid valve (paramembranous VSD). Rarely, VSDs are located in the subpulmonic position (subpulmonic VSD) or in the muscular septum (muscular VSD). VSDs can also be part of complex malformations.
  - Patent ductus arteriosus (PDA) is uncommon in foals and is detected most frequently in combination with complex defects. However, functional closure of the ductus arteriosus may not occur until 72 to 96 hours after birth, resulting in a murmur that can be heard up to an age of four days.
  - Other congenital defects (e.g. atrial septal defect, patent foramen ovale, valvular dysplasia or atresia) are relatively rare. Complex congenital defects usually lead to fetal death, birth of a non-viable foal, or rapid deterioration early after birth.

Ventricular septal defect (VSD)

- Ventricular septal defects result in systemic-to-pulmonary (left-to-right) shunting of blood. The shunt volume and the resulting haemodynamic sequelae depend on the size of the lesion and the resistances of the systemic and the pulmonary circulation, respectively.
  - Immediately after birth, pulmonary vascular resistance is still high and systemic pressures are low, limiting left-to-right shunting. Over the first few weeks of life, significant shunting may develop due to a gradual decline in pulmonary vascular resistance and rise in systemic pressures.
  - Left-to-right shunt increases pulmonary blood flow and pulmonary venous return to the left heart and results in compensatory left atrial and left ventricular enlargement. With large shunt volumes, left-sided volume overload may be severe, leading to left-sided or biventricular congestive heart failure.
  - Pulmonary hypertension can occur due to increased transpulmonary flow and flow-related pulmonary vascular changes. A rise in pulmonary vascular and right ventricular pressures decreases the shunt volume and protects the left heart from severe volume overload while imposing an increased work load on the right ventricle. Rarely, severe pulmonary hypertension may result in reversed (right-to-left) shunt with development of arterial hypoxemia (Eisenmenger's physiology).
- The clinical presentation varies depending on the size of the defect and shunt volume.

  - Foals and adults with small defects are often asymptomatic. Murmurs may be detected incidentally during examination for another problem or during prepurchase examination.
  - Some horses may be asymptomatic at rest but may show signs of exercise intolerance or poor performance at the time when they enter training.
  - Foals with large defects may die early in life or may present with signs of poor growth or congestive heart failure (often misinterpreted as signs of pneumonia) at the age of a few weeks or months.
  - Cyanosis is not a typical feature of VSDs, because the shunt direction is usually left-to-right. If cyanosis is detected, complex malformations or shunt reversal should be considered.
- The typical heart murmur associated with a membranous VSD is a band-shaped, holosystolic murmur heard best over the right side of the chest, just below the tricuspid valve. Murmurs are often prominent (grade ≥3/6) and may be associated with a palpable thrill (grade ≥5/6).
  - The grade of the murmur does not correlate with the size of the defect or the shunt volume.
  - Other murmurs that may be heard are left-sided systolic murmurs of relative pulmonic stenosis (functional ejections murmurs), left-sided systolic murmurs of mitral regurgitation (due to mitral insufficiency secondary to severe left-ventricular volume overload), or left-sided diastolic murmurs of aortic insufficiency (due to involvement of the aortic valve annulus, resulting in valve prolapse).
- Echocardiographic studies usually confirm the lesion. In combination with work history and physical examination, 2D and Doppler echocardiography further allow assessment of haemodynamic consequences, severity of the malformation, and prognosis.
  - Ventricular septal defects are termed restrictive when their diameter is less than one third of the diameter of the aorta (<2.5 cm in large-breed adult horses) and the peak velocity of the shunt flow is higher than 4 m/s (corresponding to a left-to-right pressure gradient of at least 64 mmHg).
  - Restrictive VSDs are usually well tolerated and have a favourable prognosis for life and potentially even for athletic performance.
  - Horses with an excellent work history are unlikely to have a large defect.
  - Non-restrictive VSDs (diameter >2.5 cm, shunt flow velocity <4 m/s) are usually associated with a guarded to poor prognosis in regards to athletic use and possibly even in regards to life.
  - Other echocardiograpic findings that indicate poor prognosis include subpulmonic position of the VSD, left-ventricular volume overload, aortic insufficiency due to involvement of the aortic valve annulus, mitral regurgitation due to severe left-ventricular dilation, signs of pulmonary hypertension, or association with complex malformations.
- Surgical closure of VSDs in horses is not possible. Horses with signs of congestive heart failure due to non-restrictive VSDs can be treated symptomatically (see below).
- Horses with uncomplicated, small VSDs can be exercised without restrictions.
- Most VSDs probably represent spontaneous malformations and are not inherited. However, the use of affected horses for breeding purposes should be discouraged, particularly in Arabian horses (due to the potential heritability of VSDs in this breed).

Acquired valvular heart disease

- Acquired valvular heart disease is common in middle-aged to older horses. It usually results from chronic, degenerative disease leading to fibrosis and focal or generalized thickening of the valves.
- Other causes include valve prolapse, non-bacterial inflammatory lesions (nonsuppurative valvulitis), bacterial endocarditis, and rupture of one or of several chordae tendineae at the atrio-ventricular valves (spontaneously or secondary to inflammatory or degenerative lesions).
  - Valvulitis is probably more common than previously thought and should be listed as a differential diagnosis if valvular regurgitation occurs in younger horses. Valvulitis is often thought to be associated with previous viral infections, although such a relationship is usually speculative. An immune-mediated pathogenesis is possible. Signs of systemic inflammation are usually absent at the time of diagnosis.
- Mitral regurgitation and aortic insufficiency are clinically most relevant.
- Tricuspid regurgitation and pulmonic insufficiency can be found in many apparently healthy horses and are often not of clinical relevance.
- Stenosis of heart valves is exceedingly rare in horses and usually only occurs in association with congenital defects.
- Factors that determine the clinical relevance of valvular insufficiency include the valve involved, the type of pathology, the degree of regurgitation, the severity of haemodynamic consequences, and the rate of progression.
  - Medical history (including work history and results from previous cardiac exams), clinical examination, and echocardiographic findings are important to assess the clinical relevance of valvular disease. Occasionally, electrocardiographic examination and exercise testing are necessary as well to judge the importance of a murmur.
  - Many apparently healthy horses demonstrate valvular regurgitation despite normal auscultation findings. These silent, usually trivial or mild valvular leaks are usually considered normal or at least clinically irrelevant, particularly if they are observed on the right side of the heart.
  - Even audible valvular regurgitation is not necessarily of clinical relevance, as many normal horses, particularly high-performance athletes, demonstrate murmurs of tricuspid, mitral, or aortic insufficiency.
- Clinically insignificant insufficiency is haemodynamically irrelevant and is unlikely to progress over time.
- Conversely, clinically significant valvular insufficiency places a haemodynamic burden on the heart and can lead to ventricular volume overload and congestive heart failure over time.
  - In early stages of disease, the cardiovascular system is able to compensate for the volume overload and consequences of valvular insufficiency are well tolerated.
  - Over time, progression of valvular insufficiency may cause clinical signs of exercise intolerance or poor performance. Often, clinical signs will not occur until ventricular dysfunction is advanced.
  - Eventually, myocardial dysfunction and congestive heart failure may ensue from chronic volume overload.
- The distinction between clinically irrelevant and clinically important valvular insufficiencies is often difficult and may necessitate repeated examinations over a prolonged period of time (i.e. months to years) in order to detect a potential progression of disease.

Mitral regurgitation (MR)

- Next to the aortic valve, the mitral valve is the most common site for acquired valvular disease in horses.
  - Degenerative, fibrotic lesions are probably the basis for most cases of MR.
  - Other causes for MR include valve prolapse (often associated with mild, clinically insignificant MR), nonsuppurative inflammatory lesions (valvulitis; often suspected in younger horses), bacterial endocarditis, rupture of the chordae tendineae, dilatation of the valve annulus in association with left ventricular enlargement (e.g. secondary to aortic insufficiency or dilated cardiomyopathy), or papillary muscle dysfunction (e.g. due to primary or ischaemic myocardial disease). Acute papillary muscle ischemia may result in transient MR that can resolve within a few days.
- Clinical signs of MR vary depending on the cause, the degree of regurgitation, and the resulting haemodynamic effects.
  - At early stages of disease, mitral regurgitation is usually asymptomatic and may be detected as an incidental finding.
  - Trace or mild MR may be seen on echocardiography in many apparently normal horses, including young horses in athletic condition. These findings may not be clinically relevant, even if associated with an audible murmur.
  - Clinically significant MR is often associated with exercise intolerance and poor performance.
  - Increased left-atrial pressures and left-atrial dilation may predispose to atrial fibrillation and may lead to development of pulmonary hypertension.
  - Pulmonary hypertension may be associated with exercise induced pulmonary haemorrhage. Furthermore, pulmonary hypertension leads to dilation of the pulmonary artery. In severe cases, pulmonary artery rupture can ensue, leading to collapse and sudden death.
  - Acute, severe MR can result in left-sided heart failure, associated with clinical signs of pulmonary congestion and oedema.
  - Advanced chronic MR usually leads to biventricular heart failure, associated with clinical signs of predominantly right-sided failure (i.e. jugular vein distention and pulsation, peripheral oedema).
  - MR due to bacterial endocarditis is often associated with fever, weight loss, and signs of inflammation and systemic disease (e.g. polyarthritis, leukocytosis, hyperfibrinogenaemia).
- The characteristic murmur associated with MR is a grade 2–6/6, holosystolic murmur heard best over the left apical and the mitral valve area (Fig. 7.1), radiating caudodorsally.
  - Occasionally, the murmur may be a mid-systolic murmur, and it can be either coarse or blowing.
  - The grade of the murmur is not related to the severity of regurgitation.
  - Mid-systolic clicks may indicate mitral valve prolapse.
  - In severe MR, the third heart sound may be accentuated due to increased ventricular filling pressures.
- Confirmation of diagnosis and assessment of clinical significance of MR requires complete cardiovascular workup including echocardiography and potentially other ancillary diagnostic methods.
- Findings on echocardiography may include mild to moderate valve thickening consistent with degeneration or valvulitis; valve prolapse; irregular thickening, shortening, or calcification consistent with endocarditis; or flail leaflets (chaotic fluttering of a mitral structure) consistent with chordal rupture.
  - Enlargement and rounding of the LV indicates severe MR and LV volume overload.
  - Increased or normal global LV function (fractional shortening) indicates normal myocardial function in face of a reduced afterload (due to MR).
  - Decreased global LV function indicates myocardial dysfunction due to chronic volume overload or primary cardiomyopathy.
  - Enlargement of the left atrium is consistent with haemodynamically significant MR and may predispose to atrial fibrillation or pulmonary hypertension.
  - Dilation of the pulmonary artery indicates pulmonary hypertension.
- Treatment options for MR are limited in horses.
  - Asymptomatic, chronic MR is usually not treated.
  - Angiotensin converting enzyme inhibitors (e.g. enalapril, ramipril, quinapril) are occasionally used in horses with symptomatic MR in order to decrease volume retention, reduce afterload, and slow progression of disease. However, the pharmacology of ACE inhibitors in horses has not been fully investigated and their efficacy is unknown.
  - Horses with acute or severe, chronic MR may benefit from afterload reduction using an arterial dilator (e.g. acepromazine, hydralazine). Afterload reduction reduces the mitral regurgitant volume and increases forward stroke volume.
  - Horses with congestive heart failure should be treated accordingly (see below).
  - Horses suffering from bacterial endocarditis should be treated aggressively (see below).
- The prognosis of MR depends on severity and progression of disease. In most cases, it may not be possible to determine the long-term prognosis based on a single examination.
  - Mitral regurgitation is the most important valvular disease in horses leading to exercise intolerance and poor performance.
  - Horses with trace or mild MR can often perform to the owner's expectations and show slow progression of disease. Horses may remain asymptomatic for many years. However, tolerance of MR depends on the level of work, and affected horses may not be able to perform at higher levels of exercise.
  - Generally, MR due to degenerative valve disease can be expected to progress gradually, as long as the heart size is normal.
  - Horses with severe left-atrial dilation and atrial fibrillation are likely to progress into congestive heart failure.
  - Horses with severe valvular thickening, chordal rupture, bacterial endocarditis, and dilated cardiomyopathy carry a poor prognosis. Affected horses should not be exercised.
  - Signs of severe left-ventricular volume overload, poor global left-ventricular function, and clinical signs of congestive heart failure indicate a poor prognosis. These horses should not be exercised.
  - Horses with a severely dilated pulmonary artery are at risk of pulmonary artery rupture and sudden death. These horses should not be exercised.
  - Progression of disease is difficult to predict. Horses with mild, moderate, or severe MR should be re-examined every 6 to 12 months to assess the rate of progression. Also, the owner should be advised to monitor for development of clinical signs consistent with progression of MR. High-performance horses may need to undergo exercise testing.

Aortic insufficiency (AI)

- Aortic insufficiency is considered the most common valve disease in middle-aged to older horses (>10 years).

  - The most common cause for AI in this age group is degenerative valve disease.
  - However, echocardiographic studies often reveal trivial or mild aortic insufficiency without obvious valve pathology and without audible murmur in horses of all age groups. Idiopathic valvular prolapse may be detected in some of these horses.
  - In younger horses (<10 years), AI may also be caused by local inflammatory lesions (nonsuppurative valvulitis).
  - Severe valvular prolapse may accompany ventricular septal defects. Acute tears of one of the aortic cusps may occur spontaneously or secondary to severe valve prolapse, resulting in severe aortic insufficiency (flail cusp).
  - Aortic insufficiency may also be the result of large vegetative lesions due to bacterial endocarditis.
- Over time, aortic insufficiency may lead to left-ventricular dilation.
  - In early stages of disease, the increase in end-diastolic volume results in an increase in ventricular contractility and stroke volume (Frank-Starling mechanism).
  - In advanced stages of severe AI, myocardial failure can ensue, leading to a decrease in stroke volume and eventually to signs of left heart failure.
  - Severe left-ventricular enlargement can result in mitral regurgitation due to dilation of the mitral valve annulus. Affected horses are more likely to progress to congestive heart failure.
- The clinical signs vary depending on the severity of regurgitation.
  - Most horses with AI appear asymptomatic. In these horses, AI is usually detected as an incidental finding during routine physical examination. Frequently, mild AI is silent and can only be detected by Doppler echocardiographic studies.
  - Advanced disease may lead to signs of poor performance, left heart failure, or biventricular failure. However, compared to mitral regurgitation, AI is less likely to cause poor performance.
  - Horses with bacterial endocarditis usually present with fever, weight loss, and signs of systemic disease.
- The quality of the peripheral arterial pulses may serve as an indicator of severity of disease. Therefore, palpation of peripheral pulses is mandatory in horses with AI.
  - Trace or mild AI does not noticeably alter pulse quality. Arterial pulses should be strong and regular.
  - More severe AI may lead to an increase in pulse pressure (due to the increased stroke volume and rapid run-off of blood during diastole) that results in a very strong, palpable pulse, also referred to as hyperkinetic pulse, bounding pulse, or 'waterhammer pulse'. The presence of a hyperkinetic pulse indicates that the AI is clinically relevant and likely associated with left-ventricular dilation.
  - Advanced disease may lead to progressive myocardial failure, causing a decrease in stroke volume (despite the increase in preload). In these cases, the peripheral pulse is usually faint and the heart rate is increased.
- The typical heart murmur associated with AI is a holodiastolic or (occasionally) early diastolic, decrescendo, grade 2–6/6 murmur heard best over the left cardiac base (Fig. 7.1).
  - Murmurs of AI are often very characteristic and can be described as musical, cooing, buzzing, creaking, groaning, or coarse.
  - The severity of regurgitation does not correlate with the grade or the duration of the murmur.
  - In advanced cases, an accompanying murmur of mitral regurgitation may be heard.
- Echocardiography serves to confirm the diagnosis and (together with clinical findings) to assess severity and clinical relevance of disease.
  - Focal or generalized thickening of the aortic valve and valve prolapse are common findings on echocardiography.
  - Other findings include oscillations of the leaflets, flail leaflets, aortic root prolapse into a VSD, dilation of the aortic root, or aortic sinus aneurism.
  - Left-ventricular enlargement is common in horses with moderate to severe AI. Concurrent increase of the left-ventricular shortening fraction indicates adequate adaptation of systolic function to volume overload. Conversely, a decrease in left-ventricular shortening fraction is interpreted as a sign of poor systolic function due to myocardial failure.
  - Concurrent mitral regurgitation and left-atrial enlargement are considered signs of advanced disease.
- Ventricular arrhythmias may develop as a result of left ventricular enlargement. In fact, horses with AI more commonly develop ventricular arrhythmias compared to horses with other valve diseases.
  - 24-hour Holter ECG or exercise ECG may be necessary to identify these arrhythmias and to determine if the horse remains safe to ride.
- Similar to the treatment of MR, treatment options for AI are limited.
  - Most horses with AI do not require treatment.
  - Angiotensin converting enzyme inhibitors are occasionally used in horses with AI (see MR).
  - Horses with congestive heart failure should be treated accordingly (see below).
  - Horses suffering from bacterial endocarditis should be treated aggressively (see below).
- The prognosis of AI depends on severity and progression of disease.
  - Asymptomatic horses may have a good prognosis for life and for athletic use. Mild aortic insufficiency may progress slowly and may remain asymptomatic for many years. Nevertheless, repeated examinations (including echocardiography) every 12 to 24 months are recommended to monitor potential progression of disease.
  - Poor prognostic indicators include persistent tachycardia, hyperkinetic or weak peripheral pulses, severe left-ventricular enlargement with decreased systolic function, concurrent mitral regurgitation and left-atrial enlargement, flail cusps, signs of endocarditis, signs of congestive heart failure, and the presence of frequent ventricular arrhythmias.

Tricuspid regurgitation (TR)

- Tricuspid regurgitation is common in horses, particularly in racehorses in training.
- It is frequently diagnosed on auscultation or as an incidental finding on echocardiography.
- The incidence of TR is known to increase with age and training, probably due to training-related changes of the right ventricular geometry (athletic heart) and increases in pulmonary artery pressures.
- Despite the high incidence of TR, pathologic alterations of the tricuspid valve are rare.
  - Degenerative valve disease or valvulitis may affect the tricuspid valve.
  - Tricuspid regurgitation can develop secondary to pulmonary hypertension, either as a result of left-sided heart failure or as a consequence of primary lung pathology (cor pulmonale).

  - Bacterial endocarditis, sometimes complicated with rupture of a chorda tendinea, can develop in association with jugular vein thrombosis or thrombophlebitis.
- Tricuspid regurgitation is characterized by a holosystolic, grade 2–6/6 heart murmur that is heard best over the right hemithorax, in the tricuspid valve area (Figure 7.1). Murmurs detected as an incidental finding during routine examinations are often grade 2–3/6. The murmurs can be coarse or blowing.
- Affected horses are usually asymptomatic. Rarely, clinically significant TR may cause signs of exercise intolerance and poor performance, right-sided heart failure, or atrial fibrillation (associated with atrial enlargement).
- Horses with right-sided bacterial endocarditis usually present with fever and signs of jugular vein thrombosis or thrombophlebitis.
- Confirmation of the diagnosis and rule-out of a VSD (as the main differential diagnosis of right-sided systolic murmurs) is achieved by means of transthoracic echocardiography.
- Treatment of tricuspid regurgitation is usually not necessary, unless signs of congestive heart failure are present (see below).
  - Primary lung pathology leading to pulmonary hypertension should be treated as necessary.
  - Bacterial endocarditis and thrombophlebitis should be treated as described below.
- The prognosis is generally favourable in horses with trace or mild TR if they have no evidence of valvular pathology or mild valve prolapse and if they do not show signs of right-sided cardiomegaly, pulmonary hypertension, or atrial fibrillation. Re-evaluation may be indicated every 2 years to monitor potential progression of disease.
  - In all other horses, the prognosis depends on cause and severity of the regurgitation, as well as potential coexisting conditions (e.g. severe pulmonary disease, left-heart failure, thrombophlebitis). These horses should undergo more frequent re-evaluation.
  - Horses with endocarditis or ruptured chordae tendineae have a poor prognosis.

Pulmonic insufficiency (PI)

- The pulmonic valve is the least significant valve in regards to valvular heart disease.
- Pulmonic insufficiency is a common incidental finding on echocardiography. However, pulmonic valve pathology is rare, and most insufficiencies are trivial or mild.
- Clinically significant PI is usually associated with other cardiac lesions.
  - Pulmonary hypertension (due to left-ventricular failure or primary lung pathology) may lead to pulmonic insufficiency, often associated with pulmonary artery dilation.
  - Rarely, bacterial endocarditis can affect the pulmonic valve, usually in association with thrombophlebitis of the jugular vein.
  - Right-sided heart failure is rare, unless the PI is associated with other conditions.
- Even if detectable on echocardiography, mild to moderate pulmonic insufficiency is often silent. Rarely, severe PI causes a grade 2–6/6, holo-diastolic or early-diastolic, decrescendo murmur that can be heard loudest over the pulmonic valve area (Figure 7.1).
- The prognosis is usually good for horses with trivial, mild, or moderate PI, if no concurrent disease is present and no valve pathology is found on echocardiography.
- The prognosis for horses with severe PI and right-sided heart failure is poor.

Endocarditis

- Endocarditis is rare, but can affect horses of any age.
- It is caused by bacterial infections of the valves, chordae tendineae, papillary muscles, or (rarely) the ventricular walls.
  - Numerous bacteria have been identified, most commonly *Streptococcus* spp., *Actinobacillus* spp. and *Pasteurella* spp.
  - Mitral and aortic valves are most commonly affected. Vegetative lesions usually lead to valve insufficiencies and in severe cases to valve stenoses.
  - Tricuspid and pulmonic valves may be affected secondary to septic jugular vein thrombosis or thrombophlebitis.
  - Infected chordae tendineae can rupture, resulting in acute and often severe valvular regurgitation.
- Endocarditis is a systemic disease. Hence, the clinical signs are not limited to signs of cardiovascular disease, but may include signs of predisposing disease, systemic inflammation, immune-mediated host responses, metastatic infection, or thrombo-embolic complications.
  - Predisposing conditions include jugular vein thrombosis or thrombophlebitis, strangles, septic arthritis, osteomyelitis, or abscesses. However, most horses have no history of previous illness or concurrent infection.
  - Clinical signs may include therapy-resistant or recurrent fever, depression, inappetence, weight loss, polysynovitis, and intermittent lameness.
  - Tachycardia and signs of heart failure may be present.
  - Heart murmurs are common, but the absence of audible murmurs does not rule out endocarditis.
  - Supraventricular or ventricular arrhythmia may be observed.
- One should consider endocarditis in any horse with new occurrence of a cardiac murmur in association with systemic disease and therapy-resistant or recurrent fever.
  - Diagnosis is made by echocardiography. Usually, prominent, hyperechoic, irregular masses can be identified at the affected, usually thickened valves.
  - Laboratory analyses indicate systemic inflammation. Results may include mild anaemia, leukocytosis, neutrophilia, and elevated plasma fibrinogen concentration.
  - Blood cultures are best collected during febrile episodes. They allow identification of the causative agent and facilitate effective antibiotic treatment. Negative culture results do not rule out bacterial endocarditis.
- Endocarditis should be treated early and aggressively. Treatment aims at eliminating the causative agent, reducing the growth of the vegetations, reducing the risk of embolization of infected thrombi, and limiting valve damage.
  - Initial treatment should include broad-spectrum, bactericidal antibiotics (preferably parenteral), non-steroidal anti-inflammatory drugs, and antithrombotics (e.g. heparin, acetylsalicylic acid).
  - Later, the antibiotic treatment should be adjusted based on culture results and sensitivity testing.
  - Response to antibiotic treatment may be delayed or absent due to a lack of penetration of antibiotics into the large, avascular vegetations.
  - Long-term antibiotic treatment is necessary. Generally, treatment should be continued for 4 to 8 weeks or until body temperature, plasma fibrinogen concentration, and leukocyte counts have been normal for at least 2 weeks.

- The prognosis depends on the degree of valve damage, the resulting structural and functional cardiac changes, the severity of the associated systemic disease, and the response to treatment.
  - Although the infection and inflammation may be treated successfully, permanent valve damage and scarring may result in persisting valvular regurgitation that may progress to heart failure.
  - The prognosis is fair for horses with small vegetations and mild valvular regurgitation that respond well to antibiotic treatment.
  - The prognosis is poor for horses with very large vegetations, ruptured chordae tendineae, progressive cardiomegaly, or congestive heart failure.

Pericardial disease

- Pericardial disease is rare in horses and usually manifests as fibrinous pericarditis, pericardial effusion, or constrictive pericarditis.
- Pericardial disease can be idiopathic or is caused by bacterial or viral infections, trauma, tumours, or immune-mediated conditions.
- Pathophysiologically, pericardial disease is usually characterized by compression of the heart caused by fluid accumulation within the pericardial space (cardiac tamponade) or constriction of the heart by abnormally rigid epicardium or thickened pericardium (constrictive pericarditis).
  - Both conditions impair diastolic filling of the ventricles and reduce preload and cardiac output.
  - Although all four chambers are affected, the right atrium and right ventricle are more susceptible to the increasing pressure within the pericardial space because of the lower pressures within the right side of the circulation.
  - Acute pericardial effusion with cardiac tamponade reduces cardiac output and arterial blood pressures and may lead to weakness, collapse, syncope, or sudden death.
  - Chronic pericardial effusion and tamponade causes activation of the sympathetic nervous system and the renin-angiotensin-aldosterone system, leading to tachycardia, vasoconstriction, and volume retention that initially serve to maintain blood pressures and improve cardiac pump function. Eventually these compensatory mechanisms are detrimental and result in congestive heart failure.
- Development of clinical signs depends on the volume and rate of fluid accumulation, compliance of the pericardium, and aetiology of the effusion.
  - The clinical signs may include exercise intolerance, depression, fever, inappetence, weight loss, jugular vein distension, and ventral oedema.
  - Cardiac auscultation may reveal tachycardia, muffled heart sounds, and pericardial friction rubs (see above).
  - On a surface ECG, the heart rate is usually fast and regular. The amplitude of the complexes may be decreased and variable (electrical alternans). The latter may be observed if the effusion is large and the heart is swinging within the pericardial space.
  - Echocardiography serves to confirm the presence of effusion, fibrin, and thickening of the pericardium. Often, right atrial collapse and impaired right ventricular filling can be observed. Effusion is minimal or absent with constrictive pericarditis.
  - Laboratory abnormalities often include anaemia, hyperfibrinogenaemia, and leukocytosis.
  - Cytologic evaluation of the pericardial effusion serves to distinguish septic, non-septic, or neoplastic conditions.
  - Bacterial culture and sensitivity testing of the aspirated fluid should be performed in cases of septic pericarditis.
- Bradycardia, peripheral vasodilation, myocardial depression, and decrease in venous return may cause significant haemodynamic deterioration and hypotension. Therefore, sedatives and anaesthetics should be used with caution.
- Early and aggressive treatment is important and should not be delayed.
  - Horses with cardiac tamponade should be stabilized by means of pericardiocentesis. Pericardiocentesis is usually performed with ultrasonographic guidance under local anaesthesia and often results in dramatic improvement of cardiac output and pressures.
  - Furosemide is generally contraindicated prior to pericardiocentesis, as diuresis may lower filling pressures, reduce ventricular filling, decrease cardiac output, and result in syncope.
  - Severe hypotension should be treated with shock-doses of crystalline or colloid fluids to increase intravascular volume and to improve diastolic ventricular filling. Catecholamines (dobutamine) may be beneficial to increase myocardial contractility.
  - Subsequent tube or surgical drainage of the effusion with once or twice daily lavage and instillation of antibiotics should be considered.
  - Initially, broad-spectrum antibiotics should be used to treat infection. The antibiotic regimen should be adapted according to the results of bacterial cultures and antibiotic sensitivity patterns.
  - Non-steroidal anti-inflammatory drugs are indicated to fight inflammation and to reduce the risk of constrictive pericarditis. In general, steroids should be reserved for cases where infectious causes can be ruled out.
  - Local and systemic anticoagulation (e.g. heparin) may be considered to reduce development of fibrin deposits.
  - For constrictive pericarditis, surgical removal of the pericardium (pericardiectomy) under general anaesthesia may be considered.
- The prognosis for effusive pericarditis is fair if aggressive treatment is initiated early in the disease process.
  - Idiopathic (non-septic) pericarditis generally has a better prognosis compared to bacterial (septic) pericarditis, which is more likely to result in constrictive or fibrotic pericardial disease.
- The prognosis is guarded to poor for neoplastic disease or constrictive pericarditis (despite aggressive treatment and pericardiectomy).

Myocardial disease

- Myocardial disease may be more common than generally thought.
- Diagnosis of myocardial disease is often difficult and many cases may go unrecognized.
- The aetiology of myocardial disease often remains unknown.
- Primary myocardial diseases include dilated or infiltrative cardiomyopathy, ischaemia, myocardial infarction, myocardial degeneration and necrosis (eventually leading to fibrosis), bacterial or viral myocarditis, neoplasia, and trauma.
  - The cause of dilated cardiomyopathy is unknown. The diagnosis is made by exclusion of valvular, vascular, and congenital diseases that may cause ventricular enlargement and poor systolic function.
  - Infiltrative cardiomyopathy rarely occurs due to neoplasia (e.g. melanoma, lymphoma) or amyloidosis.

  - Bacterial or viral myocarditis may be associated with respiratory infections (e.g. influenza, *Streptococcus* spp.). An immune-mediated pathogenesis is possible.
  - Myocardial degeneration and necrosis can result from acute ischaemia, thrombo-embolic events, or vitamine E/selenium deficiency (white muscle disease). Myocardial damage has also been described in cases of atypical myopathy. Furthermore, a variety of toxins, including ionophores (e.g. monensin, salinomycin), cantharidin (blister beetle), plant toxins, or rattlesnake venom may lead to myocardial degeneration.
  - Focal myocardial fibrosis (probably due to ischaemia or necrosis) is a common incidental finding on post mortem examinations.
- Myocardial depression may occur due to hypoxia, electrolyte disturbances, acidosis, septicaemia, endotoxaemia, mild (transient) ischaemia, administration of sedatives and anaesthetics, chronic volume overload, pericarditis, or chronic tachycardia.
- Myocardial diseases may result in a decreased stroke volume and cardiac output due to:
  - Ventricular systolic (contraction) and/or diastolic (relaxation, filling) dysfunction.
  - Secondary insufficiencies of the atrio-ventricular valves due to papillary muscle dysfunction and dilation of the valve annulus.
  - Cardiac arrhythmias due to disturbances in impulse formation and/or impulse conduction.
- The onset of clinical signs may lag behind the initial insult.
  - Some horses do not develop detectable clinical signs at all.
  - Others may demonstrate poor performance, signs of heart failure, arrhythmias, collapse, or sudden death.
  - Prolonged recovery after exercise, tachycardia, tachypnoea, arrhythmias, and heart murmurs may be identified on physical examination.
- Diagnosis is often based on exclusion of other cardiac or extracardiac diseases and identification of potential causes of primary and secondary myocardial disease.
  - Laboratory analyses are helpful to identify myocardial disease. Particularly, increases in serum *c*TnT or *c*TnI concentrations indicate myocardial damage. However, normal values do not preclude myocardial disease.
  - Resting ECG, exercise ECG, and 24-hour Holter ECG serve to identify cardiac rhythm disturbances.
  - Echocardiography serves to rule out primary valvular disease, pericardial disease, and vascular disease. Structural abnormalities (e.g. areas of abnormal echogenicity), chamber enlargement, poor global ventricular function, or regional wall motion abnormalities may be identified.
  - Endomyocardial biopsy would be necessary to provide a final diagnosis. However, it is impractical in clinical cases and may not reveal focal disease.
- Therapy of myocardial disease is primarily supportive.
  - Horses with suspected or diagnosed myocardial disease should be stall rested or turned out on pasture.
  - Administration of corticosteroids may be considered if an infectious aetiology is unlikely.
  - Vitamin E and selenium should be administered to horses with suspected deficiency.
  - Congestive heart failure and cardiac arrhythmias should be treated as discussed below.
- The prognosis depends on the causes and the severity of myocardial damage and the resulting haemodynamic alterations.
  - The prognosis is guarded to poor with congestive heart failure or life-threatening arrhythmias (e.g. polymorphic ventricular tachycardia).
  - Some horses with acute congestive heart failure due to myocarditis may recover over time.

7.4 HEART FAILURE

- Heart failure is a clinical syndrome resulting from an acute or chronic impairment of cardiac pump function to a degree where systemic perfusion is inadequate to meet the metabolic demands of the tissues.
- Heart failure can result from any structural or functional cardiac disease. Most commonly it is caused by advanced valvular heart disease.
- In systolic heart failure, cardiac contractility is reduced. Causes of systolic heart failure include advanced valvular heart disease, myocardial disease, and congenital heart disease. Right ventricular systolic dysfunction most commonly is a consequence of left ventricular systolic dysfunction. It may also develop as a result of pulmonary hypertension or chronic, severe tricuspid regurgitation.
- In diastolic heart failure, cardiac relaxation and ventricular filling are impaired. Causes of diastolic heart failure include pericardial constriction or cardiac tamponade as well as restrictive, infiltrative, hypertrophic, and nutritional cardiomyopathies.
- The failing heart is characterized by a poor myocardial contractility, a decrease of preload reserve (diminished response to volume loading), and an increased sensitivity to ventricular afterload (wall tension, primarily determined by ventricular geometry and systemic blood pressures).
- The pathophysiology of heart failure is complex and affects many body systems. Most importantly, inadequate organ perfusion leads to an activation of multiple neurohumoral pathways.
- Decreased stimulation of baroreceptors results in activation of the sympathetic nervous system that increases heart rate and myocardial contractility, causes vasoconstriction in non-vital tissues, and stimulates the release of renin from the kidneys.
- Sympathetic activation and decreased renal perfusion result in activation of the renin-angiotensin-aldosterone system, causing systemic vasoconstriction as well as sodium and water retention.
- Other hormones released in heart failure include vasopressin (antidiuretic hormone) and endothelin.
- Initially, these neurohumoral mechanisms are compensatory and beneficial. Increase in heart rate, contractility, and circulating blood volume allow for support of cardiac pump function, while arteriolar vasoconstriction results in redistribution of blood flow to vital organs.
- However, chronic neurohumoral activation is responsible for progression of disease and results in exacerbation of clinical signs and deterioration of heart failure.
  - Continued sodium and water retention lead to volume overload, resulting in congestion of the lungs and the systemic organs (congestive heart failure).
  - Catecholamines exert proarrhythmic effects, promote myocardial remodelling, and are toxic to myocytes.
  - Aldosterone causes endothelial dysfunction and myocardial fibrosis.
  - Pro-inflammatory cytokines contribute to weight loss and apoptotic cell loss in myocardial tissues.
  - The left ventricle undergoes remodelling consisting of left ventricular dilation and hypertrophy. While the resulting increase in stroke volume is compensatory, the associated increase in wall tension worsens myocardial perfusion and

may provoke myocardial ischaemia. Furthermore, ventricular dilation may result in worsening of mitral regurgitation leading to pulmonary congestion.

- Signs of heart failure vary depending on the stage of disease.
  - Heart failure may develop suddenly (e.g. chordal rupture, acute papillary muscle ischemia) or gradually (e.g. degenerative valve disease).
  - Exercise intolerance, poor performance, and delayed recovery after exercise may be early signs of heart failure.
  - Strenuous work, anaemia, fever, or pregnancy increase the demands for cardiac output and may precipitate heart failure in a previously compensated patient.
  - At advanced stages, clinical signs of heart failure can become evident at rest and may be associated with weight loss and loss of body condition.
  - Congestive heart failure is characterized by volume overload, fluid accumulation, elevation of venous pressures, and congestion. It is uncommon in horses, as most cardiac lesions are not severe enough to lead to renal retention of sodium and water.
  - Left-ventricular failure, as might accompany mitral or aortic regurgitation, causes pulmonary venous congestion and pulmonary oedema. Clinical signs include tachycardia, weak pulses, tachypnoea, increase respiratory effort at rest or after exercise, nasal discharge, cough, and abnormal lung sounds (crackles, wheezes) on auscultation. The lung oedema of pure left-sided congestive heart failure is frequently misdiagnosed as pneumonia.
  - Signs of right-sided congestive heart failure can be due to tricuspid regurgitation, pericardial disease, or severe lung disease. Furthermore, right-ventricular dilation and failure commonly develop secondary to pulmonary venous hypertension and interstitial lung oedema that results from primary (chronic, progressive) left-sided heart disease. Typical clinical features of right-ventricular or biventricular heart failure include distension of superficial veins, jugular venous pulsation, and subcutaneous ventral oedema.
- Clinical detection of congestive heart failure should prompt the veterinarian to perform a complete cardiovascular workup in order to identify the cause of heart failure.
- Therapy of congestive heart failure is only realistic for potentially reversible diseases (e.g. acute, transient atrioventricular valve insufficiency resulting from papillary muscle ischemia, pericarditis, myocarditis, sustained ventricular tachycardia). Euthanasia is often elected because of the poor prognosis at the time of diagnosis.
  - The goals of therapy are reduction of congestion and oedema, improvement of tissue perfusion, and inhibition of deleterious neurohumoral activation.
  - If possible, the underlying disease should be treated (e.g. pericardiocentesis and drainage of pericardial effusion).
- Drug therapy for chronic heart failure in horses is often limited to furosemide and digoxin.
  - Furosemide (0.5 to 3.0 mg/kg SC, IM, or IV q8–12 hours as needed to produce diuretic effect; or 0.12 mg/kg IV, followed by 0.12 mg/kg/h CRI) is used to reduce congestion and oedema.
  - Digoxin (0.011 mg/kg q12 hours PO) is used to increase myocardial contractility and slow ventricular response rate in patients with rapid atrial fibrillation.

    Doses and dose intervals are titrated to effect. Renal function, electrolyte status, and serum digoxin concentrations should be monitored periodically.

    Digoxin peak (1–2 hours) and trough (12 hours) concentrations at steady state should fall within 0.8–2.0 ng/mL (1–2.6 nmol/L).

    Digoxin toxicity includes depression, anorexia, colic, diarrhoea, sinus bradycardia, AV block as well as supraventricular and ventricular arrhythmias (e.g. bigeminy).
- Acute heart failure or acute worsening of chronic heart failure may be treated with diuretics (furosemide), positive inotropes (dopamine, dobutamine), and intranasal oxygen.
- Antiarrhythmics may be indicated for treatment of severe arrhythmias. However, most of them should be used with caution due to their proarrhythmic and cardio-depressant effects.
- Acepromazine (0.02–0.06 mg/kg IM q8h or 0.5–1.5 mg/kg PO q12h) can be given for its anxiolytic and sedative effects. It also has some antiarrhythmic action and causes vasodilation, thereby reducing afterload.
- Vasodilators such as the arterial dilator hydralazine or the venodilator nitroglycerin may be used to reduce pulmonary congestion, lower ventricular afterload, and improve tissue perfusion. However, data on efficacy and safety of these drugs in horses with heart failure are lacking.
- Angiotensin-converting enzyme (ACE) inhibitors decrease renal sodium and water reabsorption, reduce volume overload, counteract the development of diuretic resistance, cause vasodilation with a decrease in afterload, decrease in myocardial oxygen demand, and are considered cardioprotective by decreasing myocardial remodelling and fibrosis. Enalapril, ramipril, and quinapril have been used in horses with moderate to severe aortic or mitral regurgitation as well as horses in congestive heart failure. However, the pharmacology of ACE inhibitors in horses has not been fully investigated and the efficacy for prophylaxis and treatment of heart failure is unknown.
- In humans, the use of β-adrenergic inhibitors and aldosterone antagonists has been shown to reverse some of the detrimental effects of chronic neurohumoral stimulation and to decrease mortality in heart failure. In horses, the use of these agents in heart failure has not been described to date.

7.5 CARDIAC RHYTHM DISTURBANCES

Cardiac arrhythmias are common in horses at rest, during and after exercise, and under general anaesthesia. Cardiac rhythm disturbances can usually be heard on auscultation and can be reliably diagnosed using surface electrocardiography (ECG), telemetric ECG (during exercise), or 24-hour Holter ECG (for detection of paroxysmal arrhythmias). The equine veterinarian must be familiar with the basic aspects of ECG in order to diagnose the most common physiologic and pathologic arrhythmias, determine their potential causes, assess their clinical relevance, and choose the appropriate treatment.

- Generally, cardiac arrhythmias are caused by disturbances in impulse formation or impulse conduction.
- Arrhythmias can be grouped on the basis of their site of origin and according to the resulting heart rate.
  - Supraventricular arrhythmias originate above the AV node, junctional arrhythmias originate in the bundle of His, and ventricular arrhythmias originate in the ventricles.
  - Bradyarrhythmias are characterized by a slow heart rate, while tachyarrhythmias are characterized by a fast heart rate.
- A number of physiological cardiac arrhythmias have been recognized in horses, most importantly second-degree AV block, sinus arrhythmia, and sinus arrest.

- The most important pathological arrhythmias include atrial fibrillation, atrial and ventricular premature complexes, and accelerated idioventricular rhythms.
- Of principal concern to the equine clinician are the haemodynamic consequences of arrhythmias (i.e. hypotension, decrease in cardiac output, poor peripheral perfusion) and the potential for further electrical destabilization (i.e. myocardial fibrillation).

Physiological arrhythmias

- Physiological arrhythmias can be explained by the impact of the autonomic nervous system on the sinoatrial and atrioventricular nodes. Normal horses at rest demonstrate a high vagal tone, leading to a low resting heart rate that is often associated with second-degree AV block, sinus arrhythmia, or sinus block/arrest.
- Fear or sudden stimuli may provoke rapid withdrawal of vagal tone and sympathetic activation, abolishing vagally mediated arrhythmias and resulting in sinus tachycardia. Therefore, physiologic arrhythmias can usually be abolished during light exercise (spinning round, jogging, lunging, riding), stress, or with vagolytic drugs such as atropine or glycopyrrolate.

First and second degree atrioventricular (AV) block

- Delays of impulse conduction at the atrioventricular (AV) node are classified as first, second, and third degree (or complete) AV block. Second degree AV block is the most common physiological arrhythmia in horses. Third degree AV block is considered pathological and will be discussed below.
- First-degree AV block occurs when the PQ interval exceeds the upper normal limit while the atrial impulse still transmits through the AV conduction system and activates the ventricle, causing a normal QRS-T complex on the ECG.
  - Diagnosis of first-degree AV block is difficult by auscultation, although a prolongation of the interval between the fourth (S4) and first (S1) heart sound may be heard.
- During second-degree AV block, some P waves are not conducted to the ventricles, resulting in occasional P waves not followed by a QRS-T complex on the ECG. The underlying rhythm is usually a normal sinus rhythm (Figure 7.4).
  - Auscultatory findings include a regular underlying rhythm with intermittent pauses during which the fourth heart sound (S4) may be audible.
  - Up to one in three beats may be blocked in normal horses.
  - Occasionally, two or more consecutive second-degree AV blocks are observed in presence of a normal or slow sino-atrial rate.
  - Second-degree AV block following progressive prolongation of the PQ interval is classified as Mobitz type I (Wenckebach) block (Figure 7.4). Conversely, the AV block is termed Mobitz type II if the PQ interval is constant.
- First- and second-degree AV block are considered normal variations in the horse. These rhythms are most often associated with high vagal tone and may be seen in normal, resting horses with a normal or low-normal heart rate. Furthermore, second-degree AV block can be seen during the recovery phase immediately after exercise (so called phase of autonomic imbalance) or in an anxious horse after a brief surge of sinus tachycardia.
- If second-degree AV block persists despite excitement, exercise, or vagolytic drugs, structural AV node disease should be suspected. Persistent high-grade second-degree AV block may progress into complete AV block over time.

Sinus bradycardia, sinus arrhythmia, sinus tachycardia

- Sinus rhythms are characterized by the presence of normal P-QRS-T complexes, with a P wave for every QRS complex and a QRS complex for every P wave.
- Atrioventricular conduction, generally, tends to follow sinus activity: During sinus tachycardia, AV nodal conduction is facilitated and the PQ interval shortens. During periods of progressive sinoatrial slowing, AV nodal conduction is inhibited, the PQ interval prolongs, and second-degree AV block may occur.
- Sinus arrhythmia is less common in resting horses, but may occur during the recovery phase immediately after exercise or in an anxious horse after a brief surge of sinus tachycardia.
  - On ECG, a gradual variation in PP and RR intervals can be identified. Slight changes in the conformation of the P wave can be seen from beat to beat due to the fact that impulse formation occurs in different areas of the sinoatrial node (wandering pacemaker).
  - On auscultation, a gradual variation in heart rate can be heard.
- Sinus bradycardia is generally a benign rhythm in standing horses; however, when very pronounced, sinus bradycardia or sinus arrest may decrease cardiac output and cause significant hypotension.
  - Treatment of symptomatic sinus bradycardia can include reduction of anaesthetic concentration (when bradycardia develops under general anaesthesia), administration of anticholinergic drugs (atropine, glycopyrrolate), or infusion of catecholamines (dopamine, dobutamine, epinephrine).
- Sinus tachycardia is usually a physiological response to excitement, stress, and pain or it occurs as a result of hypotension, hypovolaemia, hypercarbia, hypoxaemia, anaemia, endotoxaemia, anaphylaxis, or excessive catecholamine administration.
  - Specific therapy for sinus tachycardia is rarely required. The underlying cause of sinus tachycardia must be managed as appropriate.

Sinoatrial (SA) block, sinus arrest

- Sinoatrial block and sinus arrest is considered physiological in horses and is often seen in conjunction with sinus bradycardia and AV block.

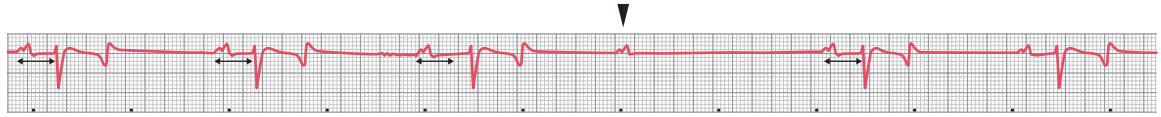


Figure 7.4 Base–apex lead ECG recorded from a horse with second degree AV block, characterized by a single P wave that is not followed by a QRS-T complex (*arrowhead*). The AV block is classified as Mobitz type I (Wenckebach type) due to the prolongation of the PQ interval prior to the AV block. The horizontal arrows are of equal length and indicate the normal PQ interval (paper speed 25 mm/sec, voltage calibration 0.5 cm/mV).

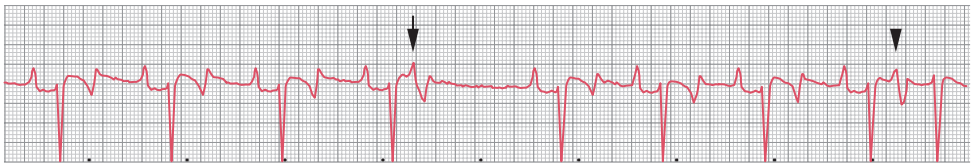


Figure 7.5 ECG recorded from a horse with atrial premature complexes. The premature P′ waves occur earlier than expected and appear superimposed on the preceding T wave. On the first occasion (*arrow*), the premature impulse arrives at the AV node before it has completely repolarized. Therefore, the P′ wave is blocked and not followed by a QRS complex. On the second occasion (*arrowhead*), P′ is followed by a normal QRS-T complex because impulse is able to enter the ventricles in a normal manner. Note that the PQ interval appears slightly prolonged, indicating that the AV node has not recovered completely from the preceding depolarization (base–apex lead, paper speed 25 mm/sec, voltage calibration 1.0 cm/mV).

- On auscultation, the regular underlying (sinus) rhythm is interrupted by intermittent pauses. Unlike with second-degree AV block, a fourth heart sound cannot be heard.
- Electrocardiographically it is characterized by a normal sinus rhythm interrupted by occasional pauses without detectable P-QRS-T activity.
- The differentiation between sinus arrest (sinus node does not discharge) and sinoatrial block (sinus node discharges but impulse is not conducted to the atrial tissue) is difficult based on a surface ECG and is clinically irrelevant.
- High-grade or persistent sinus arrest may indicate sino-atrial nodal disease.

Pathological arrhythmias

- Pathological arrhythmias can occur in association with valvular, myocardial, or pericardial disease.
- They can also occur without any obvious (echocardiographic) signs of structural cardiac disease, possibly due to microstructural or functional alterations of the myocardium or the conduction system.
- Pathological arrhythmias often develop in association with high sympathetic or high vagal tone, fever, endotoxaemia, sepsis, hypotension, acid–base and electrolyte disorders, hypoxaemia, anaemia, gastrointestinal disease, or severe pulmonary disease.
- Proarrhythmic drugs (e.g. catecholamines, digoxin, and most antiarrhythmic drugs), agents known to sensitize the heart to catecholamines (e.g. thiobarbiturates, halothane), and toxic substances (e.g. ionophore antibiotics) have to be considered as aetiologic agents.
- Primary heart disease should be suspected if other signs of heart disease are present and non-cardiac causes of arrhythmias can be ruled out.

Atrial premature complexes and atrial tachycardia

- Rhythms originating in the atria are common in the horse.
- Mitral or tricuspid insufficiency resulting in atrial enlargement predispose to atrial arrhythmias.
- Single premature impulses that arise within the atria, but outside of the sinoatrial node, are designated atrial premature complexes (APC). Atrial tachycardia is defined as a sequence of four or more atrial premature complexes.
- On auscultation, APCs can be heard as heart beats that occur early (premature) relative to the previous beat.
- On ECG, the premature P wave (often referred to as P′) occurs earlier than expected based on the previous P–P intervals, and it often differs from the normal P wave in size and morphology. It is followed by a relatively normal QRS-T complex because the impulse usually enters the ventricles in a normal manner (Figure 7.5).
- If the premature impulse occurs early in diastole and arrives at the AV node before that tissue has completely repolarized, the P′ wave is nonconducted (blocked) and therefore not followed by a QRS complex (Figure 7.5).
- Occasional isolated atrial premature complexes are usually well tolerated and are sometimes considered a normal variation if they occur infrequently (i.e. less than 1 premature beat per hour or less than 5 in 24 hours).
- Atrial premature complexes can be detected in up to 20% of clinically normal racehorses during the warm-up and/or recovery phase. They are considered insignificant, as long as they are isolated, occur infrequently, and do not develop during exercise.
- The frequent occurrence of atrial premature complexes may precede the development of atrial fibrillation. Also, APCs are frequently detected after conversion of atrial fibrillation to normal sinus rhythm.
- Sustained atrial tachyarrhythmias are abnormal and indicate heart disease. Rapid or repetitive atrial arrhythmias are most likely to decrease cardiac output at high heart rates and can be haemodynamically important, causing exercise intolerance, hypotension, syncope, or congestive heart failure.
- Treatment should be based on identification and elimination of underlying causes.
  - Specific antiarrhythmic treatment is rarely indicated, unless the arrhythmia is severe and leads to clinically significant haemodynamic derangements. Treatment options include quinidine, procainamide, and propranolol.
  - Rapid ventricular response rate may be controlled by administration of digoxin, diltiazem, or a beta-adrenergic blocker.
  - Suspected viral or immune-mediated myocarditis may be treated with corticosteroids and rest.

Atrial fibrillation

- In athletic horses, AF is commonly associated with exercise intolerance or poor performance. Conversely, pasture or pleasure horses often do not show any clinical signs.
- Horses are thought to be predisposed to AF due to the large atria and the high resting vagal tone, both of which are factors that increase the likelihood of reentrant arrhythmias.
  - Atrial premature complexes may act as triggers of AF.
  - Electrolyte abnormalities (particularly hypokalaemia and hypomagnesaemia) are thought to play some role in development of AF.
- The term 'lone AF' refers to AF occurring in the absence of any detectable underlying cardiac disease (although there may well be some predisposing microstructural or functional risk factors that cannot be easily detected by routine diagnostic measures).
- Structural heart disease associated with atrial enlargement (e.g. mitral regurgitation, tricuspid regurgitation) and conditions

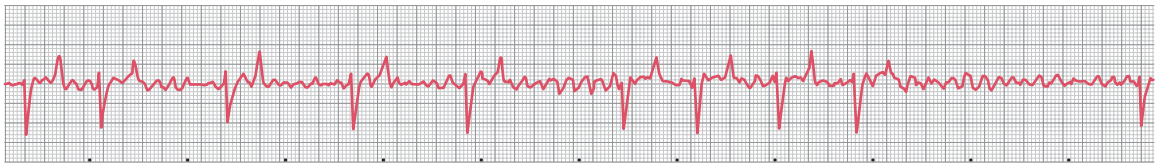


Figure 7.6 ECG recorded from a horse with atrial flutter/fibrillation. The arrhythmia is characterized by the absence of consistent P waves and undulations of the baseline. The fine, irregular undulations are termed fibrillation (f) waves while the more regular, saw-toothed waves are called flutter (F) waves (base–apex lead, paper speed 25 mm/sec, voltage calibration 1.0 cm/mV).

causing congestive heart failure increase the risk of AF. Conversely, sudden onset of AF may worsen clinical signs of heart failure in a previously compensated patient.

- Over time, AF itself may promote structural, electrical, and functional changes within the atrial myocardium that may contribute to the progressive, self-perpetuating, and recurrent nature of AF.
- Presentation of AF can be acute or chronic.
- Atrial fibrillation (AF) is the most common clinically relevant arrhythmia in horses.
  - Paroxysmal AF occurs suddenly, lasts from seconds to days, and ends spontaneously, often within 24 hours. It is most commonly seen in young racehorses or, occasionally, in horses with gastrointestinal disease.
  - Persistent AF only terminates after treatment.
  - Recurrent episodes of AF are not uncommon after successful treatment; they are more likely in the presence of concurrent structural or functional disease.
  - Permanent AF is resistant to therapy and is often associated with underlying cardiac disease.
- AF is characterized by a rapid atrial rate and a slower ventricular rate because many of the rapid atrial impulses are blocked in the AV node and do not enter the ventricles. The ventricular response is usually irregular.
- On auscultation, AF is characterized by an irregularly irregular rhythm, variable intensity of the first and second heart sound, and absence of the fourth heart sound.
  - AF can be differentiated from second degree AV block by auscultation. The latter is often characterized by a regular pattern (e.g. one block after every three normal beats) and an audible fourth heart sound.
  - The coexistence of audible heart murmurs (e.g. mitral regurgitation, tricuspid regurgitation) indicates possible underlying structural disease.
- Palpation of peripheral pulses reflects the findings on auscultation, revealing an irregularly irregular pulse with variable pulse strength.
- The finding of a pulse rate (counted by palpation) that is lower than the heart rate (counted by auscultation or on an ECG tracing) is referred to as pulse deficit. Pulse deficit occurs after a short R-R interval, when the time for ventricular filling is short and the stroke volume of the subsequent heart beat is too low to elicit a palpable pulse wave.
- In most horses, the heart rate (ventricular rate) during AF is within the normal range.
  - Tachycardia (rapid AF) can develop due to enhanced AV nodal conduction and indicates lower vagal activity and increased sympathetic tone.
  - When occurring at rest, tachycardia may indicate underlying cardiac disease or heart failure, although it can be seen in some horses due to stress, excitement, or during treatment with quinidine.
  - The ventricular response rate influences the haemodynamic effects of AF because ventricular filling is impaired at rapid, irregular rates.
- Electrocardiographically, AF is characterized by absence of P waves and irregular, often prominent undulations of the baseline (fibrillation or 'f' waves). The presence of more regular, saw-toothed waves (flutter or 'F' waves) in the baseline is referred to as atrial flutter. Occasionally, the electrocardiogram shows alternating atrial flutter and fibrillatory activity, a rhythm also referred to as 'atrial flutter/fibrillation' (Figure 7.6). From a clinical point of view, atrial flutter and atrial fibrillation can be regarded as a single type of arrhythmia in horses.
- Prior to treatment, horses with AF should be thoroughly examined to rule out underlying cardiac disease that could have an impact on treatment modalities, success of treatment, and outcome. Cardiac auscultation serves to identify heart murmurs, echocardiography can be used to detect chamber enlargement, serum biochemical analysis may reveal electrolyte abnormalities, and serum troponin concentrations are used to identify myocardial pathology.
- Horses kept as pets, breeding animals, or pleasure horses may not require antiarrhythmic treatment of uncomplicated AF, as the arrhythmia by itself is unlikely to impair quality of life or prevent normal use of the horses.
- The prognosis for successful treatment depends on the presence of underlying cardiac disease and (likely) on the duration of AF prior to treatment.
  - The prognosis is considered fair to good for young horses with uncomplicated, paroxysmal or persistent AF of short duration (i.e. <2 weeks).
  - In long-standing AF, the risk of treatment failure or recurrent AF is increased.
  - The overall prognosis is guarded when significant underlying cardiac disease or heart failure is present. Therefore, cardioversion is usually not attempted. Affected horses should not be exercised. Heart failure may be stabilized by administering digoxin and furosemide (see above).
- Prior to any antiarrhythmic treatment, electrolyte abnormalities (particularly hypokalaemia and hypomagnesaemia) should be corrected and suitable fluid volume replacement should be administered if necessary.
- Quinidine is the most commonly used antiarrhythmic drug for converting AF to sinus rhythm in horses without significant underlying cardiac disease.
  - The use of this drug is contraindicated when signs of severe underlying cardiac disease or heart failure are evident.
  - The reported efficacy of quinidine treatment ranges between 83 and 92%.
  - Quinidine sulfate is administered by nasogastric tube at a dose of 22 mg/kg q2 h until (1) conversion to sinus rhythm, (2) adverse or toxic effects occur, or (3) a total of 4 (to 6) doses have been administered.

- Quinidine has a narrow therapeutic index. Therapeutic plasma concentrations range between 2 and 5 µg/mL (6–15 µmol/L). Concentrations above 5 µg/mL (>15 µmol/L) are considered toxic. However, adverse effects may occur even at plasma quinidine concentrations within the therapeutic range.
- Plasma quinidine concentration should be measured if (1) conversion has not occurred 1 hour after the fourth dose, or (2) the patient exhibits adverse or toxic effects.
- Treatment intervals should be increased to every 6 hours if (1) plasma quinidine concentration is >4 µg/mL (12 µmol/L), or (2) after the 4th dose if concentrations cannot be measured.
- Treatment every 6 hours can be continued, until (1) conversion to sinus rhythm, (2) adverse or toxic effects occur, (3) a total cumulative dose of 80 to 90 g is reached.
- Continuous (telemetric) ECG recording is recommended for monitoring of heart rate, rhythm, and conduction times. A prolongation of the QRS duration of more then 25% compared to pre-treatment values is considered a sign of quinidine toxicity and should prompt termination of treatment.
- Serum electrolyte and creatinine concentrations should be monitored and adequate fluid intake must be ensured during prolonged quinidine treatment, because most horses will become inappetent during treatment.
- Common extracardiac adverse effects of quinidine treatment include depression, inappetence, nasal oedema, colic, and diarrhoea. In rare cases, paraphimosis, urticaria, convulsions, laminitis, and sudden death may occur.
- The most common cardiac adverse effect is acceleration of the ventricular response rate resulting in moderate to severe tachycardia. This effect may occur at therapeutic plasma quinidine concentrations and is observed in approximately half of all treated horses. If rate is below 100 beats/min and the horse is haemodynamically stable, treatment can be continued with close monitoring; if rate is above 100 beats/min, digoxin (0.0022 mg/kg IV, may repeat dose once) may be administered; if the rate is sustained in excess of 150 beats/min and/or pressures are poor, sodium bicarbonate (1 mEq/kg IV) should be administered in addition to digoxin in order to antagonize sodium-channel blocking effects of quinidine. Other options for rate control include diltiazem or propranolol (monitor ECG and direct blood pressures).
- Quinidine may induce systemic hypotension by inhibition of alpha-adrenergic receptors. Treatment of severe hypotension includes administration of polyionic fluids and phenylephrine (0.1–1 µg/kg/min CRI, titrate to effect).
- Quinidine may also exert proarrhythmic effects, resulting in ventricular tachycardia or torsades de pointes. Management of ventricular arrhythmias includes administration of lidocaine (0.25–0.5 mg/kg slow IV, repeat in 5–10 min, up to 1.5 mg/kg total dose) and magnesium sulphate (2–6 mg/kg/min IV to effect, up to a total dose of 55 (–100) mg/kg). Quinidine treatment should be discontinued.

- Transvenous electrical cardioversion has been recently introduced as a valuable alternative to quinidine treatment, especially for those animals in which quinidine is either ineffective or not well tolerated. However, compared to quinidine treatment, there is still limited experience with this technique. Furthermore, electrical cardioversion demands general anaesthesia and requires special equipment and expertise that, to date, is only available at some larger referral centres.
- Other potential treatment options for AF include procainamide, amiodarone, propafenone, and flecainide. The efficacy and safety of these agents in clinical cases have not been well established in horses with AF and routine use of these drugs is not recommended to date.
- Atrial contractile function, assessed by echocardiography, can be persistently depressed for several days after conversion. Although the clinical relevance of this finding is unknown, horses may not be able to perform at their normal level immediately after conversion and may be at a higher risk of recurrent AF. Therefore, it is recommended that horses are rested for the first 3 to 5 days after conversion.
- Recording of a 24-hour Holter ECG after successful treatment can serve to detect other arrhythmias. In particular, APCs are frequently seen after conversion to sinus rhythm.
  - A normal 24-hour ECG after conversion of uncomplicated AF indicates that the horse may return to work within 3 to 5 days after treatment.
  - If a significant number of APCs are detected after conversion (i.e. >5 APCs/24 hours), the horse should be rested and re-evaluated after 2 to 4 weeks.

Junctional and ventricular arrhythmias

- Cardiac arrhythmias that originate below the AV node are classified as junctional (bundle of His) or ventricular (ventricular conducting tissues or myocardium) arrhythmias.
- Determining the exact location of the abnormal impulse formation can be difficult, but may be achieved by careful inspection of the QRS complex.
  - Junctional impulses are more likely to result in a narrow, relatively normal-appearing QRS complex.
  - Complexes of ventricular origin, by contrast, are conducted abnormally and more slowly, resulting in a widened QRS, an abnormal QRS orientation, and abnormal T waves (Figure 7.7).
- The normal heart contains latent (subsidiary) cardiac pacemakers within the AV and ventricular specialized tissues. The activity of these potential pacemakers may become manifest during periods of sinus bradycardia or AV block, leading to single escape complexes or persistent escape rhythms.
  - Escape rhythms are characterized by slow ventricular rates, often between 15 and 25 beats/min.
  - Specific antiarrhythmic drug suppression of escape rhythms is generally not necessary and is contraindicated because these rhythms serve as rescue mechanisms for the heart. Instead, management of escape rhythms should be aimed at resolving the underlying cause of sinus bradycardia or AV block.
- Occasionally, the normal subsidiary pacemakers may be enhanced and discharge at a rate equal to or slightly above the sinoatrial rate (usually between 60 and 80 beats/min). The resulting rhythm is commonly referred to as accelerated idioventricular (idionodal) rhythm or slow ventricular tachycardia (Figure 7.7b).
  - Conditions that favour the development of accelerated idioventricular rhythms include gastrointestinal disease (possibly due to a combination of endotoxaemia, autonomic imbalance, acid–base disturbances, and electrolyte abnormalities) and administration of anaesthetics or catecholamines.
  - Idioventricular rhythms are often quite regular and may be misdiagnosed as sinus tachycardia on auscultation or on palpation of peripheral pulses.
  - Accelerated idioventricular rhythms generally are of little clinical significance and resolve spontaneously with treatment of the underlying conditions.

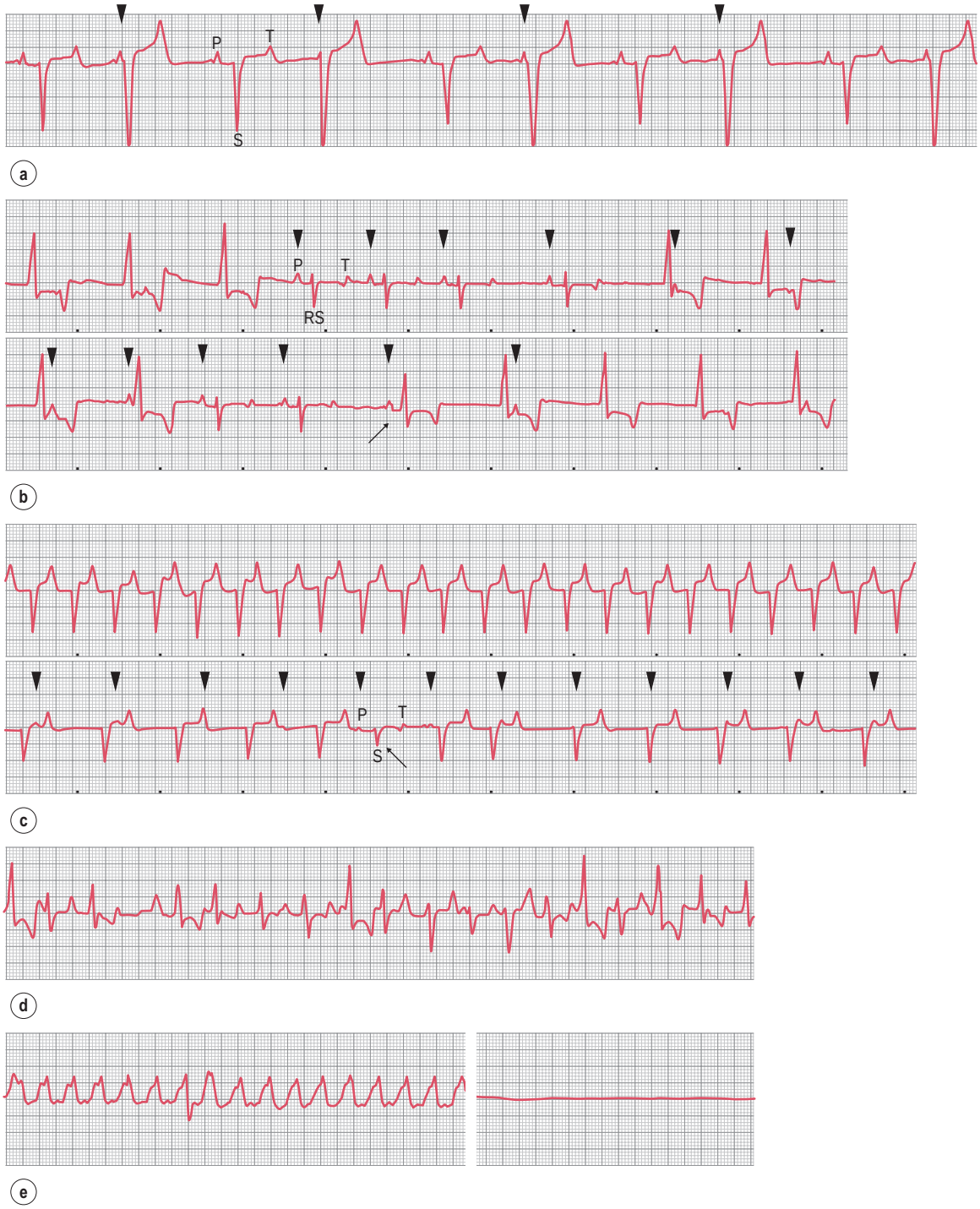


Figure 7.7 (a) Base–apex lead ECG recorded from a 15-year-old Arab mare with ventricular bigeminy. Normal sinus beats alternate with slightly larger and wider ventricular ectopic beats. SA node discharge is not affected by the ectopic beats, as indicated by the presence of non-conducted P waves immediately prior to the ectopic beats (*arrowheads*) (paper speed 25 mm/sec). (b) Base–apex lead ECG recorded from an 18-year-old Arab mare recovering from acute diarrhoea and endotoxaemia. The ECG shows an intermittent accelerated idioventricular rhythm at a rate of 50 beats/min. P wave intervals are indicated (*arrowheads*). The recording demonstrates that the ectopic focus is suppressed at higher rates of SA node discharge. The ventricular rhythm only becomes manifest when the sinoatrial rate drops below the rate of the ventricular pacemaker. SA node discharge is not affected by the ectopic rhythm, resulting in AV dissociation. A fusion beat is present (*arrow*), resulting from summation of a conducted sinus impulse with an ectopic ventricular beat (paper speed 25 mm/sec, voltage calibration 0.5 cm/mV). (c) Base–apex lead ECG recorded from a 3-year-old Clydesdale gelding. The top recording shows a regular tachycardia at a rate of 120 beats/min. The appearance of the QRS-T complexes does not allow conclusive distinction between a supraventricular rhythm with rapid ventricular response and a ventricular rhythm. However, as the rate slows down (bottom strip), AV dissociation due to a ventricular tachycardia becomes apparent. P waves (*arrowheads*) and a capture beat (*arrow*) are indicated (paper speed 25 mm/sec, voltage calibration 0.25 cm/mV). (d) Base–apex ECG recorded from a 5-year-old Clydesdale stallion with acute myocardial necrosis of unknown aetiology. The serum cardiac troponin I concentrations were severely elevated (404 ng/mL; normal <0.15 ng/mL). The ECG shows multiform ventricular tachycardia at a rate of 120 beats/min (paper speed 25 mm/sec, voltage calibration 0.5 cm/mV). (e) Lead II ECG recorded from a 3-week-old Danish Warmblood filly with botulism supported by mechanical ventilation. The rhythm (left) is consistent with ventricular flutter. Cardiac arrest occurred shortly after (right) despite attempts of resuscitation (paper speed 25 mm/sec).

Published in Schwarzwald CC, Bonagura JD, Muir WW (2009) The cardiovascular system. In: Muir WW (ed) Equine anesthesia – monitoring and emergency therapy, 2nd edn, Saunders, p 88, reprinted with permission of Elsevier Ltd.

  - Electrolyte supplementation (potassium, magnesium) and correction of fluid deficits and acid–base disturbances may be beneficial.
- Junctional and ventricular complexes that arise early relative to the next normal cardiac cycle are designated as premature junctional or ventricular complexes.
  - They are often associated with administration of drugs (e.g. catecholamines, digoxin, halothane), sympathetic stimulation, electrolyte disturbances (e.g. hypokalaemia, hypomagnesaemia), acid–base disorders, ischaemia, or inflammation.
  - Premature complexes may occur as single events, couplets (pairs), triplets, or short runs. A rhythm characterized by sinus beats immediately followed by premature ventricular beats is referred to as ventricular bigeminy (Figure 7.7a).
  - Repetitive ectopic complexes can either occur in short, self-terminating runs (nonsustained or paroxysmal ventricular tachycardias) or as sustained junctional and ventricular tachycardias (Figure 7.7c).
  - Ventricular tachycardias are referred to as uniform (monomorphic) if the QRS-T morphology of the ectopic beats is consistent throughout the recording, and as multiform (polymorphic) if two or more abnormal QRS-T configurations can be identified (Figure 7.7d). Torsades de pointes represent a specific form of polymorphic ventricular tachycardia, characterized by progressive changes in QRS direction leading to a steady undulation in the QRS axis.
  - Ventricular flutter and fibrillation are characterized by a chaotic ventricular activation pattern leading to uncoordinated undulations of the electrical baseline (Figure 7.7e).
- The clinical significance of occasional junctional or ventricular premature complexes in the horse is difficult to ascertain. They are usually considered abnormal in the horse, although isolated ectopic complexes may be more common than recognized from routine ECG studies.
- Generally, persistent or repetitive junctional or ventricular rhythms are indicative of heart disease, systemic disease, or a drug-induced abnormality of cardiac rhythm.
  - Ventricular tachycardia may be life threatening, if the arrhythmia is rapid (e.g. above 120 beats/min), multiform (polymorphic, including torsades de pointes), or characterized by a short coupling interval with R-on-T phenomenon (R-on-T refers to premature complexes occurring on the peak of the preceding T wave).
  - Ventricular tachycardia can progress into ventricular flutter or ventricular fibrillation, rhythms that commonly represent terminal events (Figure 7.7e).
- Junctional and ventricular arrhythmias usually cause interference with AV conduction of normal sinoatrial impulses while leaving atrial activation unaffected. The resulting (independent) coexistence of the sinoatrial activity (P wave) and the ectopic ventricular activity (QRS-T) is commonly referred to as AV dissociation (Figure 7.7b).
  - It is important to note that escape rhythms associated with sinus bradycardia or complete AV block also cause AV dissociation. Hence, 'AV dissociation' is a purely descriptive term of an ECG finding and neither characterizes the type and pathophysiological mechanism of the arrhythmia nor determines the therapeutic approach.
  - With sustained junctional or ventricular tachycardias, P waves may be identified but are not conducted and therefore not consistently associated with a QRS complex. Some P waves may be buried in the ectopic QRS-T complexes (especially at higher rates of ventricular activation), making their identification difficult.
  - The use of ECG calipers helps determining the P-P interval and can greatly facilitate the identification of P waves.
- Occasionally, atrial impulses may be normally conducted, leading to capture beats or fusion beats.
  - Capture beats are characterized by a normal P-QRS-T configuration, resulting from normal ventricular activation occurring before the ectopic focus discharges (Figure 7.7c).
  - Fusion beats are seen when both the conducted impulse and an ectopic impulse cause simultaneous ventricular activation. The QRS-T morphology of a fusion beat represents the summation of a normal and an ectopic beat (Figure 7.7b).
- Junctional and ventricular arrhythmias should be treated when there is evidence of hypotension, when premature complexes are frequent, or when the arrhythmias are life-threatening.
  - Oxygenation, electrolyte, and acid–base status should be re-evaluated, fluids should be infused to maintain blood pressure, and antiarrhythmic drugs should be administered when serious ventricular arrhythmias are recognized.
  - Lidocaine is commonly used as first-line treatment for junctional or ventricular arrhythmias (0.25–0.5 mg/kg slow IV, repeat in 5–10 min, up to 1.5 mg/kg total dose; followed by 0.05 mg/kg/min constant rate infusion). It is usually well tolerated, but dose recommendations should not be exceeded. At high doses, lidocaine may cause neurotoxicity and convulsions (treat with diazepam, 0.05 to 0.1 mg/kg intravenously).
  - Fluid therapy and especially maintenance of normal serum potassium concentration (4–5 mEq/L) is essential for antiarrhythmic therapy to be effective.
  - Magnesium supplementation (e.g. 25 to 150 mg/kg/day IV, diluted in polyionic isotonic solution) may also be beneficial. Therapeutic doses of magnesium are considered the treatment of choice for torsades de pointes.
  - Procainamide or quinidine gluconate may be used to treat ventricular tachyarrhythmias resistant to lidocaine and magnesium. Both can cause hypotension and reduced myocardial contractility. They must be given cautiously.
  - Generally, although acute intravenous antiarrhythmic therapy is possible, antiarrhythmic drugs may also cause considerable adverse (including proarrhythmic) effects and may only result in temporary conversion of ventricular tachycardia to normal sinus rhythm.
- The prognosis is usually favourable for infrequent, single ectopic beats in the absence of significant structural cardiac disease and for arrhythmias which can be attributed to a treatable non-cardiac disease.
- The prognosis for sustained junctional or ventricular tachycardia is usually guarded, particularly if there is evidence of significant structural heart disease or congestive heart failure.
- The prognosis for multiform ventricular tachycardia or torsades de pointes is usually guarded to poor.

Conduction disturbances

Once a cardiac electrical impulse is formed, it is conducted rapidly throughout the heart. The cardiac electric activation is usually dictated by the specialized conducting tissues in the atria, the AV node, the bundle of His, the bundle branches, and the Purkinje fibre system. This conduction system permits sequential activation of atrial and ventricular muscle and provides the basis for effective mechanical activity of the heart.

A variety of conduction disorders are recognized in horses, including SA nodal exit block, atrial standstill (usually due to hyperkalemia), AV block, and ventricular conduction disturbances.

- Third-degree or complete AV block is characterized by complete disruption of atrial to ventricular conduction, resulting in AV dissociation. To prevent ventricular asystole, a junctional or ventricular escape rhythm develops below the level of the AV block.
  - The resulting ventricular activity (manifested by QRS complexes) is considerably slower than the atrial activity (manifested by P waves), and P waves are not related to QRS complexes.
  - Complete AV block is usually persistent and indicative of organic heart disease or severe drug toxicity.
  - Life-threatening AV block and other bradyarrhythmias occasionally occur in horses or foals with severe metabolic diseases.
  - The clinical relevance depends, in part, on the basic heart rate or the rate of the escape pacemaker.
  - Treatment should include atropine or glycopyrrolate if hypotension develops. Dopamine or dobutamine may be needed if the former drugs are not successful. Sudden development of complete AV block may require administration of epinephrine or temporary pacing. Treatment of persistent complete AV block consists of implantation of a permanent transvenous pacing system.
- Intraventricular conduction disturbances or conduction blocks are less common in horses and more difficult to diagnose. Widening of the QRS complex and axis deviation are typical features. These abnormalities may also be found after atrial premature complexes, from overdosage with quinidine sulfate, or secondary to supraventricular tachycardias with rapid ventricular response.
- Hyperkalaemia most likely occurs in foals with uroperitoneum, in horses with acute renal failure and oliguria, during shock, after excessive intravenous potassium replacement, and in Quarter Horses with hyperkalaemic periodic paralysis (HYPP).
  - The cardiovascular manifestations of hyperkalaemia include significant depression of atrial, atrioventricular, and ventricular conduction and shortening of ventricular repolarization.
  - Changes in the ECG are usually evident at potassium serum concentrations greater than 6 mEq/L, with severe changes evident when serum concentrations are between 8 to 10 mEq/L (Figure 7.8).
  - Broadening and flattening of the P wave are the most consistently observed changes. Prolongation of the PQ interval and bradycardia develop, excitability decreases, and atrial standstill (sino-ventricular rhythm), characterized by complete absence of P waves, may be observed. Either inversion or enlargement (tenting) of the T waves is also likely. Marked widening of the QRS complex may be noted as near-lethal concentrations of potassium ion are approached. Ventricular asystole or fibrillation can develop.
  - Other electrolyte/acid–base alterations, including serum calcium and sodium, influence the effects of hyperkalaemia on the heart.
  - Therapy of hyperkalaemia includes correction of the underlying problem and administration of 0.9% NaCl, glucose, sodium bicarbonate (if severe acedaemia is present), and 23% calcium gluconate (for emergency treatment of life-threatening arrhythmia). Regular insulin with dextrose may be added to the treatment if the previous measures are unsuccessful. Administration of beta-2 sympathomimetics (e.g. salbutamol/albuterol by inhalation) may also aid in decreasing plasma potassium concentrations.

7.6 VASCULAR DISEASE

Jugular thrombophlebitis

- Jugular vein thrombosis or thrombophlebitis is common in horses.
- It usually occurs after intravenous (or peri-venous) injection of irritating or contaminated substances or as a complication of long-term jugular catheterization.
  - Horses with severe systemic disease associated with a hypercoagulable state (e.g. colic, endotoxaemia, septicaemia) are predisposed to jugular vein thrombosis or thrombophlebitis.
  - Catheter material, catheter placement procedures, management of indwelling catheters and fluid lines, as well as type and volume of injected substances are factors that influence the risk of jugular thrombophlebitis.
  - Aseptic placement of hyporeactive catheters (e.g. polyurethane catheters), adequate catheter management (i.e. regular change of catheter caps and fluid lines), aggressive treatment of predisposing conditions, and (potentially) prophylactic anti-inflammatory and antithrombotic treatment (e.g. heparin) reduce the risk of thrombophlebitis.
- Horses with sterile, partial jugular vein thrombosis may be asymptomatic. Partial thrombosis may not be detectable on palpation or may be felt as a firm, cylindrical mass within the jugular vein.
- Many horses present with enlarged jugular veins and swellings around the jugular groove.
- Complete thrombosis is usually obvious and leads to visible distension of the proximal part of the jugular vein and the superficial vessels of the head. Head oedema may develop during exercise and during grazing, particularly when both jugular veins are thrombosed.
- Septic jugular thrombophlebitis often leads to painful, warm swellings of the jugular groove and may be associated with fever, depression, inappetence, leukocytosis, and hyperfibrinogenaemia.
- Septic thrombophlebitis and/or periphlebitis may result in abscess formation.
- Ultrasonography is a sensitive tool to detect subclinical thrombosis. It can be used to assess the size of the thrombus and the degree of jugular vein obstruction, to detect residual or collateral blood flow (using Doppler imaging), and to demonstrate thickening of the vessel wall, perivenous oedema, or abscess formation. Cavitation of the thrombus suggests a septic process.
- With suspected septic thrombophlebitis, the thrombus and/or the altered perivenous tissue should be aspirated for bacterial culture and sensitivity testing. Blood cultures may be performed to reveal bacteraemia. If associated with an indwelling catheter, the catheter tip can be cultured.
- Management of thrombophlebitis should include topical and systemic treatment.
  - Hot compresses may be applied every 4 to 6 hours in the acute phase.
  - Topical treatment includes anti-inflammatory and anti-thrombotic ointments.

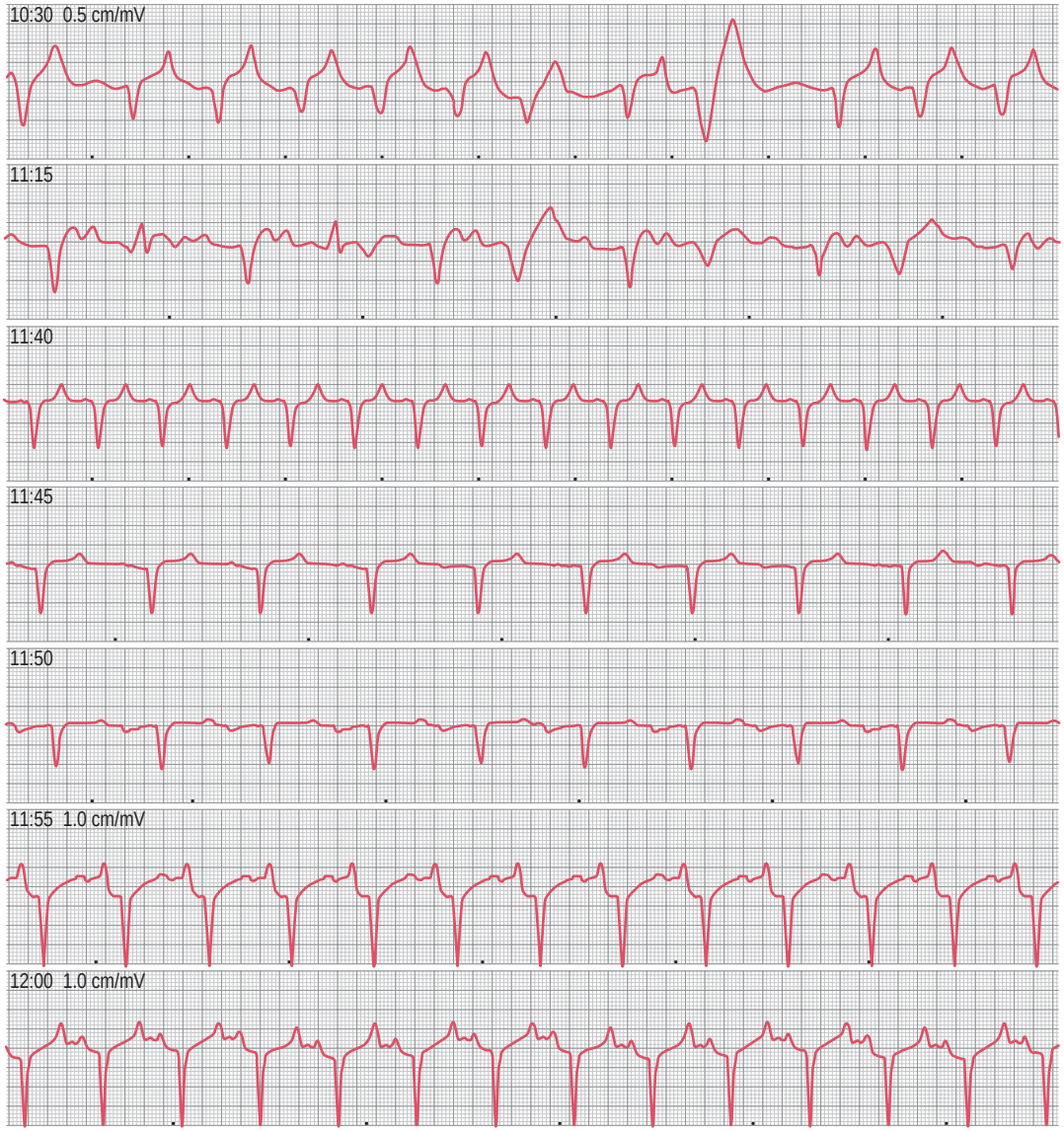


Figure 7.8 Hyperkalaemia causing atrial standstill, ventricular conduction disturbance, and ST-T abnormalities in a 4-day-old foal with acute renal failure. Serum potassium at admission (10:30) was 10.7 mEq/L (normal 2.4–4.6 mEq/L). The ECG is characterized by an absence of P waves, enlargement of T waves, and marked widening of the QRS complexes, indicating near-lethal concentrations of potassium. The following strips were obtained during therapy with intravenous fluids (NaCl 0.9%), calcium gluconate, dextrose, insulin, and peritoneal dialysis. Progressive normalization of the QRS complex and the T wave, as well as reappearance of the P wave, indicate normalization of atrial and ventricular conduction. The plasma potassium concentration at 11:30 was 7.9 mEq/L (base–apex lead, paper speed 50 mm/sec, voltage calibration 0.5 and 1.0 cm/mV as indicated).

- Systemic treatment includes broad-spectrum antibiotics (until bacterial cultures and sensitivity patterns are obtained), non-steroidal anti-inflammatory drugs, and anti-thrombotic agents (e.g. heparin, aspirin). Long-term treatment is often necessary.
- In horses with bilateral jugular thrombosis, the head should be tied up to reduce head oedema and upper airway obstruction.
- Surgical resection of the affected vein and drainage of adjacent abscesses may be indicated in severe cases. Autotransplantation of the saphenous vein may be considered after complete resolution of the infection.

- The prognosis for unilateral, non-septic jugular thrombophlebitis is usually good. Often, collateral circulation will develop and the vein may recanalize over time.
- Bilateral jugular vein thrombophlebitis is more likely to result in significant head oedema and upper airway obstruction that may cause significant dyspnoea or poor performance.
- The prognosis for septic thrombophlebitis is guarded. Treatment should be initiated very early in the disease process. Potential complications include local abscessation, bacterial endocarditis (occasionally associated with chordal rupture), pulmonary thromboembolism, and septicaemia.

Aorto-iliac thrombosis

- Aorto-iliac thrombosis is an uncommon but serious disease affecting the terminal aorta and the iliac arteries.

- It has been associated with parasite migration (*Strongylus vulgaris*) in the vessel walls, although a consistent aetiological relationship has not been proven.
- Partial or complete occlusion of the vessels can result in severe performance problems. The clinical features include:
  - Exercise-induced (usually unilateral) hind limb lameness, ataxia, or weakness.
  - Weak digital arterial pulses.
  - Decreased surface temperature, oedema, and pain at the affected limb (usually as a result of complete arterial occlusion).
  - Hyperpnoea, generalized sweating, and trembling of the affected limb in severe cases.
- Rectal examination may reveal a palpable thrill, weak or absent pulsation, or aneurysmal dilation of the affected arteries. These findings may be more prominent after exercise.
- Rectal ultrasonographic examination can be used to detect thrombosis and abnormal blood flow in the affected vessels.
- Early treatment is essential and includes non-steroidal anti-inflammatory drugs, long-term antithrombotic treatment (e.g. warfarin), larvicidal dewormings, and controlled exercise.
- The prognosis is usually guarded and no treatment has been shown to consistently improve outcome.

Vascular ruptures

- Vascular ruptures can be a cause of sudden death in exercising horses.
- Ruptures of the aortic root have been reported, most commonly in older breeding stallions.
  - Ruptures can occur into the ventricular septum, the right ventricle, the right atrium, or the pericardial space.
  - Possible underlying causes include degenerative changes in the aortic wall, aneurysms of the sinus of Valsalva, and hypertension associated with breeding, exercise, or the use of certain drugs (e.g. ketamine).
  - Aorto-cardiac fistulas are often associated with continuous heart murmurs.
  - Ruptures into the cardiac chambers or the pericardium can lead to heart failure.
  - Ruptures into the interventricular septum are often associated with ventricular arrhythmias.
  - Diagnosis is possible by two-dimensional and Doppler echocardiography.
  - The prognosis is generally poor, depending on the site and size of the fistula. Aortic ruptures are usually life-threatening and often fatal.
- Aortopulmonary fistulas have been described in Friesian horses, possibly due to a hereditary disorder.
- Pulmonary artery ruptures are usually the result of long-standing pulmonary hypertension and pulmonary artery dilation, either due to left-ventricular failure or primary lung disease.
  - Pulmonary artery rupture can lead to sudden death.
  - Horses with a severely dilated pulmonary artery, as detected on echocardiography, should be considered unsafe to exercise and should not be used for athletic activities.
- Rupture of the middle uterine artery (see 14.5) can lead to fatal haemorrhage in the postpartum period of older broodmares.
  - Affected mares can present with signs of severe colic or they can die suddenly.
  - Some mares may survive with intensive care and supportive therapy.
- Rupture of the internal carotid, external carotid, or maxillary arteries can occur secondary to vascular erosions associated with guttural pouch mycosis (see 5.6).
  - Affected horses may present with various degrees of nasal bleeding.
  - Severe, acute blood loss can lead to death before adequate treatment can be initiated.
  - Diagnosis can be easily made by endoscopic examination of the pharynx and/or the guttural pouches.
  - Due to the potentially life-threatening aetiology of the nasal haemorrhage, endoscopic examination of the airways, including the guttural pouches, should be regarded mandatory in all horses with epistaxis, independent of its severity.
  - The current treatment of choice consists of transarterial coil embolization of the affected arteries, performed by minimally invasive catheter procedure under general anaesthesia.
  - Medical treatment using antifungal drugs may be successful in some cases, but is not recommended in horses with active bleeding from the guttural pouches due to the risk of life-threatening, acute blood loss.

FURTHER READING

Berne RM, Levy MN (2001) Cardiovascular physiology, 8th edn. Mosby, St Louis

Bonagura JD, Reef VB, Schwarzwald CC (2010) Cardiovascular diseases. In: Reed SM, Bayly WM, Sellon DC (eds) Equine internal medicine, 3rd edn. Saunders Elsevier, St Louis, p 372

Durando MM (ed) (2003) Cardiovascular disease. In: Robinson NE (ed) Current therapy in equine medicine 5. Saunders, Philadelphia, p 572

Guyton AC, Hall JE (2006) III. The heart. In: Guyton AC, Hall JE (eds) Textbook of medical physiology, 11th edn. Saunders, Philadelphia, p 101

Guyton AC, Hall JE (2006) IV. The circulation. In: Guyton AC, Hall JE (eds) Textbook of medical physiology, 11th edn. Saunders, Philadelphia, p 159

Jesty SA, Reef VB (2008) Cardiovascular system. In: Orsini JA, Divers TJ (eds) Equine emergencies: Treatment and procedures, 3rd edn. Saunders Elsevier, St Louis, p 60

Lilly LS (ed) (2003) Pathophysiology of heart disease, 3rd edn. Lippincott Williams & Wilkins, Philadelphia

Marr CM, Bowen M (eds) (2010) Cardiology of the horse, 2nd edn. Saunders Elsevier, Edinburgh

Patteson MW (ed) 1997) The cardiovascular system. In: Robinson NE (ed) Current therapy in equine medicine 4. W.B. Saunders, Philadelphia, p 225

Poole DC, Erickson HH (2004) Heart and vessels: function during exercise and respose to training. In: Hinchcliff KW, Kaneps AJ, Geor RJ (eds) Equine sports medicine and surgery, 1st edn. Saunders, Edinburgh, p 699

Schwarzwald CC, Bonagura JD, Muir WW (2009) The cardiovascular system. In: Muir WW (ed) Equine anesthesia – monitoring and emergency therapy, 2nd edn, Saunders Elsevier, p 37

Schwarzwald CC (2009) Cardiovascular pharmacology. In: Robinson NE, Sprayberry KA (eds) Current therapy in equine medicine 6, Saunders Elsevier, p 182

Various authors (1999) Cardiovascular system. In: Colahan PT, et al (eds) Equine medicine and surgery, 5th edn. Mosby, St Louis, p 295

Young L (2004) Diseases of the heart and vessels. In: Hinchcliff KW, Kaneps AJ, Geor RJ (eds) Equine sports medicine and surgery, 1st edn. Elsevier, Edinburgh, p 728

Chapter 8

Diseases of the equine urinary tract

John Schumacher, Jim Schumacher, Joseph S. Spano

CONTENTS

8.1 Evaluation of the urinary system 159
Renal function and water balance in the normal horse 159
Physical examination 160
Ultrasonographic examination 160
Endoscopic examination 160
Percutaneous renal biopsy 160
Laboratory assessment of urinary tract disease 160
Renal disease vs. renal failure 161
8.2 Prerenal azotaemia 161
8.3 Acute renal failure due to renal disease 162
8.4 Chronic renal failure (CRF) 162
8.5 Cystitis 163
8.6 Urolithiasis 164
8.7 Uroperitoneum 164
8.8 Patent and persistent urachus 165
8.9 Haematuria 165
8.10 Polyuria/polydypsia (PUIPD) 167
8.11 Diagnosis of diseases of the testis and associated structures 168
History and physical examination 168
8.12 Castration 169
Indications 169
Preoperative considerations 169
Methods of restraint 169
Techniques of castration 169
Aftercare 169
Postoperative complications of castration 169
8.13 Congenital monorchidism 170
8.14 Cryptorchidism 170
8.15 Inguinal herniation and rupture 171
8.16 Torsion of the spermatic cord 173
8.17 Testicular neoplasia 173
8.18 Intersex 173
8.19 Diagnosis of diseases of the penis and prepuce 173
8.20 Penile and preputial injuries 173
8.21 Paraphimosis 173
8.22 Phimosis 174
8.23 Priapism 174
8.24 Neoplasia 174
8.25 Cutaneous habronemiasis (or 'summer sore') 174
8.26 Haemospermia 175
8.27 Diagnosis of diseases of the female reproductive tract 176
8.28 Pneumovagina 176
8.29 Perineal lacerations and fistulae 176
8.30 Urovagina 178
8.31 Cervical lacerations 178
8.32 Neoplasia 178
Perineal neoplasms 178
Neoplasms of the tubular genital tract 178
Ovarian neoplasms 178
8.33 Uterine torsion 178
8.34 Retained placenta 178
8.35 Dystocia 178
8.36 Ovariectomy (oophorectomy) 179
Further reading 179

8.1 EVALUATION OF THE URINARY SYSTEM

Renal function and water balance in the normal horse

- Water intake: varies greatly depending upon diet, ambient temperature, exercise, lactation, and psychogenic factors. Range: 4 to 6% of body weight daily.

© 2013 Elsevier Ltd
DOI: 10.1016/B978-0-7020-2801-4.00008-0

- Urination frequency: 7–11 times daily (foals urinate hourly and usually upon standing).
- Urine volume: 1–3% body weight daily.
- Urine character: pale yellow to brown (may darken upon standing); viscid due to mucus-secreting cells in the renal pelvis; often opaque due to suspension of calcium carbonate crystals. Horses fed legume hay form large amounts of calcium carbonate crystals and thus tend to have more opaque urine than horses fed grass or hay.

Physical examination

Only the caudal pole of the left kidney can be palpated *per rectum*. Evaluation is highly subjective, but:

- enlargement and pain response may be noted if the horse is in acute renal failure.
- shrinkage, firmness, and irregular surface may be noted if the horse is in chronic renal failure.

Ultrasonographic examination

- Equipment: 3.0–3.5 Mhz transducer for percutaneous examination (5.0 Mhz for foals); 5.0–7.5 Mhz transducer for examination per rectum.
- Location: level of tuber coxae and 15th to 17th intercostal space for right kidney and paralumbar fossa for left kidney.
- Indications:
  - Distinguishing acute and chronic renal disease.
    Acute – swollen kidney with decreased echogenicity.
    Chronic – small, irregular kidney with increased echogenicity.
    A kidney should be considered to be abnormally small when it measures less than 10 cm.
  - Diagnosis of renal cysts, urinary calculi, urinary neoplasia, uroperitoneum, etc.

Endoscopic examination

Useful in diagnosis of cystitis, urinary calculi, sources of haematuria, etc. For male horses, a 100 cm, or longer, endoscope with an outside diameter no greater than 12 mm is used. Polyethylene tubing can be passed through the accessory chamber of the endoscope into a ureter for collection of urine (Figure 8.1).

Percutaneous renal biopsy

Usually Vim-Silverman-type or Tru-Cut-type biopsy needles are used.

- Ultrasonically guided – the kidney is located ultrasonographically and the depth for needle penetration is measured, usually 3 cm and 7 cm for the right and left kidney, respectively.
- Assistant-guided – the left kidney is grasped per rectum by an assistant who can feel the needle contact the kidney.

Serious haemorrhage can result, but in general, biopsy is a safe procedure. Biopsy is of most value for assessment of suspected chronic renal failure. There is often poor correlation between severity of histological lesions and severity of renal dysfunction in acute ischaemic renal disease.

Laboratory assessment of urinary tract disease

- Serum urea nitrogen (SUN) (also known as blood urea nitrogen, BUN) – concentration rises when glomerular filtration rate (GFR) decreases. Not a reliable indicator of GFR because it is affected by non-renal factors. Anorexia and liver disease decrease concentration of SUN; increased protein intake increases concentration of SUN.
- Serum creatinine – concentration rises when GFR decreases. Production (from muscle) is fairly constant. Not significantly absorbed or secreted by renal tubules. Therefore, creatinine concentration approximates GFR. Azotaemia (elevated concentration of SUN and/or creatinine) is not necessarily indicative of renal disease or the severity of renal disease.
- Creatinine clearance – a reliable index of GFR in horses – determined by comparing creatinine concentrations in serum (Scr) and urine (Ucr) and the rate of urine production. Clearance cr = Ucr/Scr × mL/min/kg body wt. Reference range is 1.39 to 1.87 mL/min/kg. Concentrations below the reference range indicate decreased GFR.
- Serum electrolytes.
  - Tendency toward hyponatraemia, hypochloraemia, and hyperkalaemia in renal failure. Degree of abnormality, if any, depends upon diet, appetite, and duration of renal failure.
  - Hypo, normo, or hypercalcaemia can occur in renal failure – depends upon diet, appetite, duration of failure, and age of the horse. Hypercalcaemia in renal failure is unique to the horse and is not understood.
  - Hypo, normo, or hyperphosphataemia can be seen in equine renal failure.
- Fractional excretion (clearance) of electrolytes – in renal tubular disease, electrolytes may be inappropriately excreted in urine. Because the clearance of creatinine is constant over time, the excretion of electrolytes can be compared to the excretion of creatinine. The formula for determining fractional excretion of an electrolyte (FE_e) is:

$$FE_e = \frac{Scr}{Ucr} \times \frac{Ue}{Se} \times 100$$

in which U = urine, S = serum, cr = creatinine, e = electrolyte. Values greater than reference range indicate increased loss of electrolyte in urine, and may reflect inability of renal tubular cells to reabsorb that electrolyte.

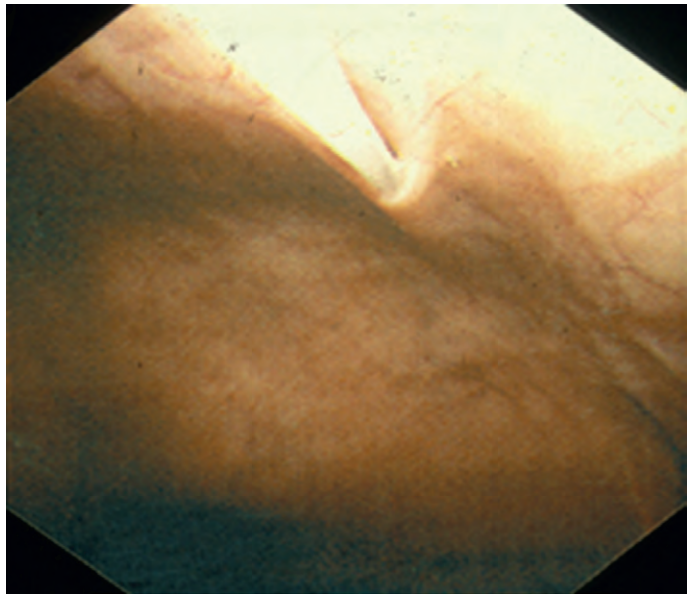


Figure 8.1 Polyethylene tubing can be passed through the accessory chamber of the endoscope into a ureter for collection of urine.
This photo was used in an EVE article (Macroscopic hematuria in horses. Equine Veterinary Education 2002;4:255–265, reprinted with permission of Wiley-Blackwell).

FE_{sodium} is normally ≤ 1%; > 3% indicates abnormal renal excretion.

$FE_{phosphorus}$ is normally ≤ 1%; increases occur not only in renal disease but also in primary or pseudohyperparathyroidism and secondary nutritional hyperparathyroidism.

- Urine to plasma or serum ratios for urea nitrogen (un), creatinine (cr) and osmolality (osm). Useful in distinguishing between renal and prerenal azotaemia, but measurements for renal and prerenal azotaemia may overlap. Based on a small number of azotaemic horses, reported measurements are:
  - U_{un}/P_{un} >15 (prerenal); <14 (renal)
  - U_{cr}/P_{cr} >50 (prerenal); <40 (renal)
  - U_{osm}/P_{osm} >1.7 (prerenal); <1.7 (renal) (overlap)
- Urinary enzymology. When renal tubular cells die, enzymes contained within these cells are released into the urine. Abnormally high concentrations of **gamma glutamyltranspeptidase (GGT)** indicate renal tubular damage. Comparison of UGGT concentration to Ucr concentration corrects for the effect of urine concentration on enzyme concentration.

$$\frac{UGGT}{Ucr} \times 100 = < 50 \text{ in normal horses}$$

Elevation indicates that tubular necrosis is occurring. Urinary concentrations of GGT are unaffected by serum concentrations of GGT. Concentration of UGGT is useful in monitoring for aminoglycoside-induced renal tubular necrosis.

- Urinary specific gravity (USG) tends to be between 1.030 and 1.060, but fluctuates widely. Random urine samples may indicate hyposthenuria (USG <1.008), isosthenuria (USG 1.008–1.014) or hypersthenuria (USG >1.015). Foals tend to be hyposthenuric because they have a large fluid intake. Possible explanations for dilute urine in the adult horse are:
  1. Excessive water intake from psychogenic causes (USG is usually <1.008).
  2. Renal disease (USG is usually 1.008–1.014).
  3. Fluid therapy.
  4. Diabetes insipidus, central or renal (USG is usually <1.008).

A water-deprivation test may distinguish between these causes if the cause is not obvious. For the water-deprivation test, the horse is weighed and held off water for 24 hours, or until it has a 5% weight loss, develops azotaemia or signs of dehydration or a USG >1.020. USG in the isosthenuric range (1.008–1.014) in a dehydrated horse suggests renal disease. The test should not be performed on horses that are azotaemic or dehydrated.

*USG <1.020 in horses with clinical dehydration and/or azotaemia is highly suggestive of renal disease. Azotaemia associated with USG >1.030 indicates prerenal azotaemia.

A modified water-deprivation test is indicated if a horse fails to concentrate urine to SG >1.020 during a water-deprivation test. Water intake is restricted to 40 mL/kg body weight per day for 3 to 4 days. The urine of horses with psychogenic polydypsia and 'medullary washout' should concentrate to >1.020 during this test.

- Urinary pH. Normally alkaline (pH is usually between 7.5 and 8.5). Urine may be acidic in cases of:
  1. Prolonged anorexia.
  2. High-grain diets.

The urine of foals tends to be acidic.

- Urinary sediment.
  - Casts – presence of granular or cellular casts usually indicates renal disease; casts dissolve quickly in alkaline urine. Leukocyte casts are indicative of bacterial nephritis.
  - Bacteria – the presence of more than 10 000 colony-forming units per mL of urine indicates urinary infection.
  - White blood cells – the presence of small numbers (≤ 8/high-power field or 400× on a free catch or ≤5/hpf on catheter collection) is not abnormal.
  - Crystals – normal urine may contain crystals of calcium carbonate, triple phosphate, and oxalate.
  - Urinary protein.

Commercial dipsticks may falsely indicate the presence of protein in alkaline urine. The sodium sulfasalicyclic acid test should be performed to determine whether the horse has proteinuria. Proteinuria without presence of blood or cellular debris is likely of renal origin, and may indicate glomerulonephritis.

Renal disease vs. renal failure

Renal disease tends to be inapparent until renal failure occurs, and renal failure can occur without the presence of renal disease. Renal failure is the inability of the kidneys to excrete nitrogenous wastes and is characterized by azotaemia (elevated concentration of serum urea nitrogen and/or creatinine). Renal failure occurs when about 75% of nephrons becomes temporarily or permanently non-functional. Azotaemia can occur in the absence of renal disease merely from decreased renal blood flow (prerenal azotaemia), or from disease of the urinary tract distal to the kidneys (urolithiasis, uroperitoneum) (postrenal azotaemia). Differentiating prerenal azotaemia from renal failure caused by renal disease (renal azotaemia) is not always straightforward.

8.2 PRERENAL AZOTAEMIA

Prerenal azotaemia represents a decrease in GFR due to renal hypoperfusion. The condition is rapidly reversible.

Aetiology

Usually caused by intravascular volume depletion caused by haemorrhage, diarrhoea, endotoxaemia, inadequate water consumption, etc.

Pathogenesis

Systemic hypotension stimulates the renin–angiotensin–aldosterone axis, release of antidiuretic hormone, and the sympathetic nervous system. As a result, blood flow is redistributed away from the renal cortex, and water is conserved.

Clinical signs

- In the horse, signs of gastrointestinal crises (such as colic or diarrhoea) are often evident.
- Tachycardia.
- Oliguria.
- Dehydration (loss of skin turgor).

Clinicopathological signs

- Haemoconcentration (increased PCV or serum albumin).
- Azotaemia with a urine specific gravity >1.030.
- Urine to plasma ratios of urea, creatinine, and osmolality may help distinguish prerenal from renal azotaemia.

Diagnosis

Based on clinical and clinicopathological signs and rapid response to appropriate therapy.

Treatment

- Correction of the initiating cause. Usually involves treatment of gastrointestinal crisis.
- Fluid therapy.

8.3 ACUTE RENAL FAILURE DUE TO RENAL DISEASE

Aetiology

Acute renal tubular necrosis (RTN) resulting in acute renal failure (ARF) is caused by sustained or severe hypoperfusion, or nephrotoxins, or a combination of both.

- Haemodynamic causes are most often initiated by endotoxaemia associated with some types of colic and acute diarrhoeal syndromes.
- Nephrotoxins:
  1. Plants – include red maple and oak trees, onions, and white snake root. Plants containing oxalates are potentially nephrotoxic, but oxalate-induced nephropathy is rare in horses. Deposition of oxalate crystals in kidneys, however, may occur secondarily to renal disease.
  2. Heavy metals – mercury, lead, arsenic, and others.
  3. Antibiotics – aminoglycosides (gentamicin, neomycin, amikacin), tetracycline, sulfonamides, cephaloridine, amphotericin B, and others.
  4. Non-steroidal anti-inflammatory drugs – (NSAIDS) phenylbutazone and flunixin meglumine. NSAIDS are more likely to cause ARF when there is concurrent dehydration.
  5. Pigments – myoglobin and haemoglobin. Haemoglobin and myoglobin are more likely to cause ARF if there is concurrent dehydration.
- Acute pyelonephritis – most likely to occur in septicaemic neonates; *Leptospira* sp. and other bacteria can cause acute tubular necrosis in adults.

Pathogenesis

Renal tubular necrosis is induced by:

1. direct effect of nephrotoxins.
2. ischaemia caused by sympathetic constriction of renal vessels during circulatory shock.

Clinical signs of renal tubular necrosis

Clinical signs of acute renal failure due to renal tubular necrosis are non-specific – anorexia, depression, and weakness. These clinical signs can also be attributed to a precipitating haemodynamic cause such as colic or colitis. Toxins that induce acute renal failure are often not renal specific, and clinical signs associated with organ damage of other systems may predominate.

Diagnosis

Acute renal failure caused by renal tubular necrosis is usually diagnosed on the basis of clinical signs, physical examination, ultrasonography, and laboratory evaluation.

Caudal pole of left kidney may feel enlarged when palpated per rectum, or kidneys may appear enlarged during ultrasonic examination. Horses with renal tubular necrosis, however, often have no ultrasonographic abnormalities of the kidneys.

- Isosthenuria with concurrent dehydration and/or azotaemia.
- Granular urinary casts.
- Measurement of urinary enzymes. The UGGT/Ucr ratio is most commonly used.
- Fractional excretion of sodium and phosphorus may be increased.
- Glycosuria without hyperglycaemia.
- Urine to plasma ratios of urea, creatinine, or osmolality may help distinguish prerenal from renal azotemia.

Of all the above tests, only isosthenuria with concurrent dehydration or azotemia indicates renal failure**.** Other tests indicate **renal disease**.

Treatment

- Treatment of the predisposing disease process (e.g. endotoxaemia, myositis).
- Removal of suspected nephrotoxins.
- Correction of fluid balance, serum electrolyte concentration, and acid–base abnormality.

Intravenous administration of balanced electrolyte solution if anuric or oliguric. Over-hydration of anuric or oliguric horses can be avoided by:

1. monitoring central venous pressure.
2. monitoring for increases in body weight.
3. auscultating lungs for evidence of oedema.
4. observing for subcutaneous oedema.
5. monitoring for marked decrease in PCV.

If the horse is anuric or oliguric despite rehydration, convert to polyuria with:

1. 10% dextrose solution IV.
2. 20% mannitol (0.25 to 2 g/kg slow IV).
3. furosamide (1 mg/kg, IV every two hours).
4. dopamine (2 to 5 mg/kg/min in 5% dextrose, IV).

- Antibiotics. Pyelonephritis is treated by antimicrobial drugs excreted by the kidney such as aminoglycosides, trimethoprim/sulfadiazine, fluoroquinolones, penicillin and cephalosporins. Pyelonephritis caused by *Leptospira* sp. is treated with tetracycline, streptomycin or penicillin.

Prognosis

Toxin-induced nephrotoxicosis has a more favourable prognosis than does haemodynamic-induced nephrotoxicosis. Reason: tubular cell basement membrane is more likely left intact in toxic nephritis, and cellular regeneration is more likely if the basement membrane is intact.

Prognosis is fair to good if:

- predisposing causes can be successfully treated.
- creatinine stabilizes within 24 hours of therapy and then progressively declines over several days.
- the horse is polyuric.

8.4 CHRONIC RENAL FAILURE (CRF)

Once thought to be a rare condition in horses, CRF is now considered to account for a significant portion of horses presented for weight-loss and anorexia.

Aetiology and pathogenesis

Chronic glomerulonephritis is the most frequent cause of CRF. Chronic glomerulonephritis does not always cause CRF. In one study 40% of equine kidneys examined at necropsy had microscopic glomerular lesions); there are two types of lesions:

- Antiglomerular basement membrane glomerulonephritis – (anti-GBMG) – is due to antibodies directed against the glomerular basement membrane.
- Immune complex glomerulonephritis (ICG) – (the most common type) – is due to deposition of immune complexes along the glomerular basement membrane. Immune complexes

may be associated with upper respiratory infections caused by *Streptococcus* spp. With either type, the glomeruli become inflamed and the glomerular membrane becomes thickened by fibroblasts.

Tubulointerstitial disease (chronic interstitial nephritis); sequelae to acute renal tubular necrosis (induced by toxins or haemodynamic causes).

Chronic pyelonephritis: an uncommon cause of renal failure in adult horses. The most commonly reported cause is ascending infection of the urinary tract, which is a consequence of urinary stasis caused by urolithiasis, neurological disease affecting the urinary tract (e.g. sorghum cystitis, herpes virus-1 myeloencephalitis), or trauma (e.g. from foaling). Renal infection can also occur by haematogenous spread. In adult horses, left-sided bacterial endocarditis is a cause.

Bilateral renal hypoplasia – probably a congenital lesion; disease becomes evident when the horse is young.

Chronic oxalate nephrosis – thought to be a consequence rather than a cause of CRF.

Renal neoplasia – uncommon in horses. Types reported: adenocarcinoma (most common) and lymphoma.

Polycystic renal disease – very rare; probably a congenital disease that becomes evident in most affected horses before they reach maturity.

Clinical signs

Signs vary depending upon the aetiology

- Cachexia.
- Anorexia and depression.
- Dehydration.
- Polyuria/polydipsia.
- Peripheral oedema. Seen in horses with chronic glomerulonephritis; caused by extensive loss of plasma proteins through damaged glomerular capillaries. Triad of oedema, hypoproteinaemia, and proteinuria is referred to as the **nephrotic syndrome**.
- Oral ulceration and dental tartar – occasionally observed.
- Fever – may be seen with pyelonephritis.
- Palpation per rectum and/or ultrasonic examination may reveal shrunken, firm kidney(s) with an irregular surface.
- Ureteral discharge of bloody or purulent urine may be seen endoscopically in horses with pyelonephritis.

Clinicopathological signs

Laboratory findings vary depending on the aetiology, stage of disease, and management factors.

- Anaemia – due to decreased production of renal erythropoietic factor and shortened RBC lifespan.
- Leukocytosis.
- Proteinuria – **if glomerulonephritis is the cause of CRF, proteinuria is a consistent finding.** Tubulointerstitial disease and pyelonephritis cause minimal proteinuria.
- Horses with chronic tubulointerstitial disease are invariably isosthenuric (1.008–1.019). Isosthenuria eventually develops in horses with chronic glomerulonephritis.
- The horse is usually azotaemic.
- Concentrations of serum electrolytes may be abnormal.
  - hypochloraemia and hyponatraemia.
  - normo or slight hyperkalaemia.
  - hypercalcaemia. Some horses with CRF on high calcium diets become hypercalcaemic.
  - Horses with pyelonephritis may have pyuria or haematuria.

Diagnosis

Based on clinical and clinicopathological signs, endoscopic and ultrasonic examination, and renal biopsy.

Treatment

Chronic renal failure is progressive. Clients should be advised that treatment is aimed at prolonging life rather than resolving the condition.

- Supplementation of electrolytes based on periodic evaluation of serum concentrations of sodium, potassium, calcium, and bicarbonate. (If oedema develops, sodium should be restricted from the diet, even if the horse is hyponatraemic.)
- Dietary supplementation with carbohydrates and fat.
- Restriction of dietary protein to less than 10%.
- Forced feeding in cases of anorexia.
- Anabolic steroids.
- Antimicrobial therapy for pyelonephritis. Selection of drug is based on culture and sensitivity and the ability of the antimicrobial drug to concentrate in renal tissue and urine.

8.5 CYSTITIS

Aetiology

- Primary (i.e. no discernible cause) is rare.
- Secondary – also rare; secondary to urinary stasis with subsequent bacterial proliferation. Caused by:
  1. Cystic calculi.
  2. Neurological disease of the urinary tract such as sorghum cystitis, herpes virus myeloencephalitis, polyneuritis equi, etc.

Clinical signs

- Pollakiuria (unduly frequent urination) that may resemble signs of oestrus; also dysuria (difficult urination), and urine dribbling.
- Perineal scalding in the mare and soiling of hind legs in the male.
- Gross haematuria in some cases.

Clinicopathological findings

Examination of urine sediment for:

- WBCs. More than 8 WBCs per high-power field of urine collected during urination or more than 5 when urine is collected by catheterization is evidence of inflammation.
- RBCs. More than 8 RBCs per high-power field of urine collected during urination or more than 5 when urine is collected by catheterization is evidence of haemorrhage.
- Large number of bacteria. Recovery of more than 10 000 colony-forming units per mL of urine collected by catheterization is diagnostic of urinary tract infection.

Diagnosis

Clinicopathological findings confirm the presence or urinary tract infection, and physical examination (thickened bladder palpated per rectum) and/or cystoscopic examination (thickened, hyperaemic, or ulcerated mucosa) localize the infection to the bladder.

Treatment

- Correction of a predisposing cause if possible, such as removal of cystic calculi.
- Antimicrobial therapy based on culture and sensitivity and ability of antimicrobial drug to concentrate in urine (such as aminoglycosides, trimethoprim/sulfadiazine, fluoroquinolones, penicillin and cephalosporins).

8.6 UROLITHIASIS

Uroliths or calculi can form in the kidney (nephrolithiasis), ureters (ureterolithiasis), bladder (cystic urolithiasis) or urethra. If small, may be voided on urination or cause urethral obstruction. Most uroliths are composed of calcium carbonate and are spiculated and fragment easily (see Figure 8.2); those that also contain phosphate are smooth and hard and uncommon.

Aetiology

- Mineralization of a nidus – renal disease may provide the nidus for nephro- and ureterolithiasis. NSAID-induced nephropathy has been speculated to be a cause of nidus formation in horses with nephro- and ureterolithiasis. Urolithiasis also may be the consequence of disease of the upper portion of the urinary tract such as pyelonephritis.
- Abnormally low concentrations of natural inhibitors of mineral complexes in urine. High content of mucus produced by glands in the renal pelvis may prevent crystal aggregation.

Clinical signs of nephrolithiasis and ureterolithiasis

Clinical signs of chronic renal failure (cachexia, anorexia, depression, dental tartar, oral ulcers, etc.). Calculi **may cause or be the result** of renal disease.

Clinical signs of cystic and urethral calculi

- Urine dribbling, dysuria or pollakiuria.
- Prolonged periods of penile protrusion.
- Haematuria, especially after exercise.
- Stilted hind limb gait.

Clinicopathological examination

- Evidence of renal failure may be found in a high percentage of horses with nephrolithiasis or ureterolithiasis.
- Haematuria.
- Evidence of cystitis – increased numbers of WBCs and RBCs, large number of bacteria.

Figure 8.2 Most uroliths are composed of calcium carbonate and are spiculated and fragment easily.

Diagnosis

- Examination *per rectum* – a cystic calculus is easily palpated. A ureterolith or dilated ureter can be palpated in some cases.
- Ultrasonographic examination – percutaneous or *per rectum*.
- Cystoscopic examination.
- Inability to pass a urinary catheter may indicate the presence of a urethral calculus.

Treatment

- Surgical removal of a cystic calculus:
  - celiotomy and cystotomy.
  - pararectal cystotomy.
  - urethral sphincterotomy (mares).
  - ischial urethrotomy (males).
- Urethrotomy at any site (for removal of urethral calculi).
- Electrohydraulic or laser lithotripsy via ischial uretrotomy.
- Antimicrobial therapy for concurrent bacterial infection.

Renal function of **both** kidneys should be assessed before nephro- or ureterolithiasis is treated surgically (nephrectomy, nephrotomy) because bilateral renal failure is a common finding.

Prevention of recurrence

Insuring complete removal of all fragments is important in preventing recurrence. Because urolithiasis may be the consequence of disease of the upper portion of the urinary tract, horses presented for urolithiasis should be examined for disease of the upper urinary tract. Treatment for pyleonephritis, if present, may prevent recurrence. Other preventive measures include:

1. Low calcium diet to just meet calcium and phosphorus requirements.
2. Urinary acidification (to pH below 6) with:
   - Ammonium chloride – commonly used but unpalatable.
   - Ascorbic acid – questionable efficacy.
   - Ammonium sulphate – 75 mg/kg twice daily.
3. Providing 1% salt (or 60–120 g) in the concentrate ration has also been recommended.

8.7 UROPERITONEUM (see also Chapter 20)

Aetiology and pathogenesis

1. Adults – bladder rupture.
   - during parturition or after parturition (due to bladder necrosis).
   - urethral obstruction by calculi (males).
2. Foals – bladder rupture and urachal tears, ureteral defects.
   - Prenatal distension of the bladder (perhaps caused by partial torsion of the umbilical cord) coupled with pressure on the full bladder during parturition leads to rupture of bladder or urachus. **Affected foals are usually male.**
   - Congenital bladder defects may be responsible for uroperitoneum of some foals.
   - Bladder and urachal rupture may occur due to lesions caused by urinary tract infections.
   - Tenesmus associated with g.i. disease may cause urachal tears.
   - Leakage of urine through a thin bladder wall.

Clinical signs (foals)

Usually occur within the first week postpartum.

- Abdominal distension; fluid can be balloted,
- Tachycardia and tachypnoea.

- Anorexia and depression.
- Pollakiuria, anuria, urine dribbling, straining to urinate, or normal urination may be noted.
- Outward bulging of vaginal mucosa may be seen in fillies with rupture of a ureter.

Clinicopathological findings

- Concentration of creatinine in peritoneal fluid containing urine is usually double that of serum creatinine (exception is foals evaluated early after bladder rupture).
- Hypochloraemia, hyponatraemia, and hyperkalaemia in foals. These electrolyte abnormalities may not be seen in the adult.
- Foals are usually, but not necessarily, azotaemic.
- Calcium carbonate crystals may be seen in peritoneal fluid.

Diagnosis

- Clinical signs and clinicopathological findings.
- Ultrasonography.
- Dye (methylene blue or fluorescein) placed into the bladder and subsequently recovered in peritoneal fluid.
- Positive contrast cystography (do not use barium).
- For diagnosis of suspected ureteral defects, exploratory laparotomy and cystotomy are performed. The ureters are infused with dye such as methylene blue, and examined for leakage. Intravenous pyelography is not very useful.

Treatment

Cystorrhaphy and/or resection of urachus. Preoperative therapy might involve:

1. Measures to lower the potential for cardiac arrythmia caused by high serum concentration of potassium.
   - isotonic or hypertonic saline solution, IV.
   - 5% dextrose, IV and insulin.
   - calcium, IV.
   - enemas of sodium polystyrene sulfonate (a potassium-removing resin).
   - mask induction and anaesthesia with isoflurane or sevoflurane, which are less arrythmogenic than halothane.
2. Peritoneal drainage.
3. Antimicrobial drugs.

8.8 PATENT AND PERSISTENT URACHUS

(see also Chapter 20)

The urachus, which connects the foal's bladder with the allantoic cavity, normally closes at or shortly after birth. A urachus that reopens after being closed is a *patent urachus*, and a urachus that does not close after birth is termed a *persistent urachus*.

Aetiology

The urachus may fail to close because of:

- Dilation of the urachus before birth caused by umbilical torsion.
- Clamping the umbilicus rather than allowing it to break spontaneously.

The urachus may reopen because of:

- Umbilical infection.
- Lifting the foal by the abdomen.

Clinical signs

- Moist navel.
- Urination from the navel.
- Navel region may be enlarged from infection.
- Clinical signs of infection, such as septicaemia or joint ill.

Diagnosis

- Clinical signs.
- Umbilical ultrasonography – umbilical remnants are scanned for evidence of infection.

Treatment

- Regardless the suspected cause, the foal should be thoroughly examined for evidence of systemic disease.
- If uncomplicated, merely preventing urine scald by daily cleaning and application of petroleum jelly to the umbilical region; administration of prophylactic antimicrobial drugs.
- If uncomplicated, cauterizing agents (silver nitrate, phenol, Lugol's iodine) can be applied to the opening of the urachus for several days.
- Resection of the urachus and antimicrobial therapy are indicated as treatment of patent urachus if there is:
  - lack of response to conservative therapy.
  - evidence of umbilical infection.
  - evidence of sepsis of any distant site (such as septicaemia or joint-ill).

8.9 HAEMATURIA

Introduction

Haematuria in horses can originate from the kidney, bladder, urethra, or reproductive tract. Haematuria is obvious if the urine is heavily contaminated with blood, but when urine is only blood-tinged, distinguishing haematuria from haemoglobinuria or myoglobinuria may be difficult. To establish that a reddish discoloration of urine is caused by red blood cells, urine can be centrifuged to observe a layer of red cells covered by clear urine. Normal equine urine may contain plant pigments that can stain bedding red or cause urine to turn brown or red after exposure to air or after contact with snow.

Aetiology

- Urethral rents – a tear in the urethra that communicates with the corpus spongiosum penis causes haematuria in geldings and haemospermia in stallions.
- Urethritis – urethritis as a cause of haematuria is most likely an erroneous interpretation of the endoscopic appearance of the normal male urethra. The vasculature and cavernosal spaces surrounding the urethra become more prominent when the urethra dilates with air during endoscopic examination creating the appearance of severe inflammation.
- Bacterial cystitis – rarely primary; usually secondary to urine retention caused by paresis or paralysis of the bladder, or by cystic urolithiasis.
- Pyelonephritis (suppurative bacterial infection of the renal pelvis and parenchyma) – may be the cause of severe, life-threatening renal haemorrhage (pyelonephritis as a cause of renal haemorrhage is disputed, however).
- Idiopathic renal haemorrhage – may be the cause of severe, life-threatening renal hemorrhage. Reported predominantly in Arabians.
- Urolithiasis.
- Verminous nephritis – caused by *Halicephalobus gingivalis* (previously known as *Micronema deletrix*).

- Renal neoplasia – of the most common tumours affecting the kidney, adenocarcinoma is more likely than lymphosarcoma to cause haematuria.
- Vesicular neoplasia – squamous cell carcinoma and transitional cell carcinomas.
- Blister beetle toxicosis – a cause of haematuria for horses fed alfalfa hay contaminated with the dead beetle.
- Exercise-induced haematuria – usually microscopic but occasionally macroscopic; caused by repeated concussion of the bladder against the pelvis sufficient to cause mucosal damage during exercise.
- Nephrotoxic drugs – most commonly phenylbutazone administered to dehydrated horses.
- Vascular anomalies – such as renal arteriovenous fistulas; rare.
- Polycystic kidney disease – congenital deformity of the renal tubular system leads to formation of cystic structures filled with urine: cysts eventually expand to cause renal failure (usually before 2 years of age); some affected horses have haematuria.

Diagnosis

- Urethral rents – haemorrhage is observed to occur at the very end of urination; during endoscopy a tear in the urethra is seen on its convex surface at the level of the ischial arch (Figure 8.3).
- Urethritis – usually not considered to be a cause of haematuria; could be suspected when there is history of excess washing of the penis with disinfectant soaps.
- Bacterial cystitis – clinicopathological findings confirm the presence of urinary tract infection, and physical examination (thickened bladder palpated per rectum) and/or cystoscopic examination (thickened, hyperaemic, or ulcerated mucosa) localize the infection to the bladder.
- Pyelonephritis – clinicopathological findings confirm the presence of urinary tract infection, and urine collected from a ureter localizes the infection to the kidney. Renal biopsy confirms the diagnosis.
- Idiopathic renal haemorrhage – this diagnosis is made when known causes of renal haemorrhage are ruled out.
- Urolithiasis – typical history of haematuria after exercise; palpation of the bladder *per rectum*; endoscopic examination of the urethra and bladder, and ultrasonographic examination of the urethra, bladder, or kidney.

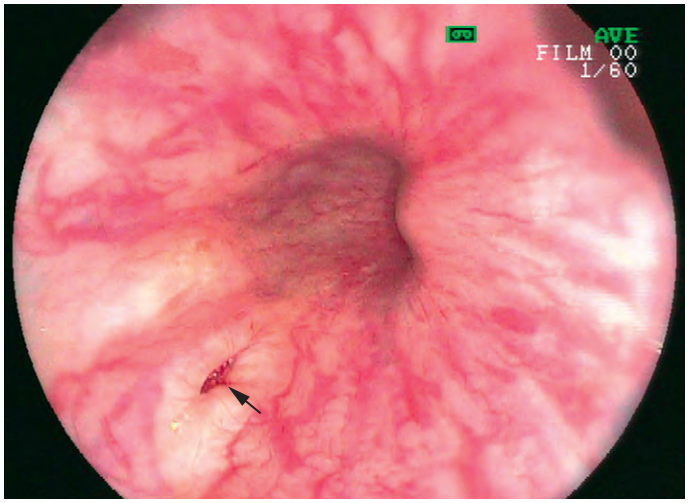


Figure 8.3 A urethral rent is diagnosed during endoscopy. A tear in the urethra is seen on its convex surface at the level of the ischial arch.

- Verminous nephritis – clinical signs of disease of other systems; *Halicephalobus* often causes concurrent encephalitis, osteomyelitis, and orchitis; larvae can be found in urine; renal mass seen during ultrasonography; renal biopsy may be diagnostic.
- Renal neoplasia – renal mass seen during ultrasonography; renal biopsy.
- Vesicular neoplasia – cystoscopy and biopsy.
- Blister beetle toxicosis – history of feeding alfalfa hay; signs of abdominal pain are seen concurrently; affected horses are often hypocalcaemic.
- Exercise-induced haematuria – a history of haematuria seen post exercise; horses that urinate before exercise are more likely to be affected (urine in the bladder acts a cushion against trauma).
- Nephrotoxic drugs – history of administration of a non-steroidal anti-inflammatory drug (often in excess) to a dehydrated or hypovolaemic horse.
- Vascular anomalies – lesion found during colour-flow doppler ultrasonography.
- Polycystic kidney disease – signs of renal failure; during ultrasonography an enlarged kidney containing well defined cysts is seen.

Treatment

- Urethral rents – some heal without treatment. An 8-cm, vertical, cutaneous incision is created on the perineal raphe and centred on the ischial arch. The incision is extended through the tunica albuginea that surrounds the corpus spongiosum penis. Blood is thus diverted out this incision rather than through the rent which allows the rent to heal.
- Urethritis – horses usually respond to systemic antimicrobial therapy in conjunction with lavage of the urethral lumen with nonirritating antimicrobial drugs and sexual rest.
- Bacterial cystitis – correction of a predisposing cause (cystolithiasis or bladder paralysis); antimicrobial therapy based on culture and sensitivity and ability of antimicrobial drug to concentrate in urine (such as aminoglycosides, trimethoprim/sulfadiazine, fluoroquinolones, penicillin, and cephalosporins).
- Pyelonephritis – antimicrobial therapy based on culture and sensitivity and ability of antimicrobial drug to concentrate in urine.
- Idiopathic renal haemorrhage – nephrectomy if only one kidney is involved (after nephrectomy, however, the other kidney may bleed).
- Urolithiasis – surgical removal of the cystoliths and urethroliths; consider nephrectomy or nephrotomy for a nephrolith.
- Verminous nephritis – administration of larvicidal anthelminitic; possible nephrectomy.
- Renal neoplasia – no reported survivors; nephrectomy can be considered but metastasis will have likely occurred by the time the diagnosis is made.
- Vesicular neoplasia – no reported survivors; consider instillation of antineoplastic drugs into the bladder or partial cystectomy.
- Blister beetle toxicosis – symptomatic treatment with IV fluids.
- Exercise–induced–haematuria – treatment is not neccessary.
- Nephrotoxic drugs – cease administration of non-steroidal anti-inflammatory drugs, and intravenously administer fluids.
- Vascular anomalies – spontaneous resolution is reported. Surgical removal of an affected kidney.
- Polycystic kidney disease – treatment is usually unwarranted.

8.10 POLYURIA/POLYDYPSIA (PU/PD)

Introduction

The horse normally drinks 4–6% of its body weight (40–60 mL/kg) and produces half this amount (15 to 30 mL/kg body weight) of urine each day. When water consumption is greater than 10% body weight and urine production is greater than 5% body weight per day, the horse has PU/PD. Water consumption is easily measured, but, because urine production is not, PU often is assumed when stall bedding is unduly wet.

Aetiology

Excessive water intake that leads to polyuria:

- Psychogenic polydipsia – the most common cause of PU/PD in stabled horses; a type of stereotypic behaviour.
- Excessive salt consumption – a very rare vice.

Excessive urine production that leads to polyuria:

- Cushing's disease (pars pituitary intermedia dysfunction) (see Chapter 9) – can cause polyuria by several different mechanisms:
  1. Type 2 diabetes – excessively produced cortisol antagonizes insulin to cause glucosuria (diabetes mellitus).
  2. Cortisol antagonizes antidiuretic hormone at the collecting duct (peripheral diabetes insipidus).
  3. Pressure of the tumour within the pituitary gland may inhibit secretion of antidiuretic hormone (i.e. central diabetes insipidus).
- Type 1 diabetes (failure of pancreas to secrete insulin) – very rare in the horse; glucosuria results.
- Chronic renal failure – renal tubules fail to reabsorb water and electrolytes (an uncommon cause of PU/PD).
- Nephrogenic diabetes insipidus – hereditary condition or secondary to bacterial infection of the kidney.

Diagnosis

See Figure 8.4.

Treatment

- Psychogenic polydipsia – restrict water availability to meet maintenance (50 mg/kg/day) and increased requirements considering ambient temperature and exercise. Reduce boredom by turnout, increase forage and exercise.
- Cushing's syndrome – treatment with pergolide mesylate, trilostane or cyproheptadine.
- Central diabetes insipidus – ADH replacement therapy is likely cost prohibitive.
- Nephrogenic diabetes insipidus – restrict water and salt consumption.
- Chronic renal failure – dietary supplementation with carbohydrates and fat, restriction of dietary protein to less than

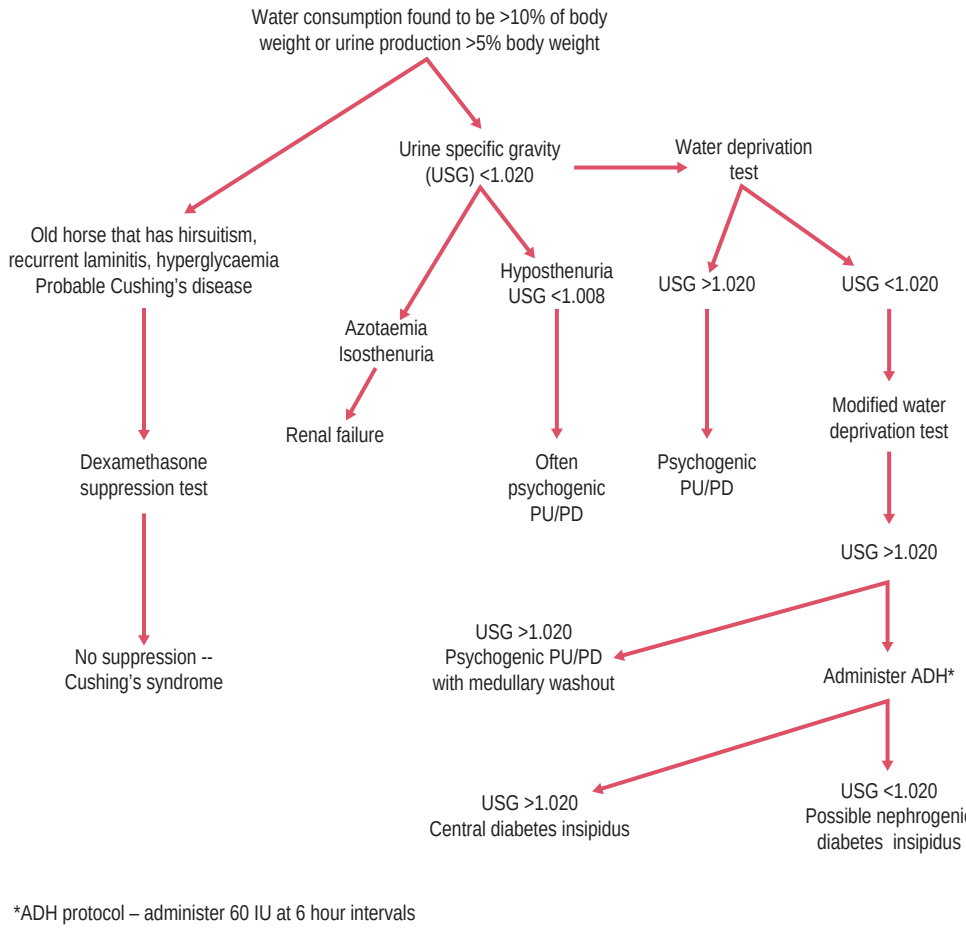


Figure 8.4 Diagnostic flow chart for the horse with PU/PD (water consumption found to be >10% of body weight, or urine production >5% body weight).

10%, anabolic steroids, and antimicrobial therapy if it is caused by pyelonephritis.

8.11 DIAGNOSIS OF DISEASES OF THE TESTIS AND ASSOCIATED STRUCTURES

History and physical examination

History may include:

- Evidence of fertility.
- Presence of testicular retention.
- Changes in testicular size – **insidious increase in size** suggests neoplasia or hydrocele. **Rapid increase in size** suggests torsion, inguinal herniation, or orchitis.
- Inguinal or scrotal swelling.
- Testicular pain – (neoplasia is usually painless).
- Previous urogenital surgery.
- Previous illnesses.

Physical examination of the genitalia should include inspection and palpation of the testes.

- Left testis is usually larger and more pendulous than the right.
- Body of epididymis should be located on the dorsolateral surface of testis, and tail of epididymis should be located on posterior pole of testis.
- Changes in testicular size, texture may indicate: orchitis, torsion, herniation, or neoplasia. A neoplastic testis is usually insensitive to compression and heavier than normal. **Hydrocele** should be suspected if scrotum is fluid-filled, and testis is smaller than normal.
- If a testis is absent from the scrotum, the horse can be sedated to relax its cremaster muscles. Scrotum should be examined for scrotal scars. ***A scar means only that orchidectomy was attempted – not that it was accomplished.***
- Only the distal half of the inguinal canal can be palpated. The epididymis descends before the testis, and an epididymis that lies within the canal can be mistaken for an inguinal testis.
- Inguinal hernia may appear as an enlargement in the spermatic cord or scrotum; palpation of a scrotal hernia may elicit crepitus, and intestinal peristalsis may be evident. Skin over a strangulated hernia may be cold and oedematous. Fluid-filled, enlargement of the healed scrotum following castration is indicative of a hydrocele.

Examination per rectum

This is helpful in diagnosis and evaluation of cryptorchidism, inguinal herniation, and other urogenital conditions, but risk of rectal tear should be weighed against the value of information to be gained. Small size and fractious nature of young horses predisposes them to rectal damage. Vaginal rings should always be palpated for inguinal incarceration of intestine when examining a colicky stallion *per rectum*.

- In average-sized horses, the **vaginal rings** can be palpated 6 to 8 cm cranial to the ilio-pectineal eminence and 10 to12 cm abaxial to the midline.
- Vaginal rings are palpable in geldings as a slight depression, but, in stallions, the rings can accommodate a finger.
- For horses with unknown history of castration that display sexual behaviour but have no discernible testes, examination *per rectum* may be useful in determining whether testes have entered the inguinal canal.
- Identification **of an abdominal testis,** ***per rectum***, is difficult, so inability to rectally palpate an abdominal testis is diagnostically unreliable.
- Vaginal ring of a **complete abdominal cryptorchid** (i.e. one in which both the testis and epididymis are within the abdomen) cannot be palpated. If the vaginal ring can be palpated, the testis, or at least its epididymis, has probably descended through the ring into the inguinal canal. Distinguishing a **partial abdominal cryptorchid** (i.e. one in which the testis is in the abdomen, but the epididymis is descended into the canal) from a horse whose testis and epididymis have entered the inguinal canal is difficult.

Hormonal assays

Hormonal assays are used to determine whether a horse with persistent masculine behaviour has a retained testis:

- Determination **of concentration of testosterone in the plasma or serum**. Stallions, including cryptorchids, have significantly greater concentrations of testosterone in the serum than do geldings. Concentration of testosterone in geldings is generally less than 40 pg/mL of serum and that of stallions is greater than 100 pg/mL. Concentration of testosterone of stallions varies seasonally (reaching its highest levels in the spring and summer) and with age. Concentration of testosterone is lowest in horses under 3 years of age.
- Testosterone **stimulation test.** Concentration of testosterone in response to stimulation by **human chorionic gonadotropin (hCG)** is determined. The test may be more accurate in determining whether a horse has testicular tissue than is evaluation of the horse's basal concentration of testosterone. The test is based on the fact that administration of human chorionic gonadotropin elicits a rise in concentration of serum testosterone. Following collection of serum for evaluation of basal concentration of testosterone, 6000 or 12 000 units of hCG is administered intravenously, and serum is again collected after 30 minutes (or up to 2 to 3 days later). A rise in the concentration of serum testosterone indicates that the horse has testicular tissue. Response to hCG, however, may be poor in horses less than 18 months old.
- Concentrations **of conjugated oestrogens.** There is a high correlation between the presence of testicular tissue and the concentration of conjugated oestrogen. Exceptions are cryptorchid horses under 3 years of age and cyptorchid donkeys of any age. Horses with less than 50 pg of oestrone sulphate/mL of plasma or serum can be considered geldings, and horses with concentrations in excess of 400 pg/mL can be considered to have testicular tissue.

Because a laboratory may have different standards than those listed above, it is **important to know the laboratory's standards** for normal hormonal concentrations of geldings and stallions when evaluating hormonal assays.

Other diagnostic tests

- Seminal evaluation. May aid in diagnosis of inflammatory testicular disease.
- Scrotal thermography. To detect variation in temperature between testes.
- Ultrasonography. To locate cryptorchid testes or detect other testicular abnormalites.
- Karyotyping. To determine sexual identity if intersexuality is suspected.
- Testicular biopsy. Seldom performed on horses, but is unlikely to affect semen quality.

8.12 CASTRATION

Synonyms include orchidectomy, emasculation, gelding, and cutting.

Indications

1. To eliminate masculine behaviour and managerial problems that arise from this behaviour. Horses can be castrated at any age, but most are castrated when objectionable masculine behaviour begins, usually between 1 and 2 years of age.
2. Indications for unilateral castration include:
   - Testicular neoplasia.
   - Testicular trauma.
   - Torsion of the spermatic cord.
   - Inguinal herniation.

Preoperative considerations

Physical examination should be performed before castration. The scrotum should be palpated, if the horse allows, to ensure that the horse is not a cryptorchid and that it has no inguinal hernia. The presence of either condition may change anaesthetic protocol or surgical approach.

Methods of restraint

Castration can be performed with the horse sedated and standing, after the spermatic cord is anaesthetized. Or, the horse can be anaesthetized and castrated in recumbency.

Advantages of standing castration:

- Expense and risk of general anaesthesia are avoided.
- No need to wait for the horse to recover from anaesthesia.
- A clean, safe area to anaesthetize the horse is not required.

Disadvantages of standing castration:

- Can be difficult and dangerous to the surgeon. Only docile stallions with well-developed testes should be castrated standing.
- Primary closure of scrotum is difficult.

Common agents used to anaesthetize horses for castration include:

- Thiobarbiturates following sedation with xylazine.
- Ketamine HCl following sedation with xylazine, detomidine, or romifidine.
- Guaifenesin (5–10%) in combination with ketamine HCl or a thiobarbiturate.
- Succinylcholine can be used to immobilize horses, but it provides no analgesia, and its use to restrain horses for castration is inhumane.

Techniques of castration

Regardless of whether the horse is castrated standing or anaesthetized and recumbent, the testes can be removed using one of three techniques:

1. **Closed technique**. Using this technique, each testis is isolated and removed while still encased by its parietal tunic. Removing the parietal tunic may decrease the incidence of postoperative complications such as infection of the cord and hydrocele. The closed technique is indicated when the testes are removed because of disease such as testicular neoplasia or infection.
2. **Open technique**. With this technique, the parietal tunic of each testis is opened, and the testis is removed. The parietal tunic of each testis remains with the horse. This technique requires less dissection than the closed technique and is often preferred when performing a standing castration. The open technique allows components of the cords to be examined before the cord is severed.
3. **Half-closed technique**. Each testis is isolated while still enclosed within its parietal tunic (as in the closed technique), but the parietal tunic is incised and the testis and cord are exteriorized from the parietal tunic before the tunic and testis are excised.

Aftercare

- If previously vaccinated against tetanus, the horse should receive a tetanus toxoid booster.
- If not previously vaccinated against tetanus, the horse should receive both tetanus toxoid and tetanus antitoxin.
- Antimicrobial therapy and fly control are usually unnecessary.
- Horse should be exercised daily to prevent preputial and scrotal swelling. The horse should not be exercised for 24 hours after surgery, however, to prevent haemorrhage.

Scrotal incisions are usually left unsutured to heal by second intention, but they can be primarily closed. Sutured scrotal wounds heal with less complication, but primary closure increases anaesthetic time and must be performed under strict aseptic conditions.

Postoperative complications of castration

Oedema

Some oedema is inevitable, especially on the 4th or 5th postoperative day, but excessive oedema is **usually caused by insufficient exercise**. Excessive oedema can be treated by increasing the horse's exercise and opening the scrotal wound if it is sealed.

Haemorrhage

The usual source of excessive haemorrhage is the testicular artery. Causes of excessive haemorrhage:

- Improperly **applied emasculators**. Emasculators should be applied to the cord so that the crushing part of the emasculator's jaws is proximal to the cutting part.
- Emasculators that are too sharp causing the cord to be cut before it is properly crushed.
- Attempting to crush and cut a cord that is too large for the emasculator. Large cords should be separated into two sections and each crushed and cut separately.
- Heavy exercise of the horse before castration.

Treatment for excessive haemorrhage:

- If the horse has been castrated standing, the cord may still be anaesthetized with local anaesthetic, and, with the horse standing, the **cord can be grasped and recrushed with the emasculator.** Or a **haemostat can be applied to the cord** and removed the following day. If the horse was castrated under general anaesthesia, it may need to be re-anaesthetized to recrush the cord.
- Scrotum **can be packed with gauze**, and temporarily closed with sutures or towel clamps.
- Fluid **therapy and blood transfusions** may be necessary.

Evisceration

- Evisceration may follow castration if the horse has an inapparent inguinal hernia. It may occur after several days, but it

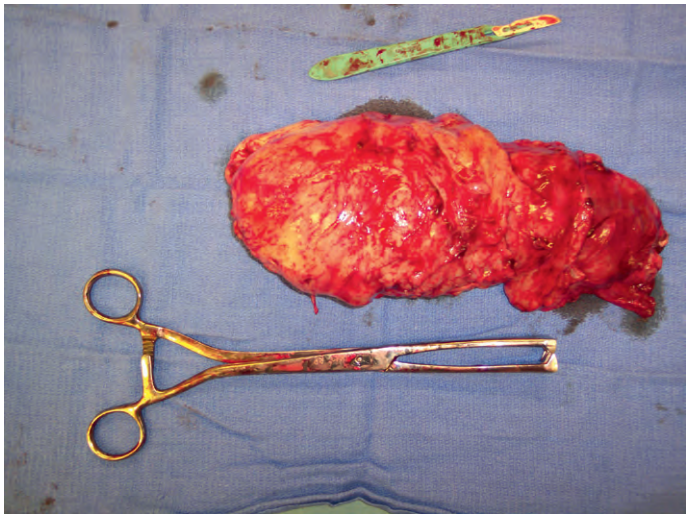


Figure 8.5 A spermatic cord chronically infected with pyogenic bacteria is commonly called a scirrhous cord. The only sure treatment of this condition is removal of the infected tissue.

most often occurs within several hours. Intestine that has entered the canal rapidly becomes strangulated. The horse should be immediately anaesthetized, and exposed intestine cleaned and replaced into the abdomen. The superficial inguinal ring should be sutured, or the canal packed with gauze. Devitalized and damaged intestine should be resected, and this is usually most easily accomplished though a ventral midline celiotomy. If only omentum is visible at the scrotal wound, the horse's vaginal ring should be palpated *per rectum* to ensure that no intestine has entered the canal, and the omentum can be transected at the scrotum.

Funiculitis

Funiculitis, or infection of the cord, can occur from extension of scrotal infection or may arise from a contaminated emasculator or ligature. A cord chronically infected with pyogenic bacteria is commonly called a **'scirrhous cord'** (Figure 8.5). Infection of the stump of the cord caused by *Streptococcus* is sometimes referred to as **'champignon'.**

Clinical signs of funiculitis include pyrexia, swelling in the inguinal region with discharging tracts, and lameness. The swelling may or may not be painful. Clinical signs may not become apparent for months after castration. Treatment:

- Antimicrobial therapy.
- Re-establishment of drainage.
- Surgical excision of the infected cord – only sure method of treatment.

Peritonitis (see also Chapter 4)

Subclinical, nonseptic peritonitis probably occurs in many horses following castration because the **vaginal and peritoneal cavities communicate**. **Non-septic peritonitis** may result from irritation of the peritoneal cavity by blood. Nucleated cell counts in excess of 10 000 cells/μL in the peritoneal fluid can frequently be found following castration and indicate peritoneal inflammation. If high nucleated cell counts are **unaccompanied by degenerate neutrophils or bacteria**, the peritonitis should be considered nonseptic.

Signs of septic peritonitis following castration may include:

- Pyrexia.
- Signs of colic.
- Diarrhoea.
- Reluctance to move.

Treatment for septic peritonitis may include:

- Antimicrobial drugs.
- Non-steroidal antiinflammatory drugs.
- Fluid therapy.
- Peritoneal lavage.
- Removal of source of peritoneal contamination, such as contaminated ligatures on the cord.

Penile damage

This is an uncommon complication of castration and usually occurs when the surgeon is unfamilar with genital anatomy and the surgical procedure. The penis can be mistaken for an inguinal testis. Laceration of the urethra may result in urethral stricture and urethral fistulae.

Hydrocele (vaginocele)

A hydrocele or vaginocele is **an idiopathic, painless, fluid-filled enlargement** in the scrotal area that may occur weeks or months after castration. Fluid fills the vaginal cavity once occupied by the testis. A hydrocele should not occur if the horse was castrated using a closed or half-closed technique. Treatment is removal of the vaginal sac through an inguinal or scrotal incision.

Continued stallion-like behaviour

Horses that continue to display stallion-like behaviour after castration are sometimes called **'false rigs'.** Purported causes include:

- **Improper castration resulting in retention of epididymal tissue.** Geldings that display stallion-like behavior and contain epididymal tissue are sometimes said to be **'proud-cut'.** The epididymis, however, contains no cells capable of producing androgens, and there is probably no such thing as a proud-cut horse.
- **Heterotopic testicular tissue**. Heterotopic testicular tissue has been found in pigs, but never in horses.
- **Production of high concentrations of androgens by the adrenal cortex**. This has never been shown to be responsible for stallion-like behaviour of geldings.
- **Psychic causes**. Stallion-like behaviour in geldings should be attributed to psychic causes. **Twenty to 30 per cent** of horses may display some stallion-like behaviour regardless of the age at which they were castrated.

8.13 CONGENITAL MONORCHIDISM

Cryptorchid horses with one descended testis are sometimes improperly referred to as monorchids. Congenital monorchidism (i.e. agenesis of a testis) is extremely rare.

8.14 CRYPTORCHIDISM

Cryptorchidism is **failure of one or both testes to descend into the scrotum**. Horses with this condition are termed cryptorchids. Horses may be affected unilaterally or bilaterally. Colloquial terms for the condition include **'rigs'** and **'ridglings'.** Types:

- **Complete abdominal cryptorchid**. Both the epididymis and testis are located within the abdomen.
- **Partial abdominal cryptorchid**. The epididymis, but not the testis, has descended into the inguinal canal.
- **Inguinal cryptorchid**. The epididymis and testis have descended into the inguinal canal, but not into the scrotum. Inguinal cryptorchids are sometimes referred to as **'high flankers'.**

A cryptorchid testis produces androgens but is incapable of producing sperm. Bilaterally affected horses are sterile.

Aetiology

Cryptorchidism may result from improper function of the gubernaculum, a mesenchymal cord that extends between the caudal pole of the testis and scrotum and guides the fetal testis from the caudal pole of the kidney to the scrotum. Or, the testis may not descend from the abdomen if it is too large to pass into the inguinal canal. The condition is inheritable, but mechanisms of inheritance are complex and have not been clearly defined.

Prevalence

Approximately 15% of two- to three-year-old colts are cryptorchid. Horses most commonly affected are Quarter Horses, Percherons, American Saddle Horses, ponies, and crossbred horses. The breed least commonly affected is the Thoroughbred.

Statistics:

- Failure of right and left testicular descent occurs with nearly equal frequency.
- 75% of left undescended testicles are retained within the abdomen.
- 40% of right undescended testicles are located abdominally.
- About 10–15% of cryptorchids are bilaterally affected.

Diagnosis

Diagnosis of cryptorchidism is based on history, palpation (both external and per rectum), hormonal assays, and surgical exploration of the inguinal canal.

- **External palpation**. The testes of prepubescent stallions are often difficult to palpate because they are small and retractile. To aid palpation, a sedative or tranquillizer can be administered to relax the cremaster muscles. Inguinal testes can sometimes be palpated, but, **because the inguinal canal of the average-sized stallion is about 10 cm deep, inguinal testes high in the canal often cannot be palpated**. The epididymis of a partial abdominal cryptorchid lies in the inguinal canal and can be mistakenly identified, by palpation, for an inguinal testis.
- **Palpation *per rectum***. Because palpation per rectum of an abdominal testis is difficult, and, because most horses presented for cryptorchid castration are young and fractious, the risk of rectal injury should be weighed against the value of diagnostic information to be gained. For an apparent gelding that displays stallion-like behaviour and whose history of castration is unknown, examination *per rectum* may be helpful. Failure to palpate an abdominal testis per rectum, however, should not be considered diagnostic. A vaginal ring of a gelding is palpable as a slight depression, but a vaginal ring of a stallion is large enough to accommodate a finger. A partial abdominal cryptorchid (i.e. one whose epididymis has descended into the canal) cannot be distinguished by examination of the vaginal rings from a horse whose testes have descended through the vaginal ring.
- **Hormonal assay**. When history and physical examination are inadequate to determine whether a horse possesses a retained testis, hormonal assays can be used to distinguish between psychic or hormonal causes of persistent stallion-like behaviour. The concentration of serum testosterone or conjugated oestrogen in the plasma or serum can be used to distinguish between geldings and cryptorchids. (See 'Diagnosis' in section 8.11.)
- **Surgical exploration on the inguinal canal.** Finding the severed ends of the spermatic cords identifies the horse as a gelding.

Treatment

- If the horse is to be used for breeding purposes (possible only in the case of unilateral cryptorchidism), no treatment is necessary. Breeding is generally not recommended because the condition is hereditary.
- Hormonal or surgical treatment to effect descent is considered unethical and is usually ineffective. Retained testes become incapable of spermatogenesis, so bringing about their descent may not increase fertility.
- Because cryptorchidism is hereditary, affected horses are usually castrated. The scrotal testis should be removed only after the cryptorchid testis has been removed!
  - If the descended testis has been removed, and the cryptorchid testis cannot be found, the owner may misrepresent and sell the horse as a gelding.
  - Removal of only the descended testis may cause the retained testis to enlarge, via negative hormonal feedback, making future cryptorchidectomy difficult.
  - Future surgery to find the testis may be difficult if there is no written record of which testis was removed.

A cryptorchid testis can be removed through an inguinal, parainguinal, paramedian, or flank approach, including laparoscopy. Except for the flank approach, the horse must be anaesthetized. **Only an abdominal testis can be removed through a paramedian or flank approach because an inguinal testis cannot be retracted into the abdomen.** Often the location of a testis cannot be determined prior to surgery. Because both abdominal and inguinal testes can be removed through an inguinal approach, determining the location of the testis prior to surgery is not necessary when this approach is used. Using an inguinal approach, an abdominal testis can be extracted through the vaginal ring or through a small incision adjacent to the medial crus of the superficial inguinal ring.

Incomplete cryptorchid castration

This occurs when the tail of the epididymis of a partial abdominal cryptorchid is mistakenly identified as a small inguinal testis and removed (Figure 8.6). The abdominal testis remains retained within the abdomen (Figure 8.7), and the horse's stallion-like behaviour persists.

8.15 INGUINAL HERNIATION AND RUPTURE

- **Inguinal herniation** – protrusion of viscera through the vaginal ring into the vaginal sac (Figure. 8.8). If the viscera descends to the scrotum, the condition is sometimes referred to as **scrotal herniation**. Inguinal hernias are sometimes referred to as **indirect hernias**, a term borrowed from a similar condition in man.
- **Ruptured inguinal herniation** – protrusion of herniated viscera through a rent in the vaginal sac (Figure 8.9).
- **Inguinal rupture** – protusion of viscera through a rent in the peritoneum and musculature adjacent to the vaginal ring. Inguinal ruptures are sometimes inappropriately referred to as **direct hernias**. Direct hernias in man are caused by weakening in the inguinal musculature and are lined by peritoneum.

Figure 8.6 This abdominal testis was removed from a horse that previously had an incomplete crytorchid castration. Note that the epididymis is missing.

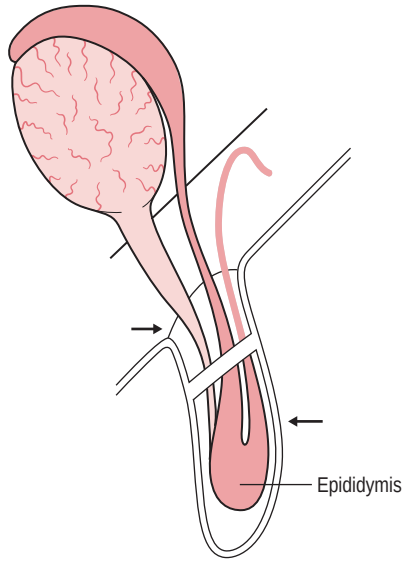


Figure 8.7 Failure to remove the abdominal testis of a partial abdominal cryptorchid can occur when the descended epididymis is mistaken for a small inguinal testis. If only the epididymis is removed, the horse retains stallion-like behaviour.
Reproduced from an article by Gayle Trotter, Journal of the AVMA 1981 178:246.

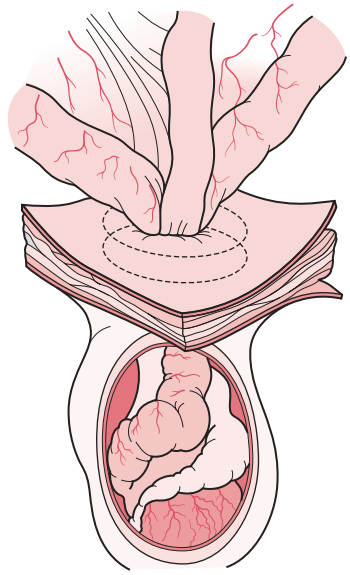


Figure 8.8 Inguinal hernia: intestine protruding through the vaginal ring into the vaginal sac. Inguinal hernias of horses are sometimes inappropriately referred to as indirect hernias.
Published in Auer JA (ed) 2005 Equine Surgery, 3rd edn. Saunders, reprinted with permission from Elsevier Ltd.

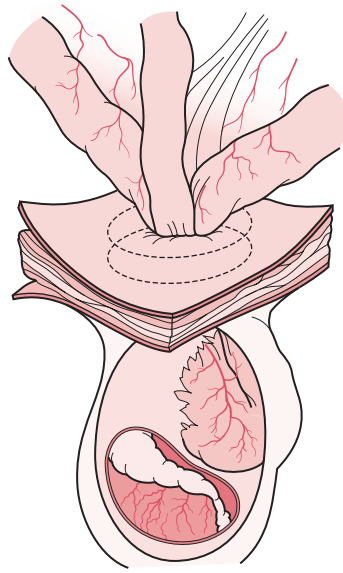


Figure 8.9 Ruptured inguinal hernia: protrusion of herniated intestine through a rent in the parietal tunic.
Published in Auer JA (ed) 2005 Equine Surgery, 3rd edn. Saunders, reprinted with permission from Elsevier Ltd.

Aetiology

Inguinal herniation may be **congenital or acquired.** Inguinal hernias of foals are congenital and are caused by an enlarged vaginal ring. Inguinal hernias of adult stallions are usually acquired and often occur during breeding or exercise.

Prevalence

Congenital and acquired inguinal hernias may occur more frequently in Standardbreds. Congenital inguinal hernias may occur unilaterally (usually the left side) or bilaterally. Ruptured inguinal hernias occur much more commonly in foals.

Clinical signs

- Congenital hernias.
  - Herniated intestine is rarely strangulated and reduces easily.
  - Palpation of a scrotal or inguinal hernia may elicit a sensation of crepitus, and peristalsis beneath the scrotal skin may be visible.
- Acquired hernias.
  - Herniated intestine becomes strangulated, which leads rapidly to severe signs of colic. It cannot be easily reduced.
  - Vasculature of the cord becomes obstructed, which leads to **scrotal and testicular oedema**.

Diagnosis

- Scrotal enlargement may suggest inguinal herniation.
- A congenital inguinal hernia may become apparent only following castration when the horse eviscerates. Inguinal area of foals should be closely examined for the presence of congenital herniation prior to castration.
- Because acquired hernias are nearly always strangulated, skin over an acquired inguinal hernia may be cold and oedematous.
- Examination *per rectum* reveals that intestine has entered a vaginal ring.

Treatment

Congenital:

- Most cogenital inguinal hernias cause no problem and spontaneously resolve by the time the foal is 6 months old.
- Application of a truss may hasten resolution.

Acquired inguinal hernias demand immediate attention because hernial contents are nearly always strangulated:

- Reduction by external manual manipulation or rectal traction on incarcerated intestine – rarely successful.
- Surgical **reduction of hernia and removal of affected testis are usually required.**

8.16 TORSION OF THE SPERMATIC CORD

Aetiology

This occurs when the testis rotates on its vertical axis. It may occur from an excessively long scrotal ligament or caudal ligament of the epididymis, which allows the testis to rotate within the scrotal fascia or parietal tunic. These ligaments are part of the gubernaculum. Rotation of the cord causes constriction of testicular blood supply.

Prevalence

Torsion of the spermatic cord is uncommon but is most often seen in Standardbreds.

Clinical signs

- Rotation of less than 180° causes no clinical signs. A tail of the epididymis located at the cranial pole of the testis indicates torsion of 180°.
- Rotation of 360° causes signs of pain and swelling of the affected testis. A swollen testis and cord with the tail of the epididymis located at the caudal pole of the testis indicates torsion of 360°.

Treatment

- Torsion of the cord that causes no clinical signs (i.e. torsion of <180°) requires no treatment.
- If torsion causes irreversible strangulation of the cord, removal of affected cord and testis is indicated.
- If the cord is not irreversibly strangulated, the testis can be permanently held in its proper position by a suture.

8.17 TESTICULAR NEOPLASIA

See Chapter 14.

8.18 INTERSEX

See Chapter 14.

8.19 DIAGNOSIS OF DISEASES OF THE PENIS AND PREPUCE

History

History pertaining to penile and preputial disease should include previous injuries, illnesses, or urogenital surgery, conception rates, drug-therapy, and duration of disease.

Physical examination

Physical examination of a horse with penile or preputial disease may include the following:

- Observation of urination. Urination can be stimulated by placing the horse in a freshly bedded stall or by administering furosemide.
- Visual inspection. The penis of a stallion can be protruded by administering xylazine or by causing sexual arousal. **Phenothiazine-derivative tranquillizers probably should not be administered to stallions because they have, on rare occasions, caused penile paralysis or priapism.**
- Palpation.
- Observation of erection and ejaculation.
- Ultrasonography.
- Cavernosography.

8.20 PENILE AND PREPUTIAL INJURIES

Aetiology

The penis and prepuce can become damaged from:

- Failed attempts at jumping barriers.
- Masturbation.
- Kicks.
- Attempting to breed a mare over a fence, or from breeding a mare with a loosely tied breeding stitch.
- Improperly fitted or dirty stallion rings.
- Poor technique of castration.

Unsutured preputial and penile lacerations become infected, and cellulitis and swelling of the preputial and penile integument ensues. **The penis and internal lamina of the prepuce may prolapse through the preputial orifice, and if left untreated, paralysis and permanent paraphimosis can result.**

Treatment

- Fresh penile and preputial wounds should be sutured.
- Infected wounds should be cleansed and treated with antimicrobial ointment.
- If the penis has prolapsed, it should be replaced into the external prepuce and secured. If the penis is so swollen that it cannot be replaced, it should be wrapped against the ventral abdomen.
- Penile haematomas should be treated with compression wraps and hydrotherapy.
- If the penis is severely damaged, the most expedient treatment may be amputation, particularly if the horse is a gelding.

8.21 PARAPHIMOSIS

Aetiology

Paraphimosis, or inability of a horse to retract its penis into the prepuce, is caused by:

- Preputial oedema caused by genital trauma or systemic disease (e.g. dourine (see Chapter 19) and purpura haemorrhagica (see Chapter 10)).
- Damage to penile innervation. Can occur with spinal disease, trauma, and infectious diseases.
- Debilitation.
- Phenothiazine-derivative tranquillizers (generally not a problem in geldings).

Pathophysiology

- Prolapse impairs venous and lymphatic drainage.
- Penis and internal preputial lamina swell from oedema, and preputial ring constricts distal penis.
- Oedema makes tissues fragile, and penile and preputial integument becomes excoriated.
- Excoriated integument becomes infected.
- Fibroblasts invade penile integument and fascia, permanently impairing the normal invaginating action of the prepuce.
- Pudendal nerves become stretched and damaged, causing penile paralysis.
- Urination is unaffected, but **impotence usually accompanies paralysis**.

Treatment

Penile and preputial oedema and further trauma should be prevented by:

- Replacing the prolapsed penis within the external preputial lamina and retaining it with sutures, or towel clamps, or nylon netting placed across the preputial orifice.
- Compressing the prolapsed penis against the abdomen with wraps if it cannot be replaced within the external preputial lamina.
- Applying antimicrobial ointments to prevent epithelial excoriation and infection.
- Administering non-steroidal anti-inflammatory drugs to reduce swelling.
- Performing a **preputiotomy** if the inelastic preputial ring prevents penile retraction.

If paraphimosis is longstanding, the following measures must be taken:

- If preputial scarring prohibits replacement of the penis within the external preputial lamina of the prepuce, a **segmental posthetomy (reefing)** may be performed.
- If the penis is paralysed, permanent placement of the penis into the prepuce (phallopexy) or amputation of the penis may be indicated.

8.22 PHIMOSIS

Aetiology

Phimosis (i.e. **the inability of the horse to protrude its penis from the prepuce**) can occur from congenital or acquired stricture of the preputial orifice or preputial ring.

- Congenital phimosis rarely occurs in horses. (Fusion of the internal lamina of the prepuce to free part of penis is normal during first month after birth.)
- Acquired phimosis can be caused by tumours or damage at the preputial orifice or preputial ring.

Treatment

A triangle of external preputial fold based toward the preputial orifice is removed if the preputial orifice is constricted. If the preputial ring is constricted, a similar wedge is removed from the internal preputial fold.

8.23 PRIAPISM

Aetiology

Priapism (i.e. **persistent erection without sexual arousal)** is uncommon in stallions but is economically significant because impotence is the usual outcome. It occurs when disturbances in arterial inflow or venous outflow prevent detumescence of the erect corpus cavernosum penis (CCP). Vascular stasis and oedema occur, leading to fibrosis of the CCP and irreversible occlusion of the collecting veins. The condition in horses **usually occurs following administration of phenothiazine-derivative tranquilizers.**

Treatment

Because penile protrusion may eventually damage the pudendal nerves, the problem should be resolved as rapidly as possible.

- Further damage to the penis should be prevented with massage, emollient dressings, and slings.
- Cholinergic blockade to bring about detumescence – usually unsuccessful.
- **Irrigation of CCP**. If medical treatment is unsuccessful within 6 to 8 hours, the CCP should be irrigated with heparinized lactated Ringer's solution (LRS) to evacuate coagulated blood.
- Anastomosis of the CCP and CSP (corpus spongiosum penis). If erection recurs after several cavernosal evacuations, the CSP can be anastomosed to the CCP because the CSP is not involved in erection and can serve as an outlet for stagnant blood.

8.24 NEOPLASIA

Incidence

The most common neoplasm by far of the penis and prepuce is the **squamous cell carcinoma**.

- It occurs most commonly in old horses and in breeds with non-pigmented genitalia.
- Has a low grade of malignancy and grows surprisingly slowly for carcinomas.
- Metastasis to inguinal lymph nodes occurs late in disease.

Treatment

- Cryosurgery.
- Hyperthermy.
- Excision.
- Segmental posthetomy (reefing) or phallectomy.
- En bloc resection of penis, prepuce, and superficial inguinal lymph nodes.

8.25 CUTANEOUS HABRONEMIASIS (OR 'SUMMER SORE')

Uncommon in Europe.

Aetiology

- Granulomatous disease caused by cutaneous migration of the larvae of the equine stomach worm, *Habronema*. Not endemic in Europe but common in the USA.

- Preputial ring and orifice and urethral process are common sites of larval infestation.
- Lesions appear in spring and summer when flies, which are the intermediate host, are prevalent. Lesions usually disappear with appearance of cold weather.
- Lesion may recur yearly on some susceptible horses.

Clinical signs

- Lesions are pruritic.
- Lesions are characterized by exuberent granulation tissue that contains numerous, small, yellow, sand-like, caseous granules. Skin lesions are usually round (Figure 8.10).
- Mucous membrane of the affected urethral process may be hyperaemic and protrude from the urethral orifice.
- Lesions of the urethral process may cause horse to spray urine on its limbs during urination.
- Erosions into the CSP of the urethral process may result in haemospermia and haematuria.

Diagnosis

Based on:

- Appearance and clinical signs.
- Marked circulating eosinophilia of affected horses.
- Identification of larvae microscopically in exudate extruded from lesions.
- Histological identification of eosinophils, granules, and larvae.

Treatment

Medical treatment is aimed at:

- Eliminating larvae.
  - Ivermectin.
  - Organophosphates (topical, oral, or intravenous).
- Decreasing the horse's hypersensitivity to the larvae.
  - Prednisolone administered for several weeks.
  - Diethylcarbamazine administered for 1 to 2 weeks.

Surgical treatment:

- Elliptical resection of small, isolated preputial lesions.
- Segmental posthetomy for large lesions.
- Amputation of affected urethral process.
- Cryotherapy.

Figure 8.10 Lesions infected with *Habronema* larvae are characterized by exuberent granulation tissue that contains numerous, small, yellow, sand-like, caseous granules. Skin lesions are usually round.

8.26 HAEMOSPERMIA

Aetiology

Haemospermia (i.e. **blood in ejaculate**) causes **infertility** and has been attributed to:

- Bacterial urethritis, usually at area of ejaculatory ducts.
- Urethral rents of unknown origin that communicate with the CSP on the convex surface of the urethra at the level of the ischial arch (Figures 8.3 and 8.11).
- Habronemiasis of the urethral process.
- Stallion-rings.
- Seminal vesiculitis.

Diagnosis

- Gross and microscopic examination of ejaculate.
- Clinical signs.
  - Affected stallion may require several mounts to ejaculate.
  - May exhibit pain on ejaculation.
- Urethroscopy can be performed to locate the source of haemorrhage.
- Culture and cytologic examination of haemorrhage obtained by uretheral swabs.

Treatment

- Sexual abstinence.
- Medical treatment:
  - Formalin (IV).
  - Methenamine (PO) produces antibacterial concentrations of formaldehyde in the urine.
  - Antimicrobial drugs administered systemically.
- **Temporary perineal urethrostomy combined with sexual rest has been most effective in eliminating haemospermia.** Success

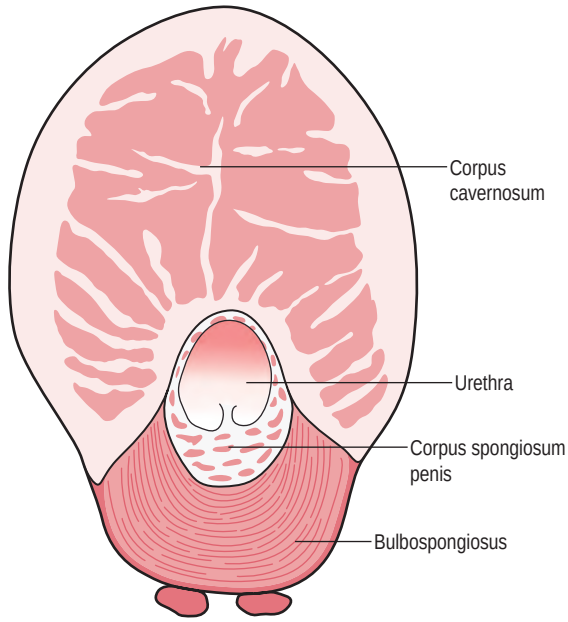


Figure 8.11 A urethral rent that communicates with the CSP. Urethral rents are usually found at the convex surface of the urethra at the level of the ischial arch.
Reprinted from McClure SR, Honnas CM, Schumacher J, Hoffman AG 1995 Permanent Trachesyomy in Standing Horses: technique and results in Vet. Surg. 456–458. Reprinted with permission of John Wiley and Sons.

of temporary urethrostomy has been attributed to decreased pressure in the CSP during ejaculation and urination because the CSP is probably the source of haemorrhage.

8.27 DIAGNOSIS OF DISEASES OF THE FEMALE REPRODUCTIVE TRACT

History

There is a need to accurately describe the mare's reproductive status. There is a need for information about previous uterine infection and treatment, abortions, twinning, neonatal deaths, and treatment history.

Examination

Examination may include the following:

- Determination of general health of the mare.
- Assessment of conformation of the perineum and pelvis.
- Identifcation of vulvar discharge or lesions.
- Palpation *per rectum* of the ovaries, uterus, and cervix.
- Ultrasonography.
- Uterine culture. Cultures taken during oestrus are more meaningful. Positive cultures are meaningless without evidence of endometritis (vaginal discharge or histological or cytological evidence).
- Histological examination of endometrial biopsy. Biopsies taken during dioestrus are more meaningful.
- Vaginoscopy.
- Digital palpation of the vagina and cervix.
- Endometrial cytology.

Less common procedures include:

- Endoscopic examination of the uterus (hysteroscopy).
- Laparoscopy.
- Determination of serum concentrations of hormones.
- Karyotyping in cases of unexplained infertility.

8.28 PNEUMOVAGINA

See Chapter 14.

8.29 PERINEAL LACERATIONS AND FISTULAE

Aetiology and incidence

- Occur at parturition when the foal's front foot or nose catches the annular fold of the hymen at the vaginovestibular junction.
- Occur predominately in primiparous mares because their annular fold at the vaginovestibular junction is more prominent than that of pluriparous mares.

Classification

- **First-degree perineal lacerations** involve the skin and mucous membrane of the dorsal commissure of the vestibule.
- **Second-degree perineal lacerations**. Constrictor vulvae muscle is also disrupted.
- **Third-degree perineal lacerations**. These are a complete disruption of tissue between the rectum and vestibule

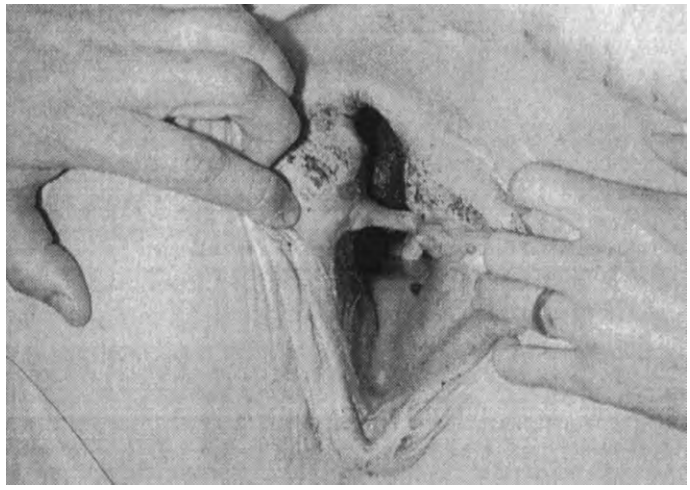


Figure 8.12 A third-degree rectovestibular laceration. These occur during foaling and result in a common rectal and vestibular vault.

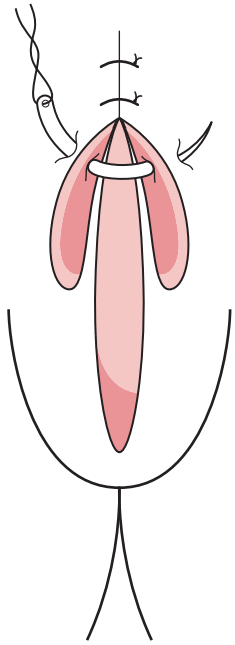


Figure 8.13 The Caslick operation is performed to prevent pneumovagina. The labia should be apposed with sutures, except for those portions that lie below the level of the pelvic floor.

(Figure 8.12). Injury creates a common rectal and vestibular vault. The condition is referred to a rectovaginal, or rectovestibular, or R–V laceration. Most are rectovestibular rather than rectovaginal. Third-degree rectovestibular lacerations usually allow faecal contamination of the mare's vagina with subsequent bacterial infection of the vagina, cervix, and uterus.

- **Rectovestibular fistulae**. Tissue between the rectum and vestibule is completely perforated, but the perineal body, or caudal portion of it, remains intact.

Treatment

- First-degree perineal lacerations. Mares with first-degree perineal lacerations can be treated with a Caslick vestibuloplasty (Figure 8.13).

- Second-degree perineal lacerations require more extensive treatment because of disruption of the constrictor vulvae muscle. If only a Caslick vulvoplasty is used to repair second-degree perineal lacerations, the perineum sinks, predisposing the mare to pneumovagina and urine pooling. Repair is sometimes called a vestibuloplasty. The aim of vestibuloplasty is to reduce the diameter of the vestibule by 30–50%.
  - Usually best to wait 4 weeks before repairing second-degree perineal lacerations.
  - Repair is similar to reconstruction of the perineal body in repair of R–V lacerations and is the same technique used to treat pneumovagina caused by faulty perineal conformation.
  - Repair involves removal of two right triangles of mucosa whose base is the cutaneous perineum and whose hypotenuse is the new dorsum of the vestibule. Fresh tissue exposed by removal of the triangles is apposed, thus lowering the roof of the vestibule.
- **Third-degree perineal lacerations**. Mares require more preoperative preparation and more postoperative care because the rectum is involved in the injury. **Attempts to repair the laceration immediately after injury are usually unsuccessful** because the lacerated tissue rapidly becomes inflamed and oedematous. Contraction of the muscles of the rectum and vagina widen and lengthen the wound. Usually best to wait 4 to 6 weeks before attempting repair. For acute injuries, the following treatment should be considered:
  - Tetanus prophylaxis.
  - Broad-spectum antimicrobial drugs for 2 or 3 days.
  - Stool-softeners, such as mineral oil, for several days to prevent painful defecation.
  - Debriding the wound of devitalized tissue.

Preparation for surgery of third-degree lacerations includes:

- Weaning the foal before the laceration or fistula is repaired. Prevents exposing foal to nosocomial disease.
- Palpation of the reproductive tract per vagina and rectum to detect cervical lacerations, pregnancy, uterine adhesions, and pyometria.
- Histological examination of the endometrium. May be indicated if the mare has gone through more than one reproductive season with an R–V laceration, especially if the mare has faulty perineal confirmation. Urine and faeces in the vagina may gravitate toward the uterus if the mare suffers from poor perineal confirmation. If the mare has severe fibrosis of the endometrium, surgery may be ineffective in returning the mare to reproductive soundness.
- Softening the mare's stool and decreasing its bulk. The ration should allow the mare to maintain weight. Administering 1 gallon of mineral oil *per os* by stomach tube the day before surgery assures that the stool is soft and unformed at surgery. Raw linseed oil, administered in the feed, is an effective stool softener.
- Administration of a broad-spectum antibiotic several hours prior to surgery.

The R–V laceration or fistula can be repaired with the mare sedated and standing or anaesthetized and in dorsal recumbency. There are two stages to the procedure:

1. Rectovestibular reconstruction.
2. Anoperineal reconstruction.

Both stages may be performed during the same operation, or the anoperineal reconstruction may be completed 3 weeks or more after rectovestibular reconstruction. If anoperineal reconstruction is postponed until the rectovestibular reconstruction is healed, less stress is placed on the rectovestibular reconstruction during defecation. **Perfoming both procedures during the same operation, however, minimizes hospitalization time, dietary changes, and expense.**

Figure 8.14 A rectovestibular fistula can be repaired using the technique described by Forssell, which spares the intact perineal body. The skin of the perineum is incised in a frontal plane between the anus and the dorsal commissure of the labia.

- Rectovestibular fistulae.
  - R–V fistula can be converted into a laceration for repair. A fistula should be converted into a laceration only if the fistula is exceptionally large.
  - The fistula can be repaired using the technique described by Forssell, which **spares the intact perineal body**. The skin of the perineum is incised in a frontal plane between the anus and the dorsal commissure of the labia (Figure 8.14). The incision is deepened cranially beyond the fistula. This separates the rectovestibular hole into two holes: a rectal hole and a vestibular hole. The rectal hole is closed in a transverse plane (because the musculature of the rectum is primarily circular, and sutures placed perpendicular to the muscle fibres are subject to less stress than sutures placed parallel to the direction of the muscle fibres). The vestibular hole is closed in a sagittal plane. The horizontal plane of dissection is quilted closed.
  - Preoperative and postoperative management of mares with an R–V fistula is similar to that of mares with an R–V laceration.

Postoperative care after repair of R–V lacerations and fistulae

Postoperative treatment of the mare after repair of an R–V laceration or fistula usually includes:

- Broad-spectum antimicrobial drugs administered for several days.
- Tetanus prophylaxis, if not previously administered.
- Stool should be kept soft and scanty for at least 8 days by modifying diet and administering stool softeners (e.g. mineral oil or raw linseed oil).
- Non-steroidal anti-inflammatory drugs may be indicated if the mare shows signs of discomfort after surgery.
- Repair should not be evaluated until the 8th or 9th postoperative day.

- Mares that strain excessively following surgery should be treated by epidural anaesthesia and sedation. **Causes of straining include faecal impaction of the rectum and cystitis**.

Other postoperative considerations

Mares are reproductively healthy before acquiring third-degree rectovestibular lacerations, and reproductively healthy mares are usually able to eliminate bacteria from the tubular genital tract within one oestrous cycle. **Not all mares develop endometritis after third-degree rectovestibular injury. Some even become pregnant.** Those that do develop endometritis appear to be capable of rapidly resolving chronic inflammation after perineal repair.

Natural breeding should not be allowed for at least two months after repair of a third-degree perineal laceration, but **mares may be bred by artificial insemination within two weeks after perineal repair**. After two weeks of convalescence, sufficient healing should have occurred to permit safe examination of the reproductive tract *per rectum* and passage of an insemination pipette through the cervix.

8.30 UROVAGINA

See Chapter 14.

8.31 CERVICAL LACERATIONS

See Chapter 14.

8.32 NEOPLASIA

Perineal neoplasms

Common perineal neoplasms include melanomas of grey mares and squamous cell carcinomas of mares with non-pigmented genitalia (i.e. cremellos, Appaloosas, paints, and palominos).

Treatment

- Melanomas. Genital melanomas of grey mares are often not treated because they usually develop slowly and do not interfere with intromission. Melanomas on horses of other colours tend to be malignant. Small tumours can be excised or removed with cryotherapy. If tumours are too numerous or too large to be treated in this manner, cimetidine may stop progression and decrease the size and number of tumours.
- Squamous cell carcinomas.
  - Surgical excision.
  - Cryotherapy.
  - Hyperthermy.

Neoplasms of the tubular genital tract

Neoplasms of the tubular genitalia are rare, but leiomyomas have been most frequently reported (Figure 8.15). Partial or total hysterectomy may be indicated.

Ovarian neoplasms

See Chapter 14.

Figure 8.15 Neoplasms of the tubular genitalia are rare, but leiomyomas have been most frequently reported. The mass in the uterine body is a leiomyoma.

8.33 UTERINE TORSION

See Chapter 14.

8.34 RETAINED PLACENTA

See Chapter 14.

8.35 DYSTOCIA

Causes (see Chapter 14)

- **Relative undersize of the mare compared to the foetus is an uncommon cause of dystocia** but can occur in primiparous mares or in mares allowed to graze tall fesque pasture infected with endophyte (*Neotyphodium coenophalum*) during late gestation.
- Abnormal presentation, position, or posture. Often the result of fetal death. A dead foal does not assume the proper birthing position. Long limbs of the foetus predispose to dystocia.
- Uterine torsion – **estimated to cause 5–10% of dystocias**.
- Pelvic fracture.
- Prepubic tendon rupture.

Treatment

- Vaginal delivery after repositioning the fetus. Most easily accomplished with the mare anaesthetized and the hindquarters elevated with a hoist. If the foal is dead, fetotomy may be required.
- Caesarean section. Indicated when vaginal delivery is not possible or is proceeding too slowly to result in birth of a live foal.
  - **The incised uterine wall of the horse bleeds more profusely than does that of other species**, and the edges must be sutured to achieve haemostasis before the uterine incision is closed.
  - Incidence of retained placenta is high following caesarean section.

8.36 OVARIECTOMY (OOPHORECTOMY)

Indications

- The most common indication for unilateral ovariectomy of the mare is removal of a granulosa cell tumour.
- Other indications for unilateral ovariectomy include other, less common, ovarian neoplasia.
- Bilateral ovariectomy may be indicated for mares with a congenital defect that prohibits breed registration without bilateral ovariectomy.
- Most common indication for bilateral ovariectomy is to alleviate undesirable behaviour attributable to a hormonal influence.

Approaches

Approaches for ovariectomy:

- Flank.
- Ventral midline.
- Oblique paramedian (Figure 8.16).
- Vaginal (**colpotomy** approach). This is a standing approach often used for bilateral removal of non-tumorous ovaries. Advantages of this approach are:
  - Ovariectomy is performed without general anaesthesia, so ovariectomy using this approach is less expensive, and, because the incision is usually not sutured, the technique is quickly performed.
  - Mare can be returned to athletic function sooner with this approach than with ovariectomy performed using other approaches.

  Disadvantages of this approach are:
  - Requires the use of a chain ecraseur (Figure 8.17)
  - The surgeon is at some risk of injury.
  - Because the ovarian pedicles cannot be ligated, the **horse is at greater risk of haemorrhage** than with ovariectomy performed using an approach that allows for ligation of the pedicles.
  - Extracting large, tumorous ovaries through the colpotomy site is impossible.
- Laparoscopy. Laparoscopic ovariectomy may be performed either in the standing mare or under general anaesthesia. This minimally invasive technique has fewer potential complications than the traditional surgical approaches.

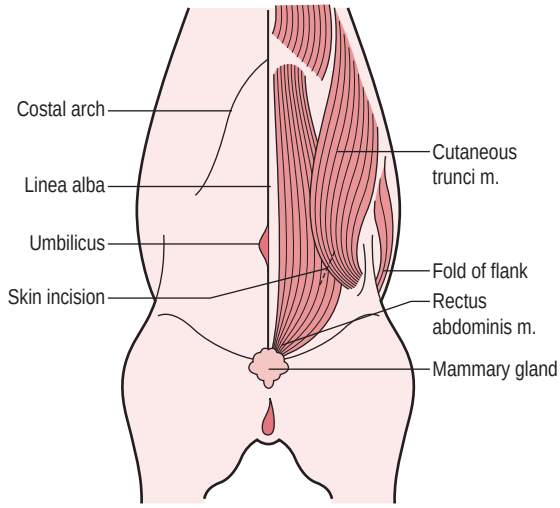


Figure 8.16 Paramedian approach to removal of a neoplastic ovary. *Reprinted from Moll DH, Slone DS, Juzwiak JS, Garrett PD 1987 Diagonal Paramedian Approach for Removal of Ovarian Tumors in the Mare, in Vet. Surg. 456–458. Reprinted with permission of John Wiley and Sons.*

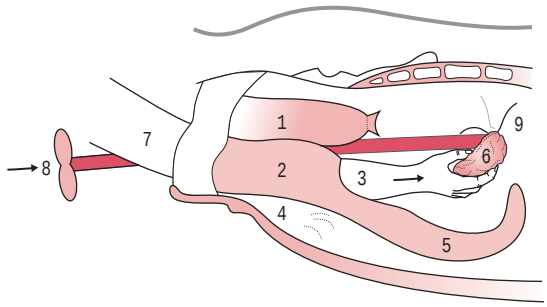


Figure 8.17 Vaginal approach to ovariectomy via colpotomy. Lateral view and important anatomical areas are identified. Rectum (1); vagina (2); colpotomy incision (3); bladder (4); uterine horn (5); ovary (6); operator's arm (7); ecraseur (8); mesovarium (9).

FURTHER READING

Bayly W (1991) A practitioners' approach to the diagnosis and treatment of renal failure in horses. Veterinary Medicine 86:632–639

Brown CM (1986) Equine nephrology. In: Grunsell CSG, Hill FWB, Raw ME (eds) The veterinary annual. John Wright, Bristol, UK, pp 1–16

Cox JE (1987) Surgery of the reproductive tract in large animals. Liverpool University Press, Liverpool

Knottenbelt DC, Holdstock N, Madigan JE (2004) Equine neonatology. Saunders, New York, pp 324–327

Kohn CW, Chew DJ (1987) Laboratory diagnosis and characterization of renal disease in horses. Veterinary Clinics of North America: Equine Practice 3:3. Saunders, Philadelphia, pp 585–615

Schumacher J (1990) Surgery of the testicle and associated structures. In: Auer JA (ed) Textbook of equine surgery. Saunders, Philadelphia, pp 674–703

Schumacher J (2012) Penis and prepuce. In: Auer JA and Stick JA (eds) Equine surgery 4th ed. Elsevier Saunders, St. Louis, pp 840–866

Van Metre DC (2009) Diseases of the renal system. In: Smith BP (ed) Large animal internal medicine, 4th ed. Mosby, Elsevier, St Louis, pp 925–971

Varner DD, Schumacher J, Blanchard TL, Johnson L (1991) Diseases and management of the breeding stallion. American Veterinary Publication, Goleta, CA, p 349

Chapter 9

Endocrinology

Nicholas Frank

CONTENTS

9.1 Anhidrosis (dry coat syndrome) 181
9.2 Pituitary pars intermedia dysfunction (PPID) or equine Cushing's disease (ECD) 182
9.3 Hypoadrenocorticism (adrenal insufficiency, turn-out syndrome, steroid let down syndrome) 185
9.4 Phaeochromocytoma 185
9.5 Insulin resistance (IR)/equine metabolic syndrome (EMS) 186
9.6 Hyperlipaemia 188
9.7 Hypothyroidism 190
9.8 Hyperthyroidism 191
9.9 Thyroid adenoma 191
9.10 Hyperparathyroidism 192
Further reading 193

9.1 ANHIDROSIS (DRY COAT SYNDROME)

Anhidrosis is characterized by an inability to sweat effectively in response to hot weather or exercise. This problem can affect adult horses of all ages, breeds, and coat colour. It sometimes occurs in horses that have recently been transported to regions with a warmer climate, but can also develop in horses that have remained in the same location for many years. Racehorses that are transported from northern states to the southern region of the United States during winter months are susceptible to anhidrosis and it is a concern for showhorses that compete in different regions. Affected horses can show partial or complete failure to sweat and this problem can significantly limit athletic performance.

Aetiology

Anhidrosis is thought to result from the down-regulation or desensitization of sweat gland β_2-adrenergic receptors, but the exact cause of this problem remains unknown. Horses from cooler climates are not predisposed to anhidrosis, but the problem is often first recognized when affected horses are moved into regions with hotter climates.

Pathogenesis

Horses sweat to cool the body through evaporation and this process accounts for up to 65% of heat loss in exercising horses. Sweating is stimulated by neural responses and circulating catecholamines that increase in concentration during exercise. Cooling mechanisms are controlled by thermoregulatory centres within the hypothalamus that initiate sweating in response to rising body temperature. Autonomic adrenergic neurons stimulate equine sweat glands and β_2 adrenergic receptors respond to agonists such as epinephrine that circulate within the blood. Denervation is an unlikely cause of anhidrosis because this disease is generally reversible and sweat can be produced by denervated horse skin. It is also unlikely that anhidrosis is caused by loss of central nervous system control because exogenous β_2 adrenergic agonists, including epinephrine, salbutamol and terbutaline, do not stimulate sweating. It is therefore likely that anhidrosis results from down-regulation of β_2 adrenergic receptors or interference with receptor function or signalling. With time, glandular cells undergo degeneration and ducts become blocked with cellular debris.

Clinical signs

- Exercise intolerance is often the first sign observed by the horse owner and, in milder cases, this may be the only clinical sign observed. Some horses can sweat during exercise, but the response is suboptimal.
- An inappropriately elevated respiratory rate may be detected. Tachypnoea is to be expected during exercise, but respiratory rates >100 breaths/min may be detected in affected horses and tachypnoea can persist for longer than expected after exercise.
- Decreased or absent sweat production is the best known characteristic of this disease, but this can be difficult to appreciate in milder cases. Sweating is sometimes judged to be inadequate for the ambient temperature and level of exercise. Horses may sweat in certain regions of the body, but fail to show the appropriate degree of sweating overall. Exercising horses should sweat across the neck, beneath the mane, within the axillary region, between the hindlegs, and around the perineum. Sweat should also be present beneath the saddle.

© 2013 Elsevier Ltd
DOI: 10.1016/B978-0-7020-2801-4.00009-2

- Dry flaky skin and alopecia can develop with chronic anhidrosis, but this clinical sign is rare.

Diagnosis

- Anhidrosis is a differential diagnosis for exercise intolerance.
- Subjective assessment of sweating and measurement of the respiratory rate during and after exercise.
- Elevation of the rectal temperature for greater than 30 minutes after exercise has been completed may be suggestive of anhidrosis.
- Intradermal terbutaline response test – terbutaline sulphate is a selective β_2 agonist that stimulates sweating. Terbutaline sulphate (1 mg/mL) is injected intradermally (0.10 mL volume) at regularly spaced intervals along the neck at dilutions (with saline) of 1 : 1000 (10^{-3}); 1 : 10 000 (10^{-4}); 1 : 100 000 (10^{-5}), 1 : 1 000 000 (10^{-6}), and 1 : 10 000 000 (10^{-7}), along with a negative control injection of physiological saline. When evaluated 30 minutes later, most horses sweat at all sites. Horses with partial anhidrosis fail to sweat at the lowest concentration and severe anhidrosis is associated with the absence of sweating at any site or only the highest concentration. It is advisable to test a healthy horse at the same time to ensure efficacy of the drug.
- Alternatively, an epinephrine response test can be performed. Epinephrine solutions of 1 : 1000, 1 : 10 000, 1 : 100 000, and 1 : 1 000 000 are injected intradermally along the neck and sweating should occur at all four injection sites within minutes in a normal horse. Anhidrotic horses sweat only at the highest concentration site and this may take longer to develop. The test should be performed in the region of the neck covered by the mane because epinephrine injections can occasionally cause hairs to turn white.
- Clinicopathology – haematological and biochemical values (including electrolytes) generally remain unaffected.
- Serum total triiodothyronine (tT_3) and total thyroxine (tT_4) concentrations are sometimes measured in affected horses because of the anecdotal association between anhidrosis and hypothyroidism. However, detection of lower serum thyroid hormone concentrations is not diagnostic for primary hypothyroidism in the horse and an assay for thyroid-stimulating hormone (TSH) is not commercially available. Abnormal tT_3 and tT_4 responses to exogenous thyrotropin-releasing hormone (**TRH**) must be documented to support a diagnosis of hypothyroidism in the horse.
- Skin biopsy – histopathological abnormalities include cellular degeneration as a result of thermal stress and occlusion of ducts with debris. These findings provide supportive evidence of anhidrosis, but are not diagnostic for the condition.

Treatment and prevention

- Exercise reduction – mildly or moderately affected horses should not be worked strenuously for 30 days. This provides time for spontaneous resolution.
- Environmental management – mildly affected horses are managed by housing them in air conditioned stalls, whereas severely affected horses are sometimes sold to owners that live in colder climates. Housing horses in a cooler environment prevents ongoing thermal damage, which allows regeneration of sweat glands over time. After rehabilitation, horses can be exercised during the cooler times of the day.
- Oral electrolytes – hypokalaemia may play a role in anhidrosis, so oral supplementation with potassium chloride is recommended.
- A feed supplement containing L-tyrosine, choline bitartrate, niacin, pyrodoxine HCl, and D-calcium pantothenate (One AC, Magic Powder Co., Phoenix, Arizona, USA) is commonly used in the United States. Tyrosine is a precursor for dopamine and therefore catecholamines and may be involved in the resensitization of sequestered β_2 receptors. Very little scientific evidence is available to support the use of this product and it cannot be determined if positive results are partially a result of the manufacturer's recommendation to avoid intense exercise for 3 weeks after initiating treatment.
- Levothyroxine sodium – this treatment remains popular despite a lack of scientific evidence because it is thought that hypothyroidism and anhidrosis are associated in horses.

9.2 PITUITARY PARS INTERMEDIA DYSFUNCTION (PPID) OR EQUINE CUSHING'S DISEASE (ECD)

This is the most common endocrinopathy affecting older horses and ponies. Pituitary pars intermedia dysfunction (**PPID**) should be considered as a potential medical problem in any horse over 10 years of age. The pathognomonic clinical sign of PPID is an abnormally long hair coat, which is referred to as hirsutism (Figure 9.1). There is no sex predilection, and all breeds of horses, ponies and donkeys are affected, but the risk of PPID increases with age.

Aetiology

Advanced PPID is caused by the presence of a small tumuor(s) within the pars intermedia of the pituitary gland. This tumour is active and produces hormones and other peptides with hormone-like activity. In humans, we refer to the anterior and posterior pituitary gland, but these terms are not relevant when discussing the equine pituitary gland because of anatomical differences between species. The anterior pituitary gland of the horse is better described as the pars distalis and the posterior pituitary should be referred to as the pars nervosa. The pars intermedia can be found between these structures. The pars distalis secretes six hormones: adrenocorticotropin hormone (**ACTH**; also called corticotropin), thyroid-stimulating hormone (**TSH**; also called thyrotropin), growth hormone (also called somatotropin), follicle-stimulating hormone, luteinizing hormone, and prolactin. In the healthy animal, melanotropes of the pars intermedia primarily secrete alpha melanocyte-stimulating hormone (**α-MSH**). Oxytocin and anti-diuretic hormone (**ADH**; also called vasopressin) are secreted by the pars nervosa.

This condition is described as pituitary dysfunction because hyperplasia and hypertrophy occur before neoplasia develops. These events are attributed to degeneration of dopaminergic neurons over time in susceptible animals. This degeneration occurs through oxidative damage and perhaps inflammation, but individual horses differ in their susceptibility, and other factors including diet and concurrent endocrine disorders such as equine metabolic syndrome may accelerate the process. Loss of dopaminergic neurons causes a decrease in dopamine secretion and therefore, loss of inhibition. This permits cell proliferation and expansion, which can be followed by neoplasia. Alterations in structure also occur with age and changes in season, so it is sometimes difficult to diagnose PPID on the basis of histopathology results alone.

It has been suggested that adrenal tumours are a cause of hyperadrenocorticism in some horses. However, this condition is rare and should be confirmed by an ACTH challenge test if suspected. In most cases, hyperadrenocorticism is attributable to pituitary dysfunction in horses.

Figure 9.1 A horse with advanced hirsutism attributed to pituitary pars intermedia dysfunction (PPID).

Clinical signs and proposed pathogenesis

In the healthy state, the hypothalamic-pituitary-adrenal axis begins with the production of corticotropin-releasing hormone (**CRH**) from the hypothalamus, which stimulates corticotropes within the pars distalis of the pituitary gland to produce the prohormone pro-opiomelanocortin (**POMC**). This large peptide is cleaved by prohormone convertase I to generate ACTH, which enters the circulation and stimulates cortisol release from the adrenal cortex. Circulating cortisol exerts negative feedback effects on both the hypothalamus and pars distalis. Hormone production within the pars intermedia also begins with POMC synthesis, but two enzymes are active within this region of the pituitary gland – prohormone convertase I that cleaves ACTH from POMC and prohormone convertase II that converts ACTH into α-MSH. Only α-MSH is normally secreted from the pars intermedia.

Loss of dopaminergic inhibition permits excessive POMC production by the pars intermedia, which increases the secretion of α-MSH and ACTH. Production of ACTH by the pars intermedia is not under negative feedback control, so hyperadrenocorticism develops over time. Other products of the POMC pathway are also produced in greater quantities, including beta endorphin, corticotropin-like intermediate peptide (**CLIP**), other melanocyte stimulating hormones, and β-lipotrophin.

- Laminitis – horses with PPID are more susceptible to insidious-onset laminitis. Hyperadrenocorticism promotes vasoconstriction and protein depletion within the dermis and epidermis, which may lower the threshold for agents that initiate laminitis. Horses or ponies with PPID are predisposed to laminitis triggered by grazing on pasture. This predisposition may be a result of concurrent insulin resistance (IR) and hyperinsulinemia. It is therefore important to diagnose and manage IR when treating a horse or pony with PPID.
- Hirsutism – this clinical sign is considered to be pathognomonic for PPID in older horses, and has been used as the gold standard for diagnosing the disease. Early evidence of hirsutism includes retention of the winter hair coat for longer than expected or detection of longer hairs on the palmar or plantar aspects of the lower leg. Hairs are arrested in telogen and may become lighter in colour over time. Hirsutism is attributed to alterations in MSH secretion or increased production of androgens by the adrenal cortex.
- Body composition changes – skeletal muscle mass decreases as PPID develops and is often accompanied by regional adiposity. Type 2A (oxidative-glycolytic) and 2B (glycolytic) muscle fibres undergo atrophy. Regional adiposity is characterized by expansion of adipose tissues in specific regions, including the neck (commonly referred to as a 'cresty neck'), either side of the tail head, and prepuce. Cortisol-induced protein catabolism results in a thinner body condition, loss of epaxial muscle mass, and rounding of the abdomen.
- Polyuria/polydipsia – owners report excessive water consumption and urination. Polyuria can be attributed to ACTH- or cortisol-mediated antagonism of ADH at the collecting tubule, glucosuria, or reduction in ADH secretion by the pars nervosa (diabetes insipidus) caused by compression or invasion of the pars nervosa by neoplastic tissue.
- Chronic infections and delayed wound healing – common examples include tooth root infections, sinusitis, and sole abscesses. Wounds and lacerations may take longer to heal. These problems are attributed to immunosuppression secondary to hyperadrenocorticism, but tissues may also be weakened by protein depletion.

- Lethargy – this has been attributed to increased beta endorphin release from the pars intermedia. Horses with advanced PPID also appear to be more tolerant of pain.
- Hyperhidrosis – this may simply result from hirsutism, but some horses that are clipped continue to sweat excessively, so a disturbance in hypothalamic body temperature regulation is suspected.
- Other clinical signs include central nervous system deficits such as seizures, which should only be attributed to compression by a pituitary adenoma in advanced cases, and persistent lactation. The latter problem may result from increased prolactin production.

Diagnosis

1. Detection of hirsutism in an older horse is sufficient for diagnosis, particularly when accompanied by other clinical signs.
2. Complete blood count (CBC) – hyperadrenocorticism is associated with mature neutrophilia, lymphopenia, and monocytosis. This is the stress response recognized in many patients when they first present for evaluation, but in the case of PPID, these alterations persist after stress has subsided.
3. Resting hormone measurements – commercial laboratories currently measure plasma ACTH levels and may soon offer α-MSH measurements. Care should be taken to collect blood samples under the conditions recommended by the laboratory. Blood should be collected in the morning after the horse has remained calm overnight. Sampling horses under stressful or painful conditions (i.e. during acute laminitis) is likely to lead to spurious results. Blood should be collected in a plastic tube containing ethylenediamine tetraacetic acid (EDTA) and immediately cooled in ice. Samples should be refrigerated until centrifuged, and centrifugation should be performed as soon as possible after blood collection. Harvested plasma should be shipped overnight with ice packs or frozen. Pituitary pars intermedia dysfunction is confirmed when the resting plasma ACTH concentrations exceed 35 pg/mL (7.7 pmol/L). It is important to use a seasonally adjusted reference range in August, September, and October because ACTH concentrations increase during these months of the year. If the laboratory provides a seasonally adjusted reference range, testing in the late summer and autumn is encouraged to diagnose early disease and assess treated horses during this period of natural stimulation.
4. Resting glucose and insulin concentrations – horses with PPID can be normoglycaemic or hyperglycaemic (fasting glucose >110 mg/dL or >6.1 mmol/L), and glucosuria may be present if blood glucose levels exceed 180 mg/dL (10.0 mmol/L). Serum insulin concentrations are elevated if compensated IR is present. Insulin resistance is detected in some patients with PPID and this is likely to be determined by whether the animal was insulin resistant prior to developing PPID. Cortisol inhibits the action of insulin at the tissue level and CLIP stimulates release of insulin from the pancreas. Pituitary pars intermedia dysfunction may therefore exacerbate hyperinsulinaemia and IR.
5. Diurnal cortisol rhythm test – blood cortisol concentrations vary markedly within minutes, so single cortisol measurements do not aid in the diagnosis of PPID. However, plasma or serum cortisol concentrations generally decrease throughout the day, so morning and evening concentrations can be compared. A healthy horse usually has an evening blood cortisol concentration that is >30% lower than the morning value. Unfortunately, healthy horses often have false-positive results because blood cortisol concentrations are fluctuating throughout the day and false-positive test results occur more frequently in older horses, so this test not recommended.
6. Dynamic testing for PPID – none of the available tests have sensitivity or specificity values of 100%, so it is important to use clinical judgement when interpreting results.
   - Overnight dexamethasone suppression test (**DST**) – this test is performed by collecting a pre-injection blood sample, injecting dexamethasone intravenously (or intramuscularly) at a dosage of 40 µg/kg body weight (20 mg for a 500-kg horse), and collecting a second blood sample 19 or 24 hours post-injection. In the healthy horse, dexamethasone acts via negative feedback to suppress CRH and ACTH production, which lowers plasma cortisol concentrations for more than 24 hours. Detection of a plasma cortisol concentration below 10 ng/mL (equivalent to 1.0 µg/dL or 27 nmol/L) after 24 hours is the response expected in a healthy horse, so a value >10 ng/mL is a positive result for PPID. In the affected horse, dexamethasone transiently suppresses ACTH release from the pituitary gland, but the pars intermedia remains unaffected. Continued ACTH secretion by this region of the pituitary gland causes blood cortisol concentrations to rise above 10 ng/mL by 24 hours post-injection. As with ACTH measurements, more false-positive dexamethasone suppression test results occur in the autumn, so horses should be tested in the spring or early summer. It has been suggested that horses with equine metabolic syndrome are more likely to develop laminitis following the DST, so this should be discussed with the client prior to testing.
   - Thyrotropin-releasing hormone (TRH) stimulation test – this test is recommended when early disease is suspected and other tests yield normal results. In the author's opinion the TRH stimulation test is the most sensitive diagnostic test for PPID in horses. A pre-injection blood sample is collected, followed by injection of TRH (1.0 mg total) intravenously. After 30 minutes, a second blood sample is collected and both samples are submitted for measurement of ACTH concentrations. Detection of a plasma ACTH concentration > 35 pg/mL at 0 or 30 minutes supports the diagnosis of PPID. Horses with PPID show an increase in plasma ACTH concentration following TRH administration because cells within the abnormal pars intermedia possess more receptors for this hormone and secrete ACTH when stimulated. The major limitation of this test is the cost and availability of medical grade TRH. Horses occasionally yawn or show mild muscle fasciculations after injection of TRH, but these signs are transient.
   - ACTH stimulation test – this test should be selected if an adrenal tumour is suspected, but results can be difficult to interpret because horses with PPID sometimes develop adrenal hyperplasia. Adrenal tumours should be suspected when clinical signs of advanced hyperadrenocorticism are present in the absence of hirsutism.
   - Oral domperidone test – domperidone is a D2 dopamine receptor antagonist. Transient blocking of dopamine action causes an exaggerated release of ACTH from the pars intermedia because this region is synthesizing more hormone than normal in the PPID patient.

Treatment

There are two important components of PPID management – drug therapy and laminitis prevention.

1. Three drugs are used to treat PPID in horses:
   - Pergolide mesylate – this is a dopaminergic agonist which compensates for the loss of dopaminergic neurons and

may slow the progression of PPID. Dopamine suppresses ACTH production by the pars intermedia, which is why clinical signs of PPID improve with treatment. This drug is widely used and is recognized to be the most effective treatment available for PPID in horses. A starting dosage of 1 mg pergolide total dose (or 0.002 mg/kg body weight) once daily orally is recommended. Some practitioners recommend twice daily administration because of perceived improvement in efficacy. Responses to therapy include increased activity, decreased recurrence of laminitis, improved insulin sensitivity, gain in muscle mass, return to normal shedding pattern, and improvement with respect to polyuria/polydipsia. Plasma ACTH measurements or dexamethasone suppression testing can be repeated to assess the response to therapy. The dose of pergolide can be increased in 0.5 mg/day increments for 30-day periods until clinical signs improve or a maximum dosage of 3 mg/day is attained.

- Trilostane – this drug is used in Europe and has recently become available in the United States. Trilostane acts at the adrenal cortex by inhibiting the enzyme 3-β-hydroxysteroid dehydrogenase, which is involved in cortisol production. This drug is available in 30, 60 or 120 mg capsules (Vetoryl®, Arnolds Veterinary Products Ltd, UK) and is given orally in the evening at a dosage of 1.0 mg/kg body weight once daily.
- Cyproheptadine – this drug inhibits the action of the excitatory neurotransmitter serotonin and was widely used before pergolide was prescribed. Some horses or ponies respond well to the drug and it is occasionally used in combination with pergolide.

2. Laminitis is a major concern for horses with PPID, so every effort should be made to lower the risk of disease. If the patient suffers from IR as well as PPID, diet and exercise recommendations should be made to improve insulin sensitivity (refer to the section on equine metabolic syndrome).

9.3 HYPOADRENOCORTICISM (ADRENAL INSUFFICIENCY, TURN-OUT SYNDROME, STEROID LET DOWN SYNDROME)

This condition sometimes develops when corticosteroid therapy is discontinued abruptly or after prolonged stress. Adrenal insufficiency has also been reported in one neonatal foal that presented with watery diarrhoea and developed seizures as a result of hyponatraemia, hypochloraemia, and hyperkalaemia. A low sodium-to-potassium ratio was detected (reference range <27) and the foal responded to exogenous corticosteroid therapy. In adult horses, adrenal insufficiency is most commonly associated with improper drug withdrawal strategies after administration of dexamethasone at immunosuppressive dosages of 0.1 to 0.2 mg/kg body weight for the treatment of purpura haemorrhagica, immune-mediated haemolytic anaemia, or infiltrative bowel disease. Corticosteroids are also administered to racehorses to treat musculoskeletal pain or as a stimulant. Intensive training, racing, showing, or travel are also thought to cause chronic stress and adrenal insufficiency in some cases.

Aetiology and pathogenesis

Prolonged corticosteroid administration causes down-regulation of ACTH synthesis within the pituitary gland because exogenous hormone exerts negative feedback upon the hypothalamic-pituitary-adrenal axis. Atrophy of the zona fasciculata within the adrenal cortex occurs over time, but the zona glomerulosa is minimally affected, which allows continued secretion of mineralocorticoids. It takes days to weeks for the axis to recover and the time required for recovery depends upon the steroid used, dosage, and duration of treatment. Dexamethasone is a corticosteroid routinely prescribed to induce immunosuppression, so adrenal insufficiency is a concern after use of this drug. Chronically stressed horses may develop adrenal insufficiency after ACTH synthesis is down-regulated as a result of repeated stimulation. With the exception of adrenal insufficiency in neonatal foals, mineralocorticoid deficiency is very rare. Addison's disease (primary hypoadrenocorticism) caused by autoimmune adrenalitis and accompanied by hyponatraemia and hyperkalaemia, has not been reported in the horse.

Clinical signs

- Depression and lethargy.
- Anorexia and weight loss.
- Exercise intolerance.
- Poor hair coat.

Diagnosis

- History and clinical signs.
- Inadequate response to synthetic ACTH (Cosyntropin; 250 µg vial). A pre-injection blood sample is collected for the measurement of cortisol and 0.1 µg/kg synthetic ACTH (50 µg total dose for a 500-kg horse) is injected intravenously. Blood samples are collected at 30 and 60 minutes post-injection and a greater than 1.5-fold increase in plasma cortisol concentration is expected relative to baseline at 30 or 60 minutes in the healthy horse or pony.

Treatment

- If caused by chronic stress, horses should be rested and ideally turned out on pasture for several months.
- Dexamethasone can be administered every other day at a low dosage (1 or 2 mg/day), but prednisolone therapy is preferable. Administer 100 to 300 mg prednisolone daily or every other day for 2 to 3 months and slowly taper the dosage over 4 weeks when the drug is withdrawn.

9.4 PHAEOCHROMOCYTOMA

This tumour develops from the chromaffin cells of the adrenal medulla and non-functional phaeochromocytomas are sometimes identified upon post mortem examination. Functional tumours are very rare in horses and affected horses present with clinical signs of colic and suffer from anxiety, tachycardia, tachypnoea, profuse sweating, muscle tremors, and mydriasis.

Clinical and clinicopathological signs

This condition should be a primary differential diagnosis for a horse that presents with profuse sweating and tachycardia, without evidence of intestinal disease. Detection of bilateral mydriasis supports the diagnosis and clinical pathology findings include an elevated hematocrit (splenic contraction and haemoconcentration), stress leukogram (mature neutrophilia with lymphopenia), hyperglycaemia, and azotaemia. Haemoabdomen has also been associated with phaeochromocytomas. Haemorrhage from the tumour results in haemoabdomen or accumulation of blood within the retroperitoneal space, which can be felt as a mass located on the dorsal aspect of the abdomen upon rectal palpation.

Diagnosis

Measurement of catecholamine concentrations within the blood or urine can be attempted by contacting a human hospital laboratory, but these hormones are labile and samples must be transported quickly. Samples should also be collected from an unaffected healthy horse to provide control values. Direct or indirect measurement of arterial blood pressure and documentation of hypertension provides further support for the diagnosis.

Treatment

Surgical resection of the affected tissue has not been attempted in the horse and the development of arrhythmias during anaesthesia increases the risk of the procedure. There are no medical treatments described for condition in horses.

9.5 INSULIN RESISTANCE (IR)/EQUINE METABOLIC SYNDROME (EMS)

Insulin resistance (**IR**) can be broadly defined as a decrease in tissue responses to circulating insulin, which impairs insulin-mediated glucose uptake into skeletal muscle, adipose, and liver tissues. Causes of IR include a reduction in the density of insulin receptors on cell surfaces, malfunction of insulin receptors, defective internal signalling pathways, or interference with the translocation or function of glucose transporter proteins. Compensated IR is the most common glucose and insulin metabolism abnormality identified in horses and ponies. In this situation, serum insulin concentrations are higher than normal because more insulin is secreted from the pancreas to compensate for lower tissue responses.

Compensated IR is thought to lead to uncompensated IR if pancreatic insufficiency develops. This is also referred to as beta cell exhaustion and it is recognized by detection of a serum insulin concentration that is lower than expected for the measured blood glucose concentration. Type 2 diabetes mellitus occurs when insulin secretion can no longer compensate for the IR state and hyperglycaemia develops. Diabetes actually refers to excessive production of urine (polyuria). In the case of type 2 diabetes mellitus, polyuria develops as a result of osmotic diuresis secondary to glucosuria. Diabetes mellitus is very rare in the horse and is most likely to be encountered when managing a horse with advanced PPID.

Aetiology

Equine metabolic syndrome (EMS) includes (1) increased adiposity, (2) IR, and (3) hyperinsulinemia, and predisposes affected animals to insidious-onset or pasture-associated laminitis. Clinical signs of EMS have previously been attributed to hypothyroidism, but it is now known that low serum thyroid hormone concentrations are a consequence rather than a cause of obesity and IR in horses.

Clinical signs and proposed pathogenesis

Equine metabolic syndrome occurs most commonly in ponies, Miniature horses, donkeys, and horses of the Morgan horse, Paso Fino, and Norwegian Fjord breeds. It has also been detected in Arabians, Quarter Horses, Mustangs, Saddlebreds, Tennessee Walking Horses, Thoroughbreds, and Warmbloods. This syndrome develops in adult ponies and horses, with the onset of laminitis depending upon interactions between genetic and environmental factors such as overfeeding. Founder lines are sometimes present on hoof surfaces, indicating previous episodes of subclinical laminitis. These protruding growth rings are wider at the heel than they are dorsally, and are thought to occur when laminitis inhibits dorsal hoof wall growth. Physical characteristics of EMS include generalized obesity and/or regional adiposity. Regional adiposity takes the form of cresty neck in horses and neck circumference has been negatively correlated with insulin sensitivity. Other manifestations of regional adiposity include abnormal adipose tissue deposits close to the tailhead, within the prepuce, or randomly distributed beneath the skin of the trunk region (Figure 9.2).

Grass consumed on pasture probably provides too many calories for the genetically susceptible horse. These horses and ponies are evolutionarily adapted to poorer forages, so they rapidly gain weight when grazed on lush pasture. Pasture grass contains hydrolysable carbohydrates such as sugars that are digested and absorbed in the small intestine and fermentable carbohydrates that are primarily digested within the large intestine. Episodes of laminitis usually occur when affected horses or ponies are grazing on pasture. Non-structural carbohydrates (NSC) within pasture grass play an important role in this process. This group of carbohydrates include simple sugars, starch, and fructans (polymers of fructose), and levels of these components vary considerably within grass according to geographical location, soil type, weather conditions, and time of day.

Relationships between obesity and IR are complex because obese horses and ponies are more likely to be insulin resistant, but not every overweight horse is insulin resistant. Insulin resistance can also occur without obesity. It is likely that the accumulation of body fat predisposes the animal to IR, but obesity may have to be present for months or years before IR develops. Laminitis and IR are potentially related through three mechanisms: (1) lamellar damage induced by hyperinsulinemia or hyperglycemia, (2) endothelial cell dysfunction within the hoof vessels, and (3) pro-inflammatory or pro-oxidative states associated with chronic IR and/or obesity.

Diagnosis

1. Resting serum insulin concentrations – this is a useful screening test because compensatory hyperinsulinaemia is a common feature of IR in horses. Pancreatic insulin secretion increases to compensate for the decrease in tissue effectiveness, so resting serum insulin concentrations are elevated in horses with moderate or severe IR. However, this test is less likely to identify animals with mild/early IR or rarer cases of diabetes mellitus when pancreatic insufficiency is occurring. It is ideal for horses and ponies to be screened for IR once yearly in the spring as part of annual wellness examinations. Blood samples should be collected after a short fasting period and an insulin concentration ≥20 μU/mL (mU/L) supports the diagnosis of IR. Fasting conditions can be created by leaving only one flake of hay in the horse's stall after 10 PM the night before and collecting blood in the morning. Some insulin-resistant horses and ponies have fasting insulin concentrations <20 μU/mL, so these animals represent a diagnostic challenge. There are two approaches to diagnosis in these cases: (1) measurement of serum insulin concentrations on multiple occasions or after feeding grass hay or (2) glucose tolerance testing. Differences in hay composition make the interpretation of serum insulin concentrations in the fed state more challenging, so a universal cut-off value has not been established. However, blood samples can be drawn from the patient and a matched control animal that is fed the same hay, and results compared. As an approximate guideline, most healthy horses have resting serum insulin concentrations <30 μU/mL when consuming grass hay. There are several glucose tolerance tests that can be selected, including the oral glucose tolerance test, intravenous glucose tolerance test, and combined glucose-insulin test (**CGIT**). The CGIT is preferred because insulin sensitivity is assessed by administering exogenous insulin.

Figure 9.2 Mixed-breed mare with equine metabolic syndrome (EMS); note the enlarged neck crest and overall obese body condition.

2. Combined glucose-insulin test – this dynamic test should be considered when the fasting serum insulin concentration is <20 μU/mL, yet IR is suspected. Horses should be brought in from pasture the night before testing and only one flake of grass hay should be available after 10 PM, in order to perform the test under short-term fasting conditions. An intravenous catheter should ideally be placed the night before to minimize stress, which induces transient IR and will lead to a false positive result. A pre-infusion (baseline) blood sample is collected and then 150 mg/kg body weight (bwt) 50% dextrose solution (150 mL for a 500-kg horse) is quickly infused through the catheter, immediately followed by 0.10 units/kg bwt regular insulin (100 units/mL; 0.50 mL for a 500-kg horse). Short-acting recombinant human insulin or soluble porcine insulin should be used for this test. Blood samples are collected at 5, 15, 25, 35, 45, 60, 75, 90, 105, 120, 135 and 150 minutes post-infusion, but the test can be halted once the blood glucose level falls below baseline. Blood glucose concentrations are measured with a hand-held glucometer. Insulin concentrations should be measured prior to injection and at the 45 minutes post-injection time point. Both glucose and insulin responses to the CGIT are examined to interpret results. Insulin resistance is diagnosed when the blood glucose concentration remains above baseline for ≥45 minutes and/or the insulin concentration exceeds 100 μU/mL at 45 minutes. There is a small risk of hypoglycaemia developing in horses with higher insulin sensitivity, so two 60-mL syringes containing 50% dextrose should be kept on hand and administered if sweating, muscle fasciculations, or weakness are observed, or if the blood glucose concentration drops below 40 mg/dL (approximately 2.0 mmol/L).

Management

1. Management of obesity – weight loss should be induced in obese horses by limiting caloric intake, increasing exercise, and eliminating access to pasture until the ideal body condition has been attained. Concentrates must be eliminated from the diet and hay should be fed in amounts equivalent to 1.5% of body weight (i.e. 15 lb hay for a 1000-lb horse; 7.5 kg for a 500-kg horse). It is often necessary to further reduce the amount of hay fed to obese horses and ponies because of their enhanced metabolic efficiency. Amounts can be lowered to 1% of body weight in hay per day, which is considered to be the minimum forage requirement for equids. This is initially calculated on current body weight, but is lowered to 1% of target body weight after 30 days. For example, an obese horse may weigh 600 kg initially, but have a target body weight of 500 kg. Hay with low (<10%) non-structural carbohydrate (NSC) content should be selected and horse owners should be asked to weigh their hay to ensure that correct amounts are fed. If the NSC content exceeds 10%, soaking it in cold water for 60 minutes will lower the sugar and fructan content prior to feeding. However, this

approach does not address the problem of higher sugar concentrations in all cases, so it should not be used as a substitute for forage analysis and the selection of appropriate hay. Complete feeds and bagged forages are also available for insulin resistant horses. Horses should also receive a multivitamin/mineral supplement, with 1000 IU vitamin E provided per day when access to green grass is restricted. Patients with laminitis should not be exercised until hoof structures have stabilized, but unaffected horses should be exercised regularly. At minimum, horses should be walked on a lead rope, exercised on a lunge line, or ridden at a walk. Ideally, the exercise programme should be adjusted over time until it includes >30 minutes trotting or more strenuous exercise every day or every other day.

2. Management of insulin resistance in the obese horse – insulin resistance can be managed by controlling obesity, instituting an exercise regimen, and avoiding feeds that exacerbate the condition. Pasture grass is the most difficult component of the diet to control, so access to pasture must be restricted or eliminated. Construction of a 'starvation paddock' is recommended in order to allow affected horses and ponies to be turned out in a smaller area. Many horses will need to be held off pasture for only a few weeks or months until the IR has been controlled, but there are some animals that are so sensitive to changes in pasture grass carbohydrate content that they must be permanently taken off pasture. Many horses and ponies with EMS can be effectively managed by limiting grazing time to 1 to 2 hours per day, housing in a small grass paddock, strip grazing using an electric fence, or using a grazing muzzle. Feeds that are lower in starches and sugar should be selected, and treats such as sugar cubes must be avoided altogether. Basic guidelines for lowering the risk of pasture-associated laminitis include restricted turnout when the pasture grass is in a dynamic phase. These phases include new growth in the spring or after heavy rain, drought conditions when grasses are stressed, and at the onset of winter when frosts occur at night.
3. Management of insulin resistance in the leaner horse – some insulin-resistant horses are leaner, but still exhibit regional adiposity. In these cases, additional calories must be provided without exacerbating IR. Feeds that are low in starch and sugar are appropriate in these situations. It is also advisable to feed hay before concentrates and to feed smaller meals more frequently.

Treatment

Medical treatments for insulin resistance should not be used as substitutes for diet and exercise strategies, but there are two indications for treatment: (1) medical management of IR during the time it will take for diet and exercise approaches to take effect (3 to 6 months) and (2) for patients that do not respond to changes in diet and exercise. Metformin and levothyroxine sodium are currently used for the medical management of IR in horses and ponies.

- Metformin – this member of the biguanide family of antidiabetic drugs is commonly used in human medicine. Blood glucose concentrations are lowered by metformin and tissue insulin sensitivity increases in response to treatment in humans. This drug has been administered to insulin-resistant horses and ponies at a dosage of 15 mg/kg orally every 12 hours, but a recent pharmacokinetic study has raised questions regarding the oral bioavailability of this drug in equids. A dosage of 30 mg/kg orally every 8 to 12 hours may therefore be more effective. Potential side effects include hypoglycaemia when treatment is initiated and mild colic, which can be addressed by discontinuing metformin for 24 hours and then initiating treatment again the next day using a lower dose that is increased over several days.
- Levothyroxine sodium – this drug induces weight loss and improves insulin sensitivity and can be prescribed when obese insulin resistant horses are threatened by laminitis. This drug should not be used as substitute for diet and exercise changes, but is useful when affected horses cannot be exercised. Levothyroxine sodium can be administered by mouth or in the feed at a dosage of 48 mg/day (4 teaspoons per day; approximately 0.1 mg/kg) for 3 to 6 months. At this dosage, levothyroxine is likely to act as a metabolic stimulant and therefore induces weight loss. Serum tT4 concentrations range between 40 and 100 ng/mL in treated horses, but clinical signs of hyperthyroidism such as sweating or tachycardia have not been observed. When levothyroxine treatment is discontinued, the dosage should be lowered to 24 mg (2 tsp)/day for 2 weeks and then 1 tsp (12 mg)/day for 2 weeks. The benefits of treating horses with levothyroxine at lower dosages for extended periods of time have not been scientifically evaluated. Levothyroxine is commonly used in the United States, but is more expensive in Europe.
- Equine Cushing's disease (also called pituitary pars intermedia dysfunction) can develop in horses and ponies with equine metabolic syndrome as they reach middle age (10 to 20 years). This condition can be diagnosed by recognizing clinical signs such as delayed shedding of the winter haircoat, hirsutism, polyuria/polydipsia, and skeletal muscle atrophy, or by performing diagnostic tests. If diagnostic tests are consistent with ECD or the condition is suspected on the basis of clinical judgment, then treatment with pergolide should be initiated. The starting dose for this drug is 1 mg (total)/day orally. Equine Cushing's disease may exacerbate IR in equids, so early recognition of this condition is important.

9.6 HYPERLIPAEMIA

Obesity and insulin resistance are important predisposing factors for this condition and it is triggered by negative energy balance. The term hyperlipidaemia refers to higher than normal blood concentrations of lipids, whereas equine hyperlipaemia is a metabolic disorder characterized by markedly elevated serum triglyceride (TG) concentrations (>500 mg/dL), depression, anorexia, and organ dysfunction.

Equine hyperlipaemia should be considered as a potential secondary complication of negative energy balance in any Miniature horse, pony, or donkey because these animals are at greatest risk. However, horses with EMS or PPID, and pregnant mares are also at greater risk for hyperlipaemia when deprived of feed.

Congenital hyperlipaemia can occur in neonatal foals born to mares that suffer from equine hyperlipaemia. Blood lipid concentrations return to normal in the foal over several days and affected mares improve after parturition. Note that lipaemia is sometimes detected in healthy foals after nursing because chylomicrons are being produced by the intestine after digestion of milk fats.

A mortality rate of 75% has been reported in the past, but patients can be successfully treated if the condition is recognized soon enough. Blood TG concentrations should be monitored in any high-risk animal that is not receiving sufficient calories for their needs.

Aetiology

- Inherent differences in metabolism make Miniature horses, ponies, and donkeys more susceptible to equine hyperlipaemia.
- Obesity and IR significantly increase the risk of the condition developing.

- Equine metabolic syndrome and PPID increase the susceptibility to equine hyperlipaemia.
- Primary problems that induce negative energy balance include starvation, oral trauma or dental problems, oesophageal obstruction, and colic.
- Stress, severe parasitism, pregnancy, and lactation can also exacerbate negative energy balance.
- Potential triggers of this condition include negative energy balance, systemic shock, and endotoxaemia.

Pathophysiology

Negative energy balance or stress stimulates lipolysis within adipose tissues and causes hydrolysis of stored triglyceride (TG), which liberates fatty acids and glycerol. Fatty acids enter the blood and circulate as free fatty acids (FFAs; also called non-esterified fatty acids). Lipolysis is mediated by hormone-sensitive lipase (HSL). This enzyme is stimulated by hormones such as epinephrine, cortisol, and glucagon, and inhibited by insulin. Equine hyperlipaemia is associated with obesity and IR for two major reasons: 1) obese animals have more TG stored within their adipose tissues, and 2) the normal dampening effect of insulin on HSL activity is diminished in the insulin resistant animal. These two factors combine to cause a greatly exaggerated response to negative energy balance that results in massive mobilization of fatty acids from adipose tissues. Circulating FFAs are removed from the blood by the liver, and used as a substrate for energy production (via beta oxidation) or re-esterified into TG. Newly synthesized TG can be stored within hepatocytes or exported from the liver in the form of very-low density lipoprotein (**VLDL**). Packaging of TG within VLDL allows this hydrophobic lipid to be transported within the aqueous environment of the blood.

Equids are very efficient at producing VLDL within the liver and can produce two forms of this lipoprotein. This contrasts with the cow, which cannot up-regulate TG export from the liver and synthesizes only one form of VLDL. Horses therefore accumulate lipid within the circulation (hyperlipidaemia), whereas cows develop hepatic lipidosis (fatty liver). Hepatic lipidosis can sometimes occur towards the end of the disease course in horses, but it is a secondary condition that develops after circulating TG levels become so high that lipid begins to accumulate in all tissues. Under healthy conditions, TG that is circulating within VLDL is utilized by peripheral tissues. As these lipoproteins circulate through capillaries, lipoprotein lipase that is present on the endothelial surfaces catalyses the hydrolysis of TG into fatty acids and glycerol. Fatty acids enter the tissues and are used for energy production or stored. In the case of equine hyperlipaemia, the disorder is driven by excessive mobilization of fatty acids from adipose tissues, which overwhelms downstream processes. Accumulation of triglyceride-rich VLDL within the circulation suppresses appetite which perpetuates a vicious cycle. Liver, kidney, and brain damage occur as lipid accumulates within tissues.

Clinical signs

- Initial clinical signs are nonspecific and include anorexia, depression, and lethargy. These signs are often attributed to the primary disease, so it is important to recognize the high-risk animal by its appearance and begin monitoring of serum or plasma TG concentrations.
- Steatorrhoea (fat in the faeces) or diarrhoea.
- Clinical signs of hepatic encephalopathy with advanced disease.
- Opaque appearance of plasma or serum when samples are drawn for routine blood tests.

Diagnosis

- Recognition of the at-risk animal.
- Detection of hepatic encephalopathy.

Figure 9.3 Markedly lipaemic serum.

- Hyperlipidaemia (specifically hypertriglyceridaemia) is the clinicopathological abnormality associated with this disorder. A plasma or serum TG concentration above 65 mg/dL (1.13 mmol/L; multiply by 0.0113 to convert mg/dL to mmol/L) defines hypertriglyceridaemia, and this gives an indication of negative energy balance. However, concentrations >500 mg/dL (5.65 mmol/L) are usually detected in patients suffering from equine hyperlipaemia. Detection of elevated blood TG concentrations should prompt correction of negative energy balance.
- A newsprint test can be performed in the field. A tube of blood is collected and allowed to stand vertically for 10 to 30 minutes. As the plasma or serum separates, it is opaque and newsprint cannot be read through the tube (Figure 9.3). This degree of opacity indicates a plasma TG concentration above 500 mg/dL.
- Clinicopathological evidence of hepatic damage may be seen with advanced disease as hepatic lipidosis develops. Elevated blood gamma glutamyl transferease (GGT), sorbitol dehydrogenase (SDH), alkaline phosphatase (ALP), and aspartate aminotransferase (AST) activities are detected. Loss of hepatic function manifests as elevated plasma total bilirubin, ammonia, and bile acid concentrations and a lower blood urea nitrogen concentration. Note that lipaemia interferes with many of the assays performed by chemistry analysers.
- Azotaemia is sometimes detected and serves as an indication of renal compromise. It has been suggested that azotaemia plays a role in the development of hyperlipaemia, but a cause-and-effect relationship has not been established.

Treatment

- Correction of the primary disease process. This may even include abortion of the mare if placentitis or hydrops allantois are present.
- Increase feed intake – mildly affected patients improve when their feed intake increases. A variety of feedstuffs or treats are offered until a preferred diet is identified. Once a preferred feed has been identified, provide fresh samples frequently to maintain interest. In depressed patients, hand feeding may be necessary.
- Enteral feeding – commercial enteral feeds can be used for smaller patients. If a larger-diameter nasogastric tube can be passed, liquid preparations of alfalfa meal or soaked, pelleted feedstuffs with added dextrose can be carefully pumped or siphoned into the stomach. Administer small quantities frequently (as often as q4h). However, colic, enterocolitis, or laminitis may be induced, so intravenous nutritional support is the preferred treatment option.

- Intravenous fluids and/or parenteral nutrition – administer 5 to 10% dextrose within polyionic fluids as a continuous infusion at an initial maintenance rate of 60 mL/kg body weight per day. Ideally, blood glucose measurements and urine dipstick testing should be performed to determine an administration rate that minimizes renal overflow of glucose. Partial parenteral nutrition (PPN) can be provided using a solution composed of 50% dextrose and amino acids.
- Regular insulin can be administered as a continuous rate infusion (CRI) if blood glucose concentrations exceed 200 mg/dL or glucose is detected in the urine. Add 1.0 mL (100 units) regular insulin (100 units/mL) to 1 L saline (final concentration 0.1 units/mL) and begin the CRI at rate of 2 mL/min (0.2 units/min). Monitor blood glucose concentrations. Clinical signs of hypoglycaemia include sweating, muscle fasciculations, and weakness.
- Heparin sulphate – this treatment is given with the aim of potentiating LPL activity. However, it is likely that HSL activity is already maximized, so heparin is unlikely to be effective. A dosage of 10–20 IU/kg via subcutaneous or intravenous injection three times daily can be used. Note that heparin lowers haematocrit levels and can inhibit haemostasis, which may be relevant if surgery must be considered.
- Consider medications administered in cases of liver failure and hepatic encephalopathy, including lactulose.

9.7 HYPOTHYROIDISM

There has been confusion about hypothyroidism in horses because this diagnosis is often made on the basis of low resting serum total triiodothyronine (**tT3**) and total thyroxine (**tT4**) concentrations alone. As our understanding of thyroid dysfunction in horses has improved, it has become clear that many nonthyroidal conditions affect resting hormone concentrations. Resting tT3 and tT4 concentrations are sometimes lower than normal in horses that suffer from EMS and PPID. As a consequence, clinical signs associated with these disorders including obesity, regional adiposity, and laminitis have been incorrectly attributed to hypothyroidism. Further confusion has been generated by the effect of phenylbutazone on these resting hormone concentrations. This drug is commonly used for the treatment of laminitis and lowers serum tT4 concentrations because it is protein-bound and displaces free hormone from blood proteins.

Whether low resting thyroid hormone concentrations should be addressed with supplementation remains controversial. It is likely that non-thyroidal conditions lower thyroid hormone concentrations by suppressing thyrotropin-releasing hormone (**TRH**) secretion by the hypothalamus or thyroid-stimulating hormone (**TSH**; also called thyrotropin) release from the pituitary gland. This represents a beneficial adjustment in metabolism at times when disease is present. Adult horses with low resting serum thyroid hormone concentrations are not likely to suffer from primary hypothyroidism because they respond normally to hormone challenge tests. Unfortunately, the detection of hypothyroidism in horses is hindered by the lack of a commercially available serum TSH assay. In human medicine, thyroid gland dysfunction (primary hypothyroidism) is diagnosed by detecting elevated serum TSH concentrations. Secondary hypothyroidism resulting from non-thyroidal conditions would be expected to lower serum TSH concentrations.

Other medical problems attributed to hypothyroidism in the past include exertional rhabdomyolysis, poor racing performance, infertility in mares and stallions, and anhidrosis.

Detection of a positive response to levothyroxine supplementation has been taken as evidence of hypothyroidism in the past, but results of recent studies have shown that this drug exerts pharmacological effects upon body weight and insulin sensitivity, which may explain why obese insulin resistant horses respond positively to treatment.

Congenital hypothyroidism occurs in horses. Foals born prematurely sometimes suffer from hypothyroidism, which is detected by measuring serum thyroid hormone concentrations and performing thyroid functions tests. Other foals suffer from goitre caused by iodine deficiency or excess during gestation.

Finally, congenital hypothyroidism and dysmaturity syndrome occurs in foals primarily located in the Pacific Northwest region of the United States and western Canada. These foals may not develop goitre, but are born dysmature after a prolonged gestation. They are weak and suffer from mandibular prognathism, incomplete ossification of their cuboidal bones, angular limb deformities, and ruptured tendons. This syndrome is thought to result from a dietary iodine imbalance or subclinical nitrate toxicity in the mare.

Aetiology

Many cases of congenital hypothyroidism can be attributed to inadequate or excessive iodine in the feed of pregnant mares, but in other foals, the cause remains unknown. Mares grazing on pasture may ingest other goitrogenic compounds during pregnancy, so attention should be paid to conditions on the farm. Iodine requirements are reported to be 1 mg per day for an adult horse, but most horses receive at least 2 mg/day. This mineral is included in trace mineral blocks. Tolerance levels for iodine are 5 parts per million (ppm) in the horse and congenital goitre has been induced in foals by supplementing mares with 35 mg iodine/day. This is approximately the amount of iodine consumed by a cow each day, so there are differences between species, and cattle feeds should not be fed to horses. Feeds or supplements containing kelp or other seaweeds are a likely source of excess iodine.

Primary hypothyroidism refers to dysfunction of the thyroid gland itself, which is commonly attributed to autoimmune thyroiditis in dogs and humans. However, naturally occurring primary hypothyroidism is poorly documented in the horse and this diagnosis should be made only if the horse fails to respond to hormone stimulation tests. Hypothyroidism has been experimentally induced by administering propylthiouracil to horses or surgically removing the thyroid gland. Interestingly, thyroidectomized horses develop a poorer hair coat and seborrhoea, but otherwise remain normal in appearance. Lower rectal temperatures and heart rates are detected, and horses develop lower limb oedema when left standing in the stall, but marked hypothyroidism is remarkably well tolerated. Thyroidectomized horses do not become obese or develop laminitis, and physical activity is only slightly reduced.

Secondary hypothyroidism and tertiary hypothyroidism refer to decreased secretion of TSH or TRH from the pituitary gland or hypothalamus, respectively. It is likely that suppressed TSH secretion accounts for the lower resting thyroid hormone concentrations detected in horses with nonthyroidal illnesses including EMS and PPID, and systemic conditions such as colic, colitis, and respiratory disease. Unfortunately, the lack of a commercially available TSH assay for horses hinders diagnosis of this condition.

Sick euthyroid syndrome is described in dogs and humans, but has not been documented in the horse. One important component of this syndrome is the increased peripheral conversion of thyroxine (T4) into inactive reverse triiodothyronine (rT3) instead of active triiodothyronine (T3). This is thought to be a protective function to suppress metabolic processes within tissues and preserve calories at times when disease is present. In this situation, there is more conversion of

T4 into rT3 by 5-monodeiodinase, whereas conversion of T4 into T3 by 5′-monodeiodinase is suppressed.

Clinical signs

- Naturally occurring hypothyroidism in adult horses – this condition is poorly documented. Thyroidectomized horses have lower rectal temperatures and resting heart rates, and a dull hair coat, seborrhea, and lower limb oedema.
- Congenital hypothyroidism in foals – goitre, prematurity (gestational length <320 days) or dysmaturity, hypothermia, weakness, and slowed growth. In the case of congenital hypothyroidism and dysmaturity syndrome, musculoskeletal abnormalities would be expected, including incomplete ossification of the cuboidal bones, angular limb deformities, prognathism, and tendon contracture.

Diagnosis

- Clinical signs.
- Resting tT3 and tT4 concentrations can be measured, but results must be interpreted with caution. Thyroid hormone concentrations are likely to vary according to season, time of day, and metabolic status of the horse. It is therefore recommended that concentrations within reference range be interpreted as an indication of euthyroidism (healthy state), but low concentrations should only prompt further evaluation of the patient, not a diagnosis of hypothyroidism.
- Low resting tT3 or tT4 concentrations should be interpreted within the context of the signalment and history. Horses currently suffering from other medical conditions, including infectious diseases, musculoskeletal injuries, or starvation, should be re-evaluated after these primary nonthyroidal conditions have resolved.
- Horses receiving drugs, particularly those known to bind to blood proteins such as phenylbutazone, should be re-evaluated after these therapies have been discontinued. Patients with evidence of EMS or PPID should be evaluated for these disorders before thyroid dysfunction is considered further. Both the chronic IR of EMS and hyperadrenocorticism of PPID are likely to be non-thyroidal conditions that lower resting thyroid hormone concentrations.
- If all non-thyroidal causes can be eliminated, a hormone stimulation test is warranted to assess the hypothalamic-pituitary-thyroid gland axis.
- Measurement of free T4 (fT4) by dialysis – this test has recently been evaluated for diagnostic use in horses and provides a more accurate evaluation of thyroid gland function, particularly when non-thyroidal illness is suspected. Serum fT4 concentrations vary less than tT4 concentrations and better reflect the influence of non-thyroidal disease. Low fT4 concentrations have been detected in horses suffering from colic, diarrhoea, and respiratory disease.
- Hormone stimulation tests – a TSH stimulation test was used in the past, but this hormone is not available for purchase at the time of writing. The TRH response test is therefore used and a peptide with the biological activity of this hormone is available for purchase. This peptide is purchased from suppliers of laboratory chemicals and the most widely used product (P2161 thyrotropin-releasing hormone acetate) is manufactured by Sigma Chemicals (St Louis, MO) and is dissolved in physiological saline. A compounding pharmacy within the United States (Phoenix Pharmaceuticals, Inc., Belmont, CA) also supplies TRH. The test is performed by collecting a pre-injection blood sample and then injecting 1.0 mg (total dose regardless of body weight) intravenously. Post-injection blood samples are collected 2 and 4 hours later. It was previously thought that the normal response could be defined by detecting a >2-fold increase in serum tT3 and tT4 concentrations at 2 and 4 hours post-injection, respectively. However, lower responses are also detected in healthy horses, so this test is difficult to interpret. An absent or markedly blunted (<1.25-fold) response would be supportive of hypothyroidism, but should be accompanied by clinical signs.

Treatment

Treatment consists of once-daily administration of thyroid hormone orally in the form of levothyroxine sodium. Thyro-L® (Vet-A-Mix, Division of Lloyd, Inc., Shenandoah, Iowa) is widely used in the United States, and one teaspoon provides 12 mg levothyroxine sodium. In humans that suffer from hypothyroidism, a dosage of levothyroxine sodium is selected to return serum TSH concentrations to within normal range, but this assay is not available for horses. Levothyroxine sodium is usually administered empirically at a dosage of 12 or 24 mg/day. Note that levothyroxine sodium is administered at a supraphysiological dosage of 48 mg/day for 3 to 6 months when treating obesity and IR in horses with equine metabolic syndrome. When treatment is discontinued, the dosage is lowered incrementally over 2 to 4 weeks to allow endogenous thyroid hormone production to be restored.

9.8 HYPERTHYROIDISM

There are two published reports of hyperthyroidism in the horse and both affected animals had unilateral thyroid tumours that were successfully removed surgically. A 21-year-old gelding presented with an enlarged thyroid gland that was later determined to be a thyroid adenocarcinoma. This horse presented with the problems of weight loss and episodic tachypnoea and tachycardia. The mass was surgically removed along with one lobe of the thyroid gland and the horse suffered from transient side effects including seizures, but then fully recovered. In this patient, the serum tT4 concentration was within reference range, but the fT4 concentration was elevated. The second report describes a 23-year-old gelding with a thyroid adenoma that presented with emaciation, tachycardia, fever, and fractious behaviour. Serum tT3 (1.15 ng/mL) and tT4 (46.5 ng/mL) concentrations were elevated in this patient and it responded well to hemithyroidectomy.

9.9 THYROID ADENOMA

Although hyperthyroidism has been associated with thyroid gland tumours and there are rare reports of C-cell tumours in horses, almost all thyroid tumours are non-functional thyroid adenomas. Unilateral involvement of one lobe is most common and horses are usually middle-aged (10 to 20 years of age) or older when enlargement is detected. Localized swelling near the throat latch region is the most common presenting complaint, so thyroid tumours are sometimes mistaken for abscessed lymph nodes secondary to *Streptococcus equi* infection. Most thyroid adenomas only require regular monitoring and no further action should be taken. Metastasis of thyroid tumours has not been reported in horses.

Only thyroid adenomas that begin to compress the trachea or locally invade should be surgically removed. Smaller freely moveable masses can be removed for cosmetic reasons, but owners should be advised that this is not medically necessary and ligation of multiple

vessels makes the surgery challenging. It is recommended that thyroid tumours be managed conservatively unless: (1) there are clinical signs or laboratory evidence of hyperthyroidism or a calcium–phosphorus imbalance (C-cell tumour), (2) adjacent structures including the trachea are compressed and coughing or exercise intolerance are reported, or (3) the tumour is not freely moveable when palpated, indicating local invasion. Hemithyroidectomy does not require thyroid hormone supplementation, but horses that undergo complete thyroidectomy should be supplemented indefinitely. Hypocalcaemia does not develop as a result of thyroidectomy because parathyroid glands are located both within and outside the thyroid gland in horses.

9.10 HYPERPARATHYROIDISM

Calcium metabolism is dependent upon dietary intake, efficient absorption within the intestinal tract, maintenance of normal blood ionized calcium concentrations, storage of calcium within the bone, and retention or excretion of calcium by the kidneys. Two hormones are primarily involved in calcium homeostasis, with vitamin D being a third hormone when it is in its active form (calcitriol). Parathyroid hormone (**PTH**) is secreted by parathyroid cells within tissues of the thyroid gland and by parathyroid glands located along the neck as far down as the mediastinum. This hormone plays the most important role in calcium homeostasis because it provides minute-to-minute control of blood ionized calcium concentrations. Increased PTH secretion causes blood calcium levels to rise and this is achieved through four mechanisms:

1. Kidney – more calcium is retained (and more phosphorus is lost) within the renal tubules under the action of PTH, and this hormone stimulates 1α-hydroxylase, which catalyses the conversion of 25-hydroxycholecalciferol to 1,25-dihydroxycholecalciferol (active vitamin D_3).
2. Intestine – increased production of 1,25-dihydroxycholecalciferol within the kidney increases active absorption of calcium by the intestines.
3. Bone (fast pool) – bone fluid surrounding osteoclasts represents a readily available source of calcium within bone. Calcium from the bone fluid moves quickly into the circulation under the action of PTH.
4. Bone (slow pool) – parathyroid hormone stimulates osteoclast proliferation within the bone and resorption of calcium from the inorganic matrix. This process takes more than 24 hours to occur, which is why it is referred to as the slow pool.

Calcitonin lowers calcium concentrations and prevents hypercalcaemia. Administration of exogenous calcium stimulates calcitonin release from parafollicular cells (or C cells) of the thyroid gland and this hormone inhibits osteoclast activity. There are reports of C-cell tumours in horses, but the condition is rare.

Aetiology

- Primary hyperparathyroidism – elevated PTH concentrations may be attributed to the presence of a parathyroid tumour. However, the parathyroid glands are very difficult to locate in the horse because they can be found throughout the neck, so this diagnosis is difficult to confirm and address with surgery. Hypercalcaemia is detected in affected horses. Horses may present with enlargement of the head, which is a result of osteodystrophia fibrosa of the facial bones, or osteopenia of long bones. Osteodystrophia fibrosa develops as calcium is reabsorbed from bones and replaced by connective tissue, and the thin facial bones enclosing the sinuses are the most affected (Figure 9.4).

Figure 9.4 Osteodystrophia fibrosa of the facial bones in a 17-year-old pony mare with presumed primary hyperparathyroidism.

- Pseudohyperparathyroidism (hypercalcaemia of malignancy) – detection of hypercalcaemia should prompt thorough examination of the horse for evidence of neoplastic disease. Some tumours secrete peptides that possess the biological activity of PTH. Production of this PTH-related peptide (**PTHrp**) has been associated with lymphosarcoma, squamous cell carcinoma, and mesothelioma.
- Nutritional secondary hyperparathyroidism (**NSH**) – blood calcium and phosphorus concentrations often fall within reference ranges, but hyperphosphataemia is sometimes detected in affected horses. Osteodystrophia fibrosa is also associated with this condition and it has many common names, including Big Head Disease, Bran Disease, or Miller's Disease. This problem arises from a dietary excess of phosphorus relative to calcium and is associated with feeding grain-only diets. Maintenance of the recommended 2:1 ratio of calcium and phosphorus will prevent this condition from occurring. High-oxalate pastures can also lead to NSH because oxalate inhibits absorption of calcium from the intestine. Diagnosis of NSH is made from the history and detection of increased renal excretion of phosphorus.
- Renal secondary hyperparathyroidism – phosphorus retention as a result of renal disease can increase PTH secretion and inhibit 1α-hydroxylase, and therefore the amount of active vitamin D_3. However, this is offset by the retention of calcium when horses enter renal failure, so renal secondary hyperparathyroidism rarely develops.

Clinical signs and clinicopathological findings

- Osteodystrophia fibrosa of the facial bones resulting in head enlargement.
- Loosening of the teeth.
- Intermittent shifting limb lameness with osteopenia confirmed by radiographs.
- Hypercalcaemia (and variable phosphorus levels) with primary hyperparathyroidism or pseudohyperparathyroidism. Ionized calcium concentrations should be measured.
- Hyperphosphataemia with NSH.

Diagnosis

- Signalment, clinical signs, and feed analysis.
- Both PTH (reference range, 0.25 to 2.0 pmol/L) and PTHrp can be measured by commercial laboratories. In the United States, the Diagnostic Center for Population and Animal Health (Michigan State University, East Lansing, MI) offers both assays.
- Fractional excretion (FE; also called fractional clearance) of electrolytes to document excessive loss of phosphorus in the urine in the case of NSH. Phosphorus and creatinine are measured in both the plasma and urine and the FE of phosphorus (PO_4) is calculated according to the formula:

$$FE_{Phosphorus} = \frac{[Urine\ PO_4]}{[Serum\ PO_4]} \times \frac{[Serum\ creatinine]}{[Urine\ creatinine]} \times 100$$

  A value of <0.5% is expected for the healthy horse, so >0.5% is indicative of NSH.
- Radiographic loss of the dental laminae durae.

Treatment

- Primary hyperparathyroidism caused by a parathyroid gland tumour can only be corrected through surgical resection.
- Dietary imbalances should be corrected if NSH is suspected. Alfalfa hay provides additional calcium and calcium carbonate (limestone) can be administered orally.
- Response to treatment is variable and facial enlargement can persist despite correction of the diet.

Prevention

Nutritional secondary hyperparathyroidism can be prevented by maintaining a calcium–phosphorus ratio equal to or greater than 2 : 1.

FURTHER READING

Selected references

Aleman M, Watson JL, Williams DC et al (2006) Myopathy in horses with pituitary pars intermedia dysfunction (Cushing's disease). Neuromuscul Disord 16:737–744.

Beech J, Boston R, Lindborg S, Russell GE (2007) Adrenocorticotropin concentration following administration of thyrotropin-releasing hormone in healthy horses and those with pituitary pars intermedia dysfunction and pituitary gland hyperplasia. J Am Vet Med Assoc 231:417–426

Durham AE, Rendle DI, Newton JE (2008) The effect of metformin on measurements of insulin sensitivity and beta cell response in 18 horses and ponies with insulin resistance. Equine Vet J 40:493–500

Eiler H, Frank N, Andrews FM et al (2005) Physiologic assessment of blood glucose homeostasis via combined intravenous glucose and insulin testing in horses Am J Vet Res 66:1598–1604.

Frank N, Elliott SB, Brandt LE et al (2006) Physical characteristics, blood hormone concentrations, and plasma lipid concentrations in obese horses with insulin resistance. J Am Vet Med Assoc 228:1383–1390.

Frank N, Geor RJ, Bailey SR et al (2010) Equine metabolic syndrome. J Vet Intern Med 24:467–475

Geor RJ, Harris P (2009) Dietary management of obesity and insulin resistance: countering risk for laminitis. Vet Clin North Am Equine Pract 25:51–65, vi.

Hubert JD, Beadle RE, Norwood G (2002) Equine anhidrosis. Vet Clin North Am Equine Pract 18:355–369.

Hustace JL, Firshman AM, Mata JE (2009) Pharmacokinetics and bioavailability of metformin in horses. Am J Vet Res 70:665–668

Johnson PJ, Slight SH, Ganjam VK et al (2002) Glucocorticoids and laminitis in the horse. Vet Clin North Am Equine Pract 18:219–236.

McFarlane D (2011) Equine pituitary pars intermedia dysfunction. Vet Clin North Am Equine Pract 27:93–113

Stewart AJ, Behrend EN, Wright JC et al (2011) Validation of a low-dose ACTH stimulation test in healthy adult horses. J Am Vet Med Assoc 239:834–841.

Tinworth KD, Boston RC, Harris PA et al (2012) The effect of oral metformin on insulin sensitivity in insulin-resistant ponies. Vet J 191:79–84

Chapter 10

Haematopoietic and immune systems

David G.M. Sutton, Debra C. Sellon

CONTENTS

10.1 Diagnostic approach to haematopoietic and immune system diseases 195
10.2 Diagnostic features of common haematopoietic and immune disorders of horses 199
10.3 Anaemia secondary to haemorrhage 200
10.4 Immune-mediated haemolytic anaemia 201
10.5 Microangiopathic anaemia 202
10.6 Heinz body haemolytic anaemia 202
10.7 Equine infectious anaemia 202
10.8 Equine piroplasmosis (babesiosis) 202
10.9 Anaemia due to rickettsial disease 203
10.10 Iron deficiency anaemia 203
10.11 Anaemia of chronic disease 203
10.12 Generalized bone marrow suppression or failure 204
10.13 Immune-mediated thrombocytopenia 204
10.14 Disseminated intravascular coagulation 204
10.15 Purpura haemorrhagica 205
10.16 Hereditary defects of haemostasis 206
10.17 Vitamin K deficiency 206
10.18 Polycythaemia 206
10.19 Fell Pony syndrome 207
10.20 Severe combined immunodeficiency (SCID) 207
10.21 Agammaglobulinaemia 207
10.22 Selective IgM deficiency 207
10.23 Transient hypogammaglobulinaemia 208
10.24 Failure of passive transfer 208
10.25 Neonatal isoerythrolysis 208
10.26 Neonatal piroplasmosis 208
10.27 Lymphoma (lymphosarcoma) 208
10.28 Plasma cell myeloma 209
10.29 Sporadic lymphangitis ('monday morning leg'; 'monday morning disease') 209
Further reading 209

10.1 DIAGNOSTIC APPROACH TO HAEMATOPOIETIC AND IMMUNE SYSTEM DISEASES

Primary disorders of the haematopoietic and immune system are relatively rare in the horse. Alterations in peripheral blood parameters are more commonly responses to primary disease in other body systems or reflect local or systemic infection. Clinical signs associated with abnormalities of these systems may therefore be varied and complex. Clinicopathological evaluation of the blood is usually included in the diagnostic plan for investigation of many abnormalities identified on physical examination. Further evaluation of the haematopoietic and immune systems themselves may require specific diagnostic investigation, including ultrasonography, cytological evaluation of effusions and histopathological evaluation of lymph node, tissue or bone marrow biopsies. A detailed history is vital to reach probable differential diagnoses, as is a thorough physical examination.

History

- Age – congenital abnormalities of the haematopoietic and immune system are most likely to become apparent in horses <1 year of age. Most congenital immunosuppressive disorders present before 3 months.
- Breed – congenital abnormalities are also more likely in purebred horses, although they can occur in mixed-breed animals.
- Specific questioning should be directed towards establishing: Presence of exercise intolerance? Change in demeanour, appetite or resting respiratory rate? Extent and rate of any weight loss. History of epistaxis, bleeding episodes or discolored urine? Appearance of limb swelling or unexplained masses?

© 2013 Elsevier Ltd
DOI: 10.1016/B978-0-7020-2801-4.00010-9

- In-contact horses – a history of similar clinical signs in other horses may suggest an infectious or toxic aetiology. A history of *Streptococcus equi* infection on the premises might suggest purpura haemorrhagica in a horse with vasculitis.
- Season – red maple leaf toxicity is more common in the late summer and autumn.
- Environment – fallen trees or poor pasture management might predispose to ingestion of toxic plants. Tick exposure is required for haemolytic anaemia due to piroplasmosis.
- Routine herd health records – weight loss, ventral oedema, and unexplained recurrent fever in an untested horse might indicate equine infectious anaemia. Recent vaccination against *Streptococcus equi* might suggest a diagnosis of purpura haemorrhagica in a horse with vasculitis.
- Previous illnesses – repeated incidences of infectious diseases may be suggestive of an immune dysfunction. *Cryptosporidium* spp., adenovirus infections, *Candida* spp. and *Pneumocystis carinii* infections have been diagnosed in horses with immunological disease.

Physical examination

A complete physical examination is required to identify all clinical abnormalities. Specific diagnosis of a primary (rather than secondary) haematopoietic and/or immunological disease is then likely to require further diagnostic investigation.

Non-specific systemic signs

- Depression.
- Weight loss.
- Pyrexia.
- Pain.
- Tachycardia, tachypnoea.

Mucous membranes

Change in mucous membrane colour is key to detection of disease and all available surfaces should be examined, including scleral colour. Pale mucous membranes accompanied by tachycardia, tachypnoea, and a systolic heart murmur are highly suggestive of severe anaemia. However, marked anaemia is required before changes in mucous membrane colour are readily detected. In contrast, congested mucous membranes with prolonged capillary refill time are consistent with polycythaemia. Petechial or ecchymotic haemorrhages occur with thrombocytopenia or vasculitis. Detection of petechiation in the presence of endotoxaemia may be the first indication of impending disseminated intravascular coagulation (DIC). Icteric sclera may be seen in haemolytic anaemia, but will occur also with prolonged inappetance.

Haemorrhage

Often the source of blood loss is evident and a result of trauma. Intrathoracic haemorrhage (fractured rib/intercostal artery laceration) or intra-abdominal bleeding (splenic rupture, mesenteric tear) may require cytological evaluation of pleural or peritoneal fluid respectively or ultrasound examination. Prolonged bleeding from venipuncture sites, petechial or ecchymotic haemorrhages, or melaena occur with severe thrombocytopenia (usually $<20 \times 10^9$/L). Haematoma formation following relatively minor trauma, or internal haemorrhage into the thorax or abdomen may be indicative of coagulation factor abnormalities or severe DIC.

Urinalysis is important. Whole red cells may be detected in several different conditions of the urinary tract, including idiopathic renal haematuria, or haemoglobinuria in cases of haemolytic anaemia.

Palpation

Jugular veins should be palpated for patency, thickening, heat or painful foci. Jugular vein thrombosis associated with venipuncture is common in horses with endotoxemia or other conditions that cause blood to be hypercoaguable. Thrombosis may occur secondary to DIC, repeated or unskilled venipuncture or infection.

Pitting oedema of the distal extremities, ventral abdomen or head may be an indication of hypoalbuminaemia, lymph obstruction or stasis, congestive heart failure (rare), vasculitis or lymphangitis. Oedematous swelling that is hot and painful on palpation is likely to result from vasculitis, lymphangitis or cellulitis. Ventral thoracic and sternal oedema may be caused by pleural effusions, often with increased filling of the jugular veins. Similarly, ventral abdominal oedema or hind limb oedema may occur secondary to lymphatic obstruction resulting from ascites or enlarged abdominal lymph nodes. Rectal examination and abdominal ultrasound should therefore be included as part of the diagnostic investigation.

Submandibular and prescapular lymph nodes should be palpated for size, consistency, and pain. Single lymph nodes may be enlarged because of abscessation or in reaction to localized inflammatory processes. Multicentric lymphoma may cause generalized lymphadenopathy but enlargement of superficial lymph nodes due to lymphoma is relatively rare.

Peripheral blood evaluation

A complete blood count (CBC) can give invaluable information about the erythron (red cell mass), the leukon (white cell mass) and platelets. Blood samples should be collected from any patent superficial vein when the horse has been calm for at least 1 hour. Catecholamine-induced contracture of the spleen in response to excitement causes a significant increase in red cell numbers. Catecholamine release also causes physiologic leukocytosis with increased neutrophil and lymphocyte counts. Similarly, samples should not be taken after ingestion of a large concentrate meal, as this will reduce circulating plasma volume. By contrast, sedatives such as acepromazine and xylazine cause splenic relaxation and relative anaemia such that blood samples should always be taken prior to sedation.

Erythrocytes

The RBC count, haemoglobin, haematocrit and packed cell volume (PCV) are the most frequently measured parameters of erythrocyte quantitation. Haemoglobin concentrations should be approximately one-third of the PCV. This will be increased with intravascular haemolysis. Anaemia is a decrease in the circulating red blood cell (RBC) mass (decreased RBC count, haemoglobin and packed cell volume) as a result of decreased rate of erythrocyte production or increased rate of erythrocyte destruction or loss. Polycythaemia is an increase in the circulating RBC mass. Red blood cell indices can be used to characterize anaemia:

- Mean corpuscular volume (MCV) – sometimes increased in horses with regenerative anaemia; decreased with iron deficiency anaemia.
- Mean corpuscular haemoglobin (MCH) – increased with intravascular haemolysis; decreased with iron deficiency anaemia.
- Mean corpuscular haemoglobin concentration (MCHC) – increased with intravascular haemolysis; decreased with iron deficiency anaemia.
- Red cell distribution width (RDW) – may be increased in regenerative anaemia, reflecting anisocytosis.

The appearance of the erythrocytes should be evaluated on a stained blood smear. Examples of abnormal RBC morphology and its interpretation include:

- Poikilocyte – any abnormally shaped erythrocyte.
- Anisocytosis – varying sizes of erythrocytes are observed.
- Polychromasia – varying colours of erythrocytes are observed, usually due to variable haemoglobin and RNA content.
- Spherocyte – spherical erythrocytes occasionally observed in horses with immune-mediated anaemia.
- Howell–Jolly bodies – basophilic nuclear remnants occasionally observed in the cytoplasm of erythrocytes of normal horses.
- Heinz bodies – oxidized precipitated haemoglobin observed as round structures extending from the edge of the RBC membrane. Indicative of oxidative damage to the RBC with subsequent intravascular or extravascular haemolysis.

Horse erythrocytes show prominent rouleaux formation on smears, not to be confused with agglutination. Reticulocytes do not appear in the peripheral circulation. Assessment of regenerative response in anaemia is therefore more dependent on measurement of individual red cell parameters as above, collection of sequential blood samples or evaluation of bone marrow biopsies or aspirates. Erythropoiesis in anaemia is slower than in other species, taking 10 days to reach the maximum rate after haemorrhage. Cell life span is 140–155 days.

A reference range for haematology values in the adult horse is given in Table 10.1.

'Hot-blooded' horses such as Thoroughbreds and Arabians have a greater red cell count and haemoglobin content than 'cold-blooded' draught breeds, with pony and donkey values lying between the two. Red cell variables are also affected by age. Neonatal PCV is initially high, returning to adult levels over the course of the first week. Foals have microcytosis and increasing MCV until 3 years old, with variable hypochromasia and anisocytosis. In pregnant mares, PCV, haemoglobin and RBC all increase slightly towards the end of gestation, and then decrease during lactation.

Table 10.1 Reference range for haematology values in the adult horse

Haematology value	Reference range
Red cell count (RBC), × 10^{12}/L	6.5–11.6
Packed cell volume (PCV), L/L	0.30–0.48 (30–48%)
Haemoglobin, g/dL	11.3–17.9
Mean corpuscular volume (MCV), fl	39–49
Mean cell haemoglobin (MCH), pg	12.3–17.0
Mean corpuscular haemoglobin concentration (MCHC), g/L	31.0–38.6
Red cell distribution width (RDW), %	<20.0
Leukocytes (WBC), × 10^9/L	5.3–11.0
Neutrophils, × 10^9/L	2.1–6.0
Band neutrophils, × 10^9/L	0.0–0.2
Lymphocytes, × 10^9/L	1.7–5.4
Neutrophil : lymphocyte ratio	1.5 : 1.0
Monocytes, × 10^9/L	0.0–0.7
Eosinophils, × 10^9/L	0.0–0.8
Basophils, × 10^9/L	0.0–0.1
Platelets, × 10^9/L	100–350

Leukocytes

Both total WBC count and differential WBC counts should be assessed.

- Neutropenia – decreased neutrophil count – most often a result of a shift of neutrophils from the circulating to the marginating pools due to excessive tissue demand. This may occur with endotoxaemia, septicaemia, and a wide variety of acute bacterial, rickettsial or viral disorders. In endotoxaemia the neutrophils develop basophilic cytoplasm, with granulation, vacuolation and presence of dark-staining Dohle inclusion bodies ('toxic neutrophils'). Chronic neutropenia may occur with severe infectious or inflammatory diseases or bone marrow suppression.
- Band neutrophils – are immature neutrophils released prematurely into the circulation as a response to high tissue demand ('left shift'). A degenerative left shift occurs when the number of immature cells exceeds the number of mature neutrophils because of an inability of the bone marrow to keep pace with demand. It is considered a poor prognostic indicator.
- Neutrophilia – increased neutrophil count – may occur in response to stress, excitement, exercise, glucocorticoid or adrenaline (epinephrine) administration, or almost any infectious or inflammatory condition. May be accompanied by a regenerative left shift. Often follows neutropenia caused by endotoxaemia or infection.
- Eosinopenia – decreased eosinophil count – may be normal or may occur in response to stress or glucocorticoid administration.
- Eosinophilia – increased eosinophil count – occurs in response to some parasitic infections or hypersensitivity reactions e.g. allergic respiratory disease; allergic dermatitis. It may also occur as a primary myeloproliferative disease, or in response to systemic neoplasia.
- Basophilia – increased basophil count – may be observed in response to allergic, inflammatory, or neoplastic diseases or in association with lipaemia. (Basopenia is not clinically significant.)
- Monocytosis – increased monocyte count – occasionally observed in association with chronic inflammation. (Monocytopenia is not clinically significant.)
- Lymphopenia – decreased lymphocyte count – occurs in response to stress, glucocorticoid administration, many acute viral infections, endotoxaemia and in foals with severe combined immunodeficiency (SCIDs).
- Lymphocytosis – increased lymphocyte count – associated with excitement, exercise, adrenaline (epinephrine) administration, lymphocytic leukaemia, and chronic antigenic stimulation.

Foals are born with a higher neutrophil : lymphocyte ratio than adults. Neutrophil production decreases, and lymphocyte output increases during the first few weeks, such that lymphocyte : neutrophil ratio reaches 1.5 : 1.0 by 2 months.

Platelets

Total counts in the horse are usually less than in other species. Platelets may clump if they have become activated in vivo or in vitro.

Clumped platelets may be observed at the feathered edge of a stained blood smear, particularly with EDTA samples. If platelets have clumped, automated counts are not accurate. A repeat sample should be collected in sodium citrate and counted for more accurate data. Below 100×10^9/L thrombocytes is of clinical significance, but frank haemorrhage is unlikely to occur unless platelet numbers fall below 20×10^9/L. Splenic contraction will raise numbers.

Miscellaneous tests

- Autoagglutination – agglutination of erythrocytes that is not eliminated by diluting blood in sterile saline is suggestive of immune-mediated haemolytic disease (dilute 1 drop EDTA blood: 4 drops saline).
- Coombs test – direct Coombs test detects antibody and/or complement on the surface of erythrocytes; indirect Coombs test detects the presence of circulating anti-erythrocyte antibodies. Either or both may be positive in immune-mediated haemolytic disease.
- Osmotic fragility test – assay assesses stability of erythrocytes in saline solutions of decreasing tonicity. Increased fragility is associated with immune-mediated haemolytic disease.
- Flow cytometry – flow cytometric detection of platelets with surface-bound IgG can be used to identify cases of immune-mediated thrombocytopenia (IMT). Diagnosis of IMT otherwise depends on elimination of other causes of thrombocytopenia.
- Cross-matching – major cross-match checks for compatibility between blood donor erythrocytes and any alloantibody present in patient serum. The minor cross-match checks for compatibility between donor serum and patient erythrocytes. Both tests are important prior to whole blood transfusion. The minor cross-match is most important prior to plasma transfusion.
- Iron status – may be important in characterization of some anaemias. Serum ferritin concentration is indicative of stored hepatic and splenic iron. Prussian blue stain of bone marrow aspirates also allows assessment of iron stores. Serum iron concentration reflects total quantity of transport iron in the plasma (iron bound to transferrin). Total iron binding capacity (TIBC) is the amount of iron that plasma transferrin could bind if fully saturated. Percentage transferrin saturation is calculated from TIBC and serum iron concentration and should normally be approximately 30%.

Bone marrow evaluation

Bone marrow aspirates and core biopsies may be obtained from the sternebrae on the ventral midline between the forelimbs, from the tuber coxae with the needle directed toward the opposite coxofemoral joint, or from the proximal ribs. The area is clipped and surgically scrubbed. A small amount of local anaesthetic is deposited in the subcutaneous tissues and periosteum. A stab incision is made with a small surgical blade. A 16-gauge or larger bone marrow needle is inserted and forced into the marrow cavity with constant pressure and rotational movement. The stylet is removed and a sterile syringe containing a small amount of EDTA or sodium citrate as anticoagulant is used to aspirate material. Smears should be made immediately and stained for cytological analysis. Core biopsies are similarly obtained using a 10-gauge Jamshidi needle and fixed for routine histopathological analysis.

Bone marrow myeloid to erythroid ratio (M:E ratio) should be 0.5–0.9, lower than in other species. An M:E ratio of <0.5 is indicative of erythrocyte regeneration or myeloid suppression. Polychromatophilic reticulocytes should be seen in the bone marrow if erythropoiesis is ongoing.

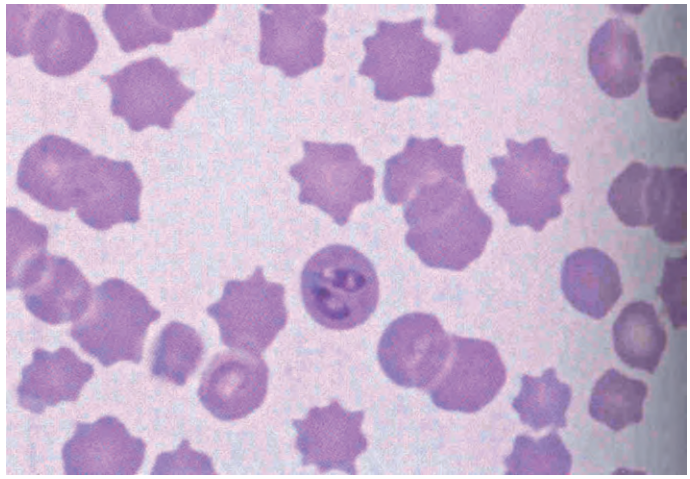


Figure 10.1 Peripheral blood smear showing erythrocyte infected with *Babesia caballi*.

Evaluation of haemostatic function

Evaluation of haemostatic function should include a total platelet count, tests to evaluate the function of extrinsic and intrinsic coagulation cascades, and assays to estimate fibrinolytic activity. Samples should be collected in a citrate tube and values compared to a healthy control horse.

Prothrombin time (PT) or one-stage prothrombin time (OSPT) measures the function of the extrinsic and common coagulation cascades.

Activated partial thromboplastin time (APTT) measures the function of the intrinsic and common coagulation pathways. Prolonged PT with a normal APTT indicates a deficiency of factor VII in the extrinsic pathway (e.g. in hepatic failure). Prolongation of both PT and APTT is more likely to occur with poisoning (e.g. vitamin K antagonists) or DIC. Mucous membrane changes in a horse with DIC are shown in Figure 10.3.

Fibrin degradation products (FDPs) are increased with excessive fibrinolysis, which commonly occurs in horses with DIC.

Antithrombin III (ATIII) activity is increased in some acute inflammatory disorders or hepatic diseases, but decreased with DIC and protein-losing nephropathy or enteropathy.

Platelet count <10 000/μL is associated with haemorrhage (Figure 10.2). Platelet function may be evaluated with template bleeding time assays or platelet aggregation tests.

Ancillary diagnostic tests

- Urinalysis – haemoglobinuria occurs with intravascular haemolysis. Haematuria with erythrocytes in urine sediment occurs with urinary tract haemorrhage and/or inflammation.
- Thoracocentesis – may be useful for identification of intrathoracic haemorrhage. Abnormal lymphocytes observed on cytology is compatible with a diagnosis of thoracic lymphoma.
- Abdominocentesis – may be useful for identification of intra-abdominal haemorrhage.
- Testing of faecal occult blood may help to indicate if gastrointestinal or respiratory tract haemorrhage has occurred but tests are considered to be unreliable.

Immune function evaluation

Immunoglobulin assays. Screening tests to assess for gross alterations in serum IgG concentration include zinc sulphate turbidity, glutaraldehyde coagulation, latex agglutination, or enzyme linked immunosorbent assay (ELISA). More accurate quantification is available with radial immunodiffusion (RID) techniques. RID test kits are available to accurately quantitate serum IgG, IgM, IgG(T) and IgA. Production of antibody in response to vaccination with a specific antigen may be used to assess responsiveness of the humoral immune system.

Lymphocyte typing. Peripheral lymphocyte counts are extremely low in Arabian foals with severe combined immunodeficiency (SCID). Dramatically increased counts with abnormal cell morphology may be observed in horses with lymphocytic neoplasias. Monoclonal antibodies are available for identification of lymphocyte subsets (CD4, CD8, pan T cell, B cell) in peripheral blood by flow cytometry. Staining of tumours with antibodies against CD3 and CD79 has been used to classify T-cell and B-cell tumours respectively.

Blood typing. Several laboratories perform erythrocyte alloantigen typing and alloantibody detection for prevention or diagnosis of neonatal isoerythrolysis (NIE). These laboratories will also perform lymphocyte typing for progeny verification. NIE is more likely to occur in the progeny of horses with blood groups Aa or Qa.

Cellular immune function. Intradermal skin testing with phytohaemagglutinin (PHA) may be used to assess delayed type hypersensitivity. A crude estimate of T cell function may be obtained with lymphocyte blastogenesis assays using PHA, concanavalin A or pokeweed mitogen. More specific tests may be available in some research laboratories.

10.2 DIAGNOSTIC FEATURES OF COMMON HAEMATOPOIETIC AND IMMUNE DISORDERS OF HORSES

Figure 10.2 Spontaneous bilateral epistaxis in mare with profound thrombocytopenia (6×10^9/L). Blood transfusion stopped haemorrhage. Immune-mediated thrombocytopenia found to be secondary to cutaneous haemangiosarcoma.

Table 10.2 Diagnostic features of haematopoietic and immune diseases

Haematopoietic or immune disorder	Diagnostic clinicopathological features
Acute haemorrhage	CBC – PCV, RBC count, plasma protein may be normal Thoracocentesis – haemorrhagic if bleeding into thorax Abdominocentesis – haemorrhagic if bleeding into abdomen
Chronic haemorrhage	CBC – PCV, RBC count, plasma protein decreased Iron stores – decreased if chronic external blood loss Bone marrow – increased erythropoiesis (unless iron deficient)
Anaemia of chronic disease	Anaemia mild (PCV 20–30%); often leukocytosis Elevated fibrinogen and serum amyloid A
Red maple leaf toxicity	CBC – Heinz body anaemia Methaemoglobin – increased as a percentage of total haemoglobin Urinalysis – haemoglobinuria
Immune-mediated anaemia	CBC – autoagglutination; anaemia; normal serum protein Coombs test – positive Urinalysis – may have haemoglobinuria RBC fragility – increased

Table 10.2 Continued

Haematopoietic or immune disorder	Diagnostic clinicopathological features
Immune-mediated thrombocytopenia	CBC – thrombocytopenia; variable thrombocyte size PT, PTT, FDP – within normal limits Bone marrow – normal or increased numbers of megakaryocytes
Equine infectious anaemia	CBC – may be normal Agar gel immunodiffusion (AGID) or C-ELISA – positive; PCR positive
Equine babesiosis	CBC – anaemia, thrombocytopenia; organisms may be observed in erythrocytes (Figure 10.1) Urinalysis – haemoglobinuria PCR positive
Disseminated intravascular coagulation	CBC – thrombocytopaenia PT, PTT, FDP – increased ATIII – decreased; often neutropenia and lymphopenia due to endotoxaemia
Purpura haemorrhagica	CBC – inflammatory leukogram; normal platelet count PT, PTT, FDP – normal
Fell Pony syndrome	CBC – severe non-regenerative anaemia, progressing from 2–3 weeks of age; Normal T-cell populations but severe B-lymphopenia; IgM significantly decreased from 4 weeks of age Bone marrow – severe erythroid hypoplasia with elevation of myeloid : erythroid ratio
Severe combined immunodeficiency (SCID)	CBC – persistent severe lymphopenia Presuckle serum IgM – decreased
Generalized bone marrow suppression	CBC – thrombocytopenia; anaemia; neutropenia Bone marrow – all cell lines decreased; may observe fibrosis, fat infiltration, or abnormal (neoplastic) cell populations. May be neoplastic cells in peripheral circulation.
Relative polycythaemia	CBC – increased PCV, haemoglobin, RBC count; increased plasma protein if due to dehydration PaO_2 – normal Erythropoietin – normal Bone marrow – normal
Absolute polycythaemia	CBC – increased PCV, haemoglobin, RBC count PaO_2 – decreased if secondary to hypoxaemia; normal if primary Erythropoietin – normal or increased Bone marrow – increased erythropoiesis

10.3 ANAEMIA SECONDARY TO HAEMORRHAGE

As in other species, anaemia in the horse is caused by either increased loss of red cells, increased destruction or by decreased production. The former two are the most common causes of severe anaemia in horses. Chronic disease is a relatively common cause of less severe chronic anaemia. The causes of increased loss of cells or increased destruction are multiple, and must be identified in order to treat the condition effectively.

Haemorrhage: aetiology and pathogenesis

Blood loss or haemorrhage may be external or internal, acute or chronic.

- External blood loss is often a result of trauma and easily recognized and diagnosed with a visual examination. An exception to this is haemorrhage into the gastrointestinal tract. Significant haemorrhage into the gastrointestinal tract may occur with gastric ulceration or intestinal neoplasia without evident melaena or haematochezia.
- Internal blood loss, into the thorax or abdomen, allows the body to re-use (autotransfuse) up to two-thirds of the erythrocytes lost. Common causes include severe exercise induced pulmonary haemorrhage (EIPH), post partum rupture of the uterine artery and splenic rupture subsequent to trauma.
- Acute blood loss – often associated with normal RBC parameters because all blood components have been lost in equal volumes. Redistribution of fluid from interstitial tissue into the intravascular space results in a detectable drop in total plasma protein after 6 hours. Anaemia only becomes apparent after 12–24 hours, with the lowest values in PCV occurring at 36–48 hours post haemorrhage. Bone marrow exhibits increased erythropoiesis by 3 days post haemorrhage; maximal by 10 days. Horses do not release reticulocytes into the circulation during a regenerative response. PCV should increase by 0.32–0.42% per day during maximum regeneration. Complete restoration of circulating red cell count may require 1 to 3 months. Weekly assessment of PCV and TP aids monitoring of regeneration process. Serial bone marrow aspirates must be examined if regenerative responses are questionable.

Table 10.3 Signs of acute and chronic blood loss

Acute blood loss	Chronic blood loss
Hypovolaemic shock	May be anorexia and weight loss
Tachycardia	Decreased exercise tolerance
Tachypnoea	Pale mucous membranes
Pale mucous membranes	Weakness, lethargy
Poor venous distension	Tachycardia when stressed
Thready pulse	Tachypnoea when stressed
Pansystolic heart murmur	Vitals may be normal at rest
Weakness, lethargy	Pansystolic heart murmur
Renal hypoxia: oliguria	
Acute renal failure	

Clinical signs

Signs depend on the source of haemorrhage, plus the rate and total volume of blood loss. Mares with post partum uterine artery rupture for example show signs of colic followed by progressive tachypnoea, tachycardia and pallor. Signs of hypovolaemic shock develop when horses lose more than 30% of circulating blood volume (i.e. 24 mL/kg or 9 L of blood for a 400 kg horse). Signs are more subtle in cases of chronic blood loss due to physiological adaptation, such that vital parameters may only be elevated when the horse is exercised or stressed. Packed cell volume may decrease as far as 8–10 L/L in chronic cases before clinical signs become readily apparent at rest.

A summary of clinical signs is shown in Table 10.3.

Diagnosis

If the haemorrhage is acute all red blood cell parameters may be within normal limits. If the haemorrhage occurred more than 24 hours prior to presentation, RBC count, PCV, haemoglobin and plasma protein concentration should be decreased.

- External haemorrhage – clinical signs and/or history.
- Intrathoracic haemorrhage – ultrasonography, radiography, and/or thoracocentesis. History of fading in race should suggest EIPH, as well as upper airway endoscopic evaluation.
- Intra-abdominal haemorrhage – abdominocentesis, ultrasonography.
- Gastrointestinal haemorrhage – melaena, haematochezia, gastroscopy, faecal occult blood test.

Treatment

1. Stop the haemorrhage.
2. Acute severe blood loss – consider:
   - Isotonic crystalline intravenous fluids at 20 mL/kg/h. Will improve circulating volume and reduce clinical signs of hypovolaemic shock. Aim to replace three times measured volume of blood lost.
   - Hypertonic saline (2–4 mL/kg). Only to be given if horse showing signs of significant hypovolaemia and colloids or blood not available, as may increase blood loss and mortality.
   - Colloidal solutions (up to 15 mL/kg) required if TP falls below 45 g/L with crystalloid fluids.
   - Blood transfusions from a compatible donor (15 mL/kg). Requirement based on clinical signs and knowledge of estimated blood loss.
3. Chronic blood loss – consider iron supplementation:
   - Ferrous sulphate at 2 mg/kg body weight orally per day. Often not necessary as equine diet relatively high in iron.
   - Never administer parenteral iron dextran to a horse (potential severe anaphylactoid reactions).
4. Whole blood transfusions.
   - A transfusion is likely to be required in the chronic scenario if PCV falls below 8 L/L or
   - below 12 L/L in the acute situation. As acute haemorrhage takes several hours for PCV to equilibrate, requirement to transfuse should be based on clinical signs of hypovolaemic shock, and anticipated if blood loss will exceed 30% of circulating volume.
   - Major and minor cross-matches should be compatible. Or, use previously untransfused gelding of similar breed as donor. First transfusion rarely causes adverse reaction as production of erythrocyte alloantibodies relatively uncommon. Antibodies will develop to transfused cells, but generally take 3–10 days, making risk of reaction to second transfusion also small if given within that time period.
   - Collect 6–8 L of blood from adult 450 kg donor.
   - Use citrate-phosphate-dextrose (CPD) or acid-citrate-dextrose (ACD) as anti-coagulants at 15 mL per 100 mL blood collected.
   - Administer through an in-line filter. Give at slow drip for first 10 minutes and monitor for adverse reactions. If safe, continue at 15–25 mL/kg/h.
   - Adverse reactions: increased heart rate, increased respiratory rate, dyspnoea, fever, trembling, weakness, hypotension, diarrhoea, abdominal pain, anaphylaxis, shock, pulmonary oedema. In case of reaction discontinue transfusion. Hypotension treated with intravenous crystalline fluids. Anaphylaxis treated with IV adrenaline (1 : 1000 solution at 4.5–9 mL per 450 kg horse).
   - Blood transfusion is a temporary solution only. Transfused cells will be removed from the circulation within 2 to 6 days by mononuclear phagocytes. The cause of blood loss must be identified and treated for successful outcome.

10.4 IMMUNE-MEDIATED HAEMOLYTIC ANAEMIA

Lysis of circulating erythrocytes due to attachment of antibody and/or complement to the surface of the cells. May be acute or chronic which determines progression of clinical signs. Multiple antigens have been implicated in coating erythrocytes and triggering destruction. These include bacterial agents such as *Streptococcus* spp., viruses (e.g. equine infectious anaemia), parasites (e.g. *Theileria equi*) and neoplasia (e.g. lymphosarcoma) and several drugs.

Intravascular haemolysis is generally complement-mediated. Extravascular haemolysis occurs when opsonized erythrocytes are removed from circulation by tissue macrophages.

Pathogenesis

- Alteration of erythrocyte membrane during systemic disease, with subsequent formation of antibodies against new antigens.
- Production of antibodies against normal erythrocyte antigens (true primary autoimmune disease).
- Immune complex deposition on erythrocyte membranes, usually via Fc or complement receptors.

- Drug combines with erythrocyte membrane to create new antigen.
- Drug combines with a carrier molecule and antibody is deposited as an immune complex on erythrocyte membranes.
- Drug induces true autoantibody production.
- Neonatal isoerythrolysis – discussed in Chapter 20.

During intravascular haemolysis, haemoglobin is released and combines with haptoglobin prior to removal from the circulation by tissue mononuclear phagocytes. If the binding capacity of haptoglobin is exceeded, free haemoglobin accumulates in the plasma and is eliminated via the kidneys and detected on urinalysis. Excessive free haemoglobin can be nephrotoxic. As haemoglobin is degraded in phagocytic cells, the haem moiety is converted enzymatically to biliverdin and then bilirubin. Unconjugated bilirubin is released into the circulation and binds to plasma albumin. Hepatocytes take up the bilirubin, conjugate it, and release it into the gastrointestinal tract via the bile. Both intravascular and extravascular haemolysis is associated with increased serum concentrations of unconjugated (indirect) bilirubin. Plasma bilirubin may remain within reference range in chronic IMHA. However, an acute haemolytic crisis causes icterus and yellowing of the sclera and mucous membranes within about 12 hours.

Clinical signs

Signs referable to primary underlying disease:

- Tachycardia, tachypnoea.
- Weakness, lethargy.
- Systolic heart murmur.
- Icterus.
- Discoloured urine (haemoglobinuria).

Diagnosis

- History, clinical signs.
- Decreased RBC count, PCV.
- Haemoglobinaemia, haemoglobinuria (if intravascular haemolysis).
- Increased serum bilirubin.
- Positive Coombs test or autoagglutination.
- Increased erythrocyte osmotic fragility.

Treatment

- Treat underlying disease condition if possible.
- Discontinue current drug therapy if possible.
- Parenteral corticosteroids, e.g. dexamethasone at 0.04–0.1 mg/kg IV or IM. (Corticosteroids have been associated with an increased risk of laminitis in horses.)
- Blood transfusion if severe, life-threatening anaemia.
- Fluid diuresis to protect against pigment nephropathy if intravascular haemolysis is occurring.

10.5 MICROANGIOPATHIC ANAEMIA

Erythrocytes are damaged while passing through abnormal vasculature or due to increased turbulence of flow and are removed from the circulation. The anaemia is therefore secondary to a primary disease process. Haemangiosarcoma is the commonest neoplastic cause, and may also cause immune-mediated thrombocytopenia. Other causes include DIC (see below), acute fulminant liver failure, uraemic crises, vasculitis and acquired arteriovenous shunting. Treatment depends on identification of the underlying cause but horses with this type of anemia are likely to have a poor prognosis.

10.6 HEINZ BODY HAEMOLYTIC ANAEMIA

Heinz bodies are oxidized, precipitated haemoglobin visible in Wright's stained or new methylene blue stained blood smears.

Aetiology and pathogenesis

If ferrous (Fe^{2+}) iron in haemoglobin is oxidized to ferric (Fe^{3+}) iron, methaemoglobin is formed. Methaemoglobin is incapable of carrying oxygen. If oxidative stress overwhelms normal protective mechanisms in erythrocytes, methaemoglobin accumulates and imparts a brownish discoloration to blood. Simultaneously, sulphydryl groups are oxidized, resulting in haemoglobin denaturation and precipitation, Heinz body formation, and intravascular haemolysis.

Familial methaemoglobinaemia and haemolytic anaemia have been described in American saddlebred horses with decreased erythrocyte glutathione reductase activity. Cytological examination shows eccentrocytes in the circulation – red cells containing eccentric haemoglobin crystals.

Toxins that can cause oxidative haemolytic anaemia include:

- Wilted red maple leaves (*Acer rubrum*).
- Onions (*Allium canadense*).
- Phenothiazine.

Clinical signs

- Peracute death with brownish discoloration of blood.
- Haemolytic syndrome – icterus, brown blood (methaemoglobinaemia), haemoglobinuria, bilirubinaemia, bilirubinuria, Heinz body anaemia, death.

Diagnosis

- Clinical signs.
- History of access to toxin or familial predisposition.
- Increased blood methaemoglobin as a percentage of total blood haemoglobin (normal = 1–2%).

Treatment

- Remove all horses from access to toxins.
- Activated charcoal to decrease toxin absorption (even if no clinical signs).
- Fluid diuresis to protect against pigment nephropathy.
- Dexamethasone.
- Ascorbic acid as an antioxidant – 30 mg/kg twice daily in intravenous fluids.
- Transfuse if life-threatening anaemia.

10.7 EQUINE INFECTIOUS ANAEMIA

See Chapter 19.

10.8 EQUINE PIROPLASMOSIS (BABESIOSIS)

This disease is widespread or endemic in most subtropical regions of the world. Northern Europe, the UK, Eire and New Zealand are among the few regions from where the disease has not been reported. Piroplasmosis leads to haemolytic anaemia of variable severity, occasionally leading to death in naive animals, haemolytic crises in neonates and abortion in mares. Distribution is determined mainly by the distribution of the requisite tick vectors.

Aetiology

Aetiological agents are intraerythrocytic protozoans, *Babesia caballi* and *Theileria equi*. The parasites develop to sporozoites in the salivary gland of the infected tick vector before transmission to the horse in saliva. Some 12–14 days after attachment of infected ticks, merozoites invade erythrocytes and undergo division and are seen as pyriform parasites within the erythrocyte (Figure 10.1). Infection can occur after inoculation of infected blood into susceptible animals or via contaminated needles. Not thought to be spread by other biting insects. In utero infection of equine fetuses by both Babesia species can occur. *T. equi* is highly pathogenic and may infect up to 80% of erythrocytes.

Clinical signs

- In endemic areas immunity develops such that subclinical infections with low-grade anaemia are common.
- Fever, anorexia, depression, icterus, petechiation, tachypnoea and weakness occur in acute cases.
- Cardiac dysrhythmias reported but rare compared with other species.
- Colic, impactions followed by diarrhoea, weight loss, poor performance and pale mucous membranes seen in more chronic cases.
- Weakness at birth with anaemia, icterus, fever and subsequent petechiation characterizes neonatal piroplasmosis.
- Anaemia, haemoglobinaemia, thrombocytopenia, increased serum bilirubin concentration and possible haemoglobinuria and acute renal failure in acute cases.

Diagnosis

- Clinical signs and identification of aetiological agents on stained blood smears (Figure 10.1).
- Complement fixation test in combination with indirect fluorescent antibody (IFA) test for detection of carrier and chronic states.
- PCR method available for simultaneous detection of *T. equi* and *B. caballi*, used for screening prior to export.
- Clinical signs frequently appear secondary to underlying disease or infection.

Treatment

- To suppress clinical signs: imidocarb dipropionate at 2.4 mg/kg IM once. Precede with glycopyrrolate 0.0025 mg/kg IV to minimize gastrointestinal effects of imidocarb.
- *B. caballi* can be eliminated by two sequential daily treatments with imidocarb. *T. equi* is more difficult to eliminate, requiring higher doses of imidocarb that are likely to have renal and hepatic toxicity.
- Imidocarb is more toxic to donkeys than horses.
- Supportive treatment including blood transfusions, fluid therapy, antibiotics, vitamin and nutritional support often required in severe cases of piroplasmosis.

10.9 ANAEMIA DUE TO RICKETTSIAL DISEASE

The rickettsial organisms *Neorickettsia risticii* (causative agent of Potomac horse fever) and *Anaplasma phagocytophilum* (equine ehrlichiosis) (see Chapter 19) may both cause anaemia, usually accompanied by leukopenia, thrombocytopenia and fever. Both infections occur primarily in the United States but have been reported in Europe and elsewhere. *N. risticii* is acquired via ingestion of trematodes present in aquatic insects and *A. phagocytophilum* is tick borne. *N. risticii* infects blood monocytes and has a predilection for the glandular epithelium cells of the large colon, causing diarrhoea as a major clinical sign and laminitis. *A. phagocytophilum* invades neutrophil and eosinophil cytoplasm, causing petechiations and oedema in subjects, with necrotizing vasculitis and anaemia.

Diagnosis

Clinical signs plus serological confirmation of exposure, or faecal PCR are used to diagnose Potomac horse fever. Examination of blood smears is more useful for diagnosis of *A. phagocytophilum* in which definitive diagnosis is based on identification of ehrlichial inclusion bodies in granulocytes. Nested polymerase chain reaction (PCR) and ELISA tests are also available for diagnosis of equine ehrlichiosis.

Treatment

Both organisms are susceptible to intravenous oxytetracycline and usually to oral doxycycline.

10.10 IRON DEFICIENCY ANAEMIA

Rarely associated with deficiencies of dietary iron or abnormal iron absorption. Usually a result of chronic external blood loss, often due to gastrointestinal tract haemorrhage.

Diagnosis

- Decreased serum ferritin and bone marrow iron stores.
- Microcytic, hypochromic anaemia (decreased MCV and MCHC).
- Decreased percentage saturation transferrin.
- Increased TIBC.

Treatment

- Identify and eliminate the source of haemorrhage.
- Oral supplementation with product containing ferrous sulphate.
- Do not give parenteral iron dextran because of risk of anaphylactic reaction.

10.11 ANAEMIA OF CHRONIC DISEASE

Mild to moderate normocytic, normochromic, non-regenerative anaemia associated with chronic inflammatory conditions or neoplasia. PCV often in range 0.20–0.30 L/L with elevated fibrinogen and serum amyloid A concentrations and leukocytosis.

Pathogenesis

- Prevention of iron release from storage pools and so iron is unavailable for haem synthesis.
- Defective bone marrow response to erythropoietin.
- Decrease in erythrocyte lifespan.

Diagnosis

- Normocytic, normochromic anaemia of mild to moderate severity.
- Concurrent chronic inflammatory, parasitic or neoplastic disease.
- Normal to increased TIBC.
- Normal to increased serum ferritin and bone marrow iron stores.
- Normal to decreased percent saturation of transferrin.

Treatment

Identify and treat the primary disease process. Oral iron supplementation is not indicated because systemic iron stores are usually normal to increased.

10.12 GENERALIZED BONE MARROW SUPPRESSION OR FAILURE

Bone marrow suppression or failure usually affects all cell lines with resulting pancytopenia (decreased circulating erythrocytes, leukocytes and platelets).

Aetiology

- Aplastic anaemia – may be secondary to viral, bacterial or neoplastic disease, but in most cases a predisposing factor is not identified. An autoimmune pathogenesis has been proposed for some cases.
- Phenylbutazone toxicity – rare; recovery reported with discontinuation of drug administration.
- Chronic renal disease – decreased erythropoietin results in moderate to severe non-regenerative anaemia.
- Haematopoietic neoplasia – almost any cell line may be affected. Neoplastic cells crowd out normal cell development. Neoplastic cells may be observed in circulation.
- Myelofibrosis.

Diagnosis

Bone marrow aspirate and/or core biopsy will usually confirm diagnosis. In horses with aplastic anaemia, the marrow is devoid of recognizable stem cells. Hypoplastic marrow is yellow due to fat infiltration; myeloid, erythroid and megakaryocytoid cells are decreased.

Treatment

Usually no treatment is recommended. Prognosis is poor. Any prior drug therapy should be discontinued if possible. Idiopathic aplastic anaemia may respond to parenteral corticosteroid therapy.

10.13 IMMUNE-MEDIATED THROMBOCYTOPENIA

Immune-mediated thrombocytopenia (IMTP) frequently occurs secondary to bacterial, viral, protozoan or neoplastic diseases, but may occur occasionally as an idiopathic or true autoimmune condition.

Pathogenesis

Pathogenesis is similar to that described for immune-mediated haemolytic anaemia.

Clinical signs

Clinical signs are most commonly observed when platelet counts are $<20 \times 10^9$/L. Frank haemorrhage is likely to occur when platelets are depleted below 10×10^9/L. See Figure 10.2.

- Petechial and ecchymotic haemorrhages of mucous membranes.
- Prolonged bleeding following venipuncture.
- Epistaxis (Figure 10.2). May be present in absence of petechiation.
- Melaena.
- Hyphaema.

Diagnosis

- Antiplatelet antibody determination using platelet factor 3 assay – difficult assay to perform with consistent, reliable results.
- Rule out other causes of thrombocytopenia; low platelet count with normal PT and PTT.
- Bone marrow aspirate or core biopsy usually shows normal to increased megakaryocytes.
- Flow cytometry has been used to identify platelets with surface bound IgG.

Treatment

- Treat underlying disease.
- Corticosteroids – dexamethasone at 0.1 mg/kg IM or IV once or twice daily. Gradually taper the dose to once daily in the morning. Do not abruptly discontinue therapy.
- Fresh whole blood or platelet rich plasma if life-threatening haemorrhage is occurring.
- If refractory to corticosteroids, azathioprine at 3.0 mg/kg by mouth once daily.

10.14 DISSEMINATED INTRAVASCULAR COAGULATION

Disseminated intravascular coagulation (DIC) is a pathological activation of coagulative and fibrinolytic systems, ultimately leading to microvascular thrombosis and secondary ischaemic organ failure. Utilization of coagulative factors in widespread inappropriate coagulation leads to a deficiency of factors available for appropriate coagulation and widespread haemorrhage is the result. Clinically, the appearance of petechial or ecchymotic haemorrhages in an endotoxaemic or septicaemic horse may herald the onset of DIC (see Figure 10.3).

Figure 10.3 Horse with DIC showing multiple petechial and ecchymotic haemorrhages in the mucosal membranes. Thrombocytopenia, with prolonged PT and APTT and increased FDPs was present.

Pathogenesis

- Coagulative and fibrinolytic cascades are often triggered by circulating endotoxin during gastrointestinal diseases or sepsis. Endotoxin is the lipopolysaccharide component of the external cell wall of Gram-negative bacteria.
- Other potential initiating factors include intravascular haemolysis, bacteraemia, viraemia, neoplasia, immune complex disorders, burns, hepatic or renal disease, or severe dystocia with fetal death in utero.
- Procoagulant inflammatory mediators are released from activated macrophages during DIC. These include tissue factor, tumour necrosis factor, platelet activating factor and interleukins 1 and 6.
- In early stages, thrombotic tendencies may be observed. However, DIC is recognized more commonly as a haemorrhagic disease because consumption of coagulative factors leads to an inability of the blood to continue normal haemostatic function.

Clinical signs

- Thrombosis after venipuncture.
- Petechia, ecchymoses (Figure 10.3), prolonged bleeding following venipuncture, epistaxis.
- Signs referable to organ dysfunction, including acute renal failure or laminitis.
- Signs referable to the primary underlying disease process.

Diagnosis

The diagnosis of DIC is usually justified in a horse with multiple haemostatic abnormalities and thrombotic or haemorrhagic tendencies.

- In very early stages, PT and PTT may be decreased.
- Later, PT and PTT will be prolonged.
- Increased concentration of circulating FDPs.
- Thrombocytopenia.
- Decreased plasma ATIII activity.

Treatment

- Treat the underlying disease process.
- Maintain peripheral perfusion and systemic acid/base/electrolyte status with appropriate fluid therapy.
- Heparin is useful only if plasma ATIII activity is adequate.
- Non-steroidal anti-inflammatory drugs are beneficial if the horse is endotoxaemic. Appropriate antibiotic therapy must be administered if the horse is septic.
- Heparin-activated fresh platelet-rich plasma may be administered if life-threatening haemorrhage is occurring. Heparin increases antithrombin 3 (ATIII) activity.

Prognosis

Early recognition of DIC and aggressive therapy are critical. Resolution of any underlying disease process will improve the prognosis. Clinical signs of haemorrhage are associated with a poor prognosis. Early aggressive treatment of impending endotoxaemia to prevent development of DIC is more likely to have successful outcome.

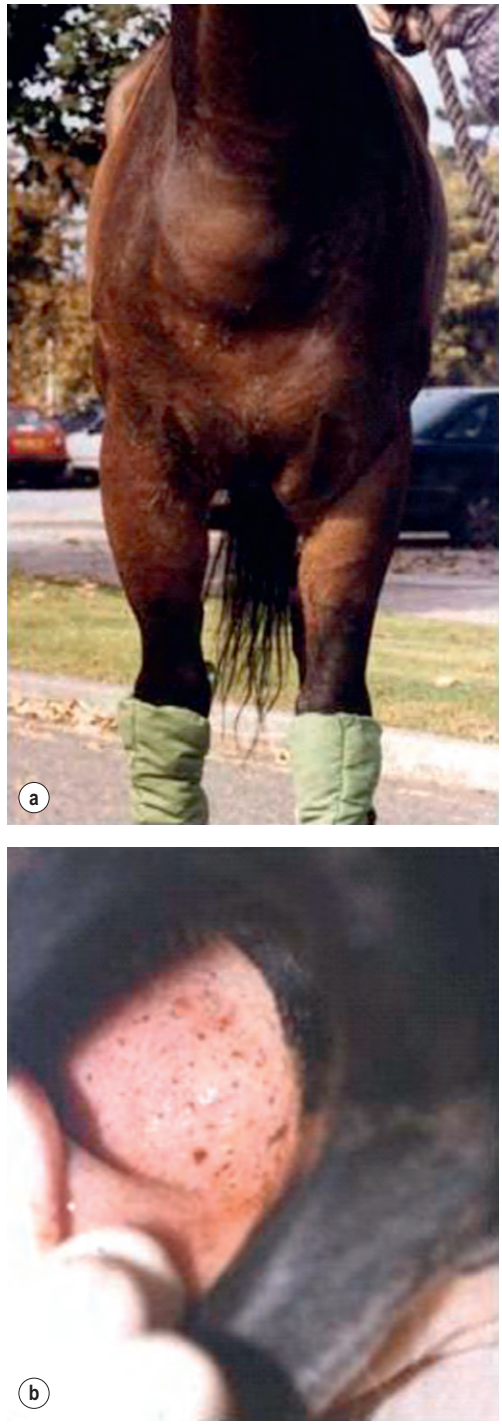


Figure 10.4 (a) Horse with purpura haemorrhagica and vasculitis of the forelimbs, subsequent to infection with *Streptococcus equi*. A sternal mass is seen at the base of the neck, which was an infected lymph node containing the causative agent. (b) Nasal mucosa from the horse shown in (a), demonstrating the presence of petechial haemorrhages secondary to purpura haemorrhagica.

10.15 PURPURA HAEMORRHAGICA

An immune-mediated vasculitis that usually occurs secondary to a streptococcal or viral respiratory tract infection (see Figure 4a). Petechial haemorrhages also occur in this condition, but in contrast to DIC, thrombocyte count is usually within the normal reference range.

Pathogenesis

Purpura haemorrhagica after *Streptococcus equi* infection occurs because of deposition of circulating immune complexes in small subcutaneous vessels. Immune complexes activate complement with influx of leukocytes and release of inflammatory mediators. Vascular permeability is increased with resultant oedema, haemorrhage, ischaemia and necrosis.

Clinical signs

- Fever, depression.
- Petechial haemorrhages of mucous membranes (Figure 10.4b).
- Painful swelling, oedema, serum exudation of distal limbs (Figure 10.4a).
- May progress to ischaemic necrosis with sloughing of skin.
- Usually not thrombocytopenic.

Diagnosis

- History and clinical signs.
- Cutaneous biopsy of affected areas will reveal neutrophilic infiltrates around small dermal and subcutaneous vessels.

Treatment

- Immunosuppressive doses of corticosteroids – dexamethasone at 0.05–0.2 mg/kg IV or IM once daily.
- Support wraps for oedematous limbs; hydrotherapy.
- Non-steroidal anti-inflammatory drugs.
- Drainage of any infected masses or abscesses followed by procaine penicillin G at 22 000 IU/kg IM twice daily or sodium penicillin at 22 000 IU/kg IV four times daily if ongoing *Streptococcus* spp. infection.
- Supportive care with intravenous fluids as indicated.

10.16 HEREDITARY DEFECTS OF HAEMOSTASIS

Most common in purebred horses. Many are inherited as autosomal recessive traits. Although they may be treated symptomatically, they cannot be cured. Affected horses should not be bred.

Clinical signs and diagnosis

- Von Willebrand's disease – defective platelet adhesion; reported in one Quarter Horse filly. Oral bleeding, conjunctival haemorrhage, prolonged bleeding after injections.
- Abnormalities of contact phase of coagulation – prekallikrein deficiency in Belgian horses and Miniature horses. Prolonged PTT with a normal PT. Affected horses may or may not have clinical signs of a bleeding tendency.
- Classic haemophilia – factor VIII deficiency; diagnosed in Thoroughbreds, Standardbreds, Arabians, and Quarter horses. Present with severe spontaneous haemorrhage or excessive haemorrhage following minor trauma or surgery. Prolonged PTT and normal PT. Factor VIII activity is <10% of normal equine plasma.
- Factor IX and XI deficiency – diagnosed in one Arabian with concurrent factor VIII deficiency.

10.17 VITAMIN K DEFICIENCY

- Rare in horses. Clinical signs of inappropriate bleeding.
- Vitamin K is essential for production of coagulation factors II, VII, IX and X.
- May occur secondary to chronic intestinal malabsorption or chronic cholestatic disorders.
- Similar syndrome produced with warfarin toxicity. Warfarin is a vitamin K antagonist.
- Early in disease only PT is prolonged. Later, PT and PTT are prolonged.

Treat with parenteral vitamin K1 (phylloquinone) at 0.3–0.5 mg/kg q6hrs subcutaneously for warfarin toxicity. Do not administer vitamin K3 (menadione sodium bisulphite) as it is nephrotoxic.

10.18 POLYCYTHAEMIA

Polycythaemia is a real or apparent increase in the circulating RBC mass. It is usually recognized as an increase in RBC count, PCV and haemoglobin concentration.

Classification

- Relative polycythaemia – a decrease in the relative plasma volume due to haemoconcentration or splenic contraction (common).
- Absolute polycythaemia – a real increase in RBC numbers due to an increase in erythropoiesis (very rare).

Aetiology and pathogenesis

Relative polycythaemia is commonly a result of dehydration secondary to decreased fluid intake or excessive water loss in diarrhoea, urine or sweat. Endotoxaemia may cause fluid shifts from plasma to interstitial space. Excitement, exercise or adrenaline may increase PCV by up to 50% due to splenic contraction and release of sequestered erythrocytes into the circulation.

Primary absolute polycythaemia occurs as a primary myeloproliferative disease (polycythaemia vera).

Secondary absolute polycythaemia is a result of increased circulating erythropoietin concentrations. It may accompany severe right-to-left shunting of blood through the heart or great vessels (e.g. tetralogy or pentalogy of Fallot), chronic hypoxia associated with severe pulmonary disease, or as an adaptation to high altitudes. Erythropoietin-like substances can be released from some tumours (e.g. renal or hepatocellular neoplasms).

Clinical signs

Mucous membranes are muddy red to blue. Capillary refill time is prolonged and horses appear weak or lethargic. Despite the increased RBC numbers, oxygen delivery to peripheral tissues is decreased because of increased blood viscosity. In cases of relative polycythaemia clinical signs of dehydration, endotoxaemia, underlying disease conditions, and/or excitement may be observed.

Diagnosis

Relative polycythaemia is diagnosed in horses with increased PCV, haemoglobin, or RBC count in conjunction with clinical signs of dehydration, excitement, or exercise. In dehydrated horses, the total serum protein concentration is often increased. Absolute polycythaemia is characterized by a persistently increased PCV, haemoglobin and RBC count in the face of normal hydration and relaxed attitude. Primary absolute polycythaemia is confirmed by bone marrow evaluation, normal serum erythropoietin concentrations, and normal PaO_2 but is extremely rare. Secondary absolute polycythaemia is accompanied by increased serum erythropoietin concentrations, decreased PaO_2 and clinical signs of an underlying disease process that would produce chronic hypoxia. Absolute polycythaemia secondary to neoplasms is characterized by normal to increased serum erythropoietin concentrations and normal PaO_2.

Treatment

Relative polycythaemia due to excitement or exercise will resolve spontaneously as the horse relaxes. Relative polycythaemia due to dehydration should be treated by correcting fluid losses and treating any underlying disease condition. In horses with absolute polycythaemia, the underlying condition must be identified and treated, if possible. Periodic phlebotomy may provide some symptomatic relief.

10.19 FELL PONY SYNDROME

A congenital fatal syndrome well recognized in the Fell Pony breed in the UK, North America and Europe that leads to progressive anaemia and immunodeficiency. There is failure of B-cell expansion in the periphery. Endogenous humoral response is affected with failure to produce IgM and IgA. Genetic inheritance with transmission as a single autosomal recessive gene. A DNA test has been developed recently that should allow detection of carrier status and elimination of such animals from the breeding population.

Clinical signs

Foals of either sex may be affected by the syndrome and appear unaffected at birth but fail to thrive. Loss of condition and signs of immunodeficiency progresses from 3–4 weeks of age as maternal antibody wanes. Clinical signs relate to severe anaemia and the development of multisystemic infections.

- Weight loss and lethargy are first signs.
- Pseudomembranous lingual coating and failure to suck.
- Tachycardia, tachypnoea and pale mucous membranes due to severe anaemia.
- Watery diarrhoea, often due to secondary *Cryptosporidium parvum* infection.
- Coughing (secondary adenoviral pneumonia; *Pneumocystis carinii*; bacterial infections).
- 100% mortality usually by age of 3 months.

Diagnosis

The condition should be suspected in young foals of this breed developing ill-thrift and anaemia. New DNA test is available for specific confirmation but diagnosis aided by clinical signs and laboratory tests:

- Profound non regenerative anaemia (PCV <15%).
- B-lymphopenia and significant reduction in IgM from 4 weeks, after waning of maternal colostrum derived antibody; T-lymphocytes not affected.
- Very few erythroid precursors on bone marrow aspirate and elevated myeloid : erythroid ratio.
- Thymic hypoplasia and absence of secondary lymphoid follicles.

Treatment

Foals respond to initial supportive therapy but the disease is invariably fatal by 3 months of age due to progressive anaemia and secondary opportunistic infections. The bone marrow at post mortem depicts pure red blood cell hypoplasia.

10.20 SEVERE COMBINED IMMUNODEFICIENCY (SCID)

A severe, inherited stem cell defect in Arabians and Arabian crossbred horses, which prevents maturation of B and T cells and inhibits expression of antigen-specific receptors on the cell surface. The condition is caused by a deletion in the gene encoding the catalytic subunit of DNA dependent protein kinase (DNA-PKcs). It is transmitted as an autosomal recessive trait with an incidence of 2–3% in Arabian foals, and carrier prevalence of up to 25%. Carriers are asymptomatic.

Clinical signs

Unable to mount specific cell-mediated or humoral immune responses, affected foals appear normal at birth but usually develop clinical signs by 2 months as maternal antibodies wane. Most affected foals die by 5–6 months of age from a variety of opportunistic infections (bacterial, viral, fungal, or protozoal) rarely seen in normal foals. Recurrent respiratory infections, particularly caused by *Pneumocystis carinii* or adenovirus, are suggestive of SCID.

Diagnosis

- Clinical signs and pedigree. A DNA PCR test is used to detect the gene. Testing requires submission of a blood or hairpluck sample from the selected individual.
- Persistent lymphopenia (<500–1000/μL).
- Absence of serum IgM in pre-suckle samples or sample collected after 3 weeks of age.
- Thymic hypoplasia with characteristic histopathological changes in lymphoid tissue.

Treatment and prognosis

Affected foals die despite intensive therapy for opportunistic infections. Both parents of an affected foal (homozygote) are carriers (heterozygotes) for the defective SCID gene. Prevention of the disease depends on increased testing and recognition of those Arabians carrying the gene. A voluntary SCID testing programme is recommended by Arabian breed societies in different countries.

10.21 AGAMMAGLOBULINAEMIA

Agammaglobulinaemia is a rare primary immunodeficiency. As it is observed only in males of several breeds, an X-linked mode of inheritance is likely. Circulating B cells are absent. Serum concentrations of IgG and IgG(T) are low while IgM and IgA are negligible. Horses may respond transiently to appropriate antimicrobial therapy and plasma transfusion but long-term prognosis is poor due to recurrent infections.

10.22 SELECTIVE IGM DEFICIENCY

Absent or decreased serum IgM concentration with normal or increased concentrations of other serum immunoglobulins. Three clinical scenarios:

- Foals with severe bacterial infections non-responsive to therapy.
- Weanlings or yearlings that present with recurrent bacterial infections and poor growth.
- Older horses that may have recurrent infections and/or lymphoma.

The mode of inheritance is undetermined. It is frequently reported in Arabians and Quarter horses. Lymphocyte counts are within normal limits and lymphoid architecture is normal. For horses with persistent reduction in IgM the prognosis is poor with most cases dying within 2 years of developing clinical signs.

10.23 TRANSIENT HYPOGAMMAGLOBULINAEMIA

In healthy foals IgM production begins in utero, whilst autogenous IgG production starts soon after birth. In this rare disorder, there is delayed onset of immunoglobulin production. IgG and IgG(T) concentrations in affected foals are decreased between 2 and 4 months of age. Reported in Arabians, but may be underdiagnosed due to transient nature. Some cases require antimicrobial therapy or plasma to combat recurrent infections, but prognosis for recovery is good once the immune system matures.

10.24 FAILURE OF PASSIVE TRANSFER

As there is no transplacental transfer of immunoglobulins, the neonate depends on the intestinal absorption of colostral antibodies for effective humoral immunity. Failure of passive transfer (FPT) of maternal antibodies from a mare's colostrum to her foal is the commonest equine immunological disorder, occurring in up to 10% foals. FPT has been correlated to the development of neonatal sepsis. This secondary immune disorder and its potentially serious consequences are discussed in detail in Chapter 20.

10.25 NEONATAL ISOERYTHROLYSIS

Alloantibodies in a mare's colostrum will occasionally react with her foal's erythrocyte alloantigens, resulting in severe haemolytic anaemia. This condition is discussed in detail in Chapter 20.

10.26 NEONATAL PIROPLASMOSIS

Transplacental transfer of *T. equi* parasites can result in neonates with high burdens of parasites and post partum signs of severe haemolytic anaemia and icterus. The condition is differentiated from NIE by examination of a blood smear. Initial treatment consists of correcting metabolic acidosis, hypoglycaemia and promoting diuresis to reduce haemoglobinaemia, and support renal function. Specific treatment for piroplasmosis should only be instigated once adequate renal function has been restored (see section 10.8).

10.27 LYMPHOMA (LYMPHOSARCOMA)

Haemolymphatic tumours in horses are relatively uncommon but lymphoma occurs most frequently. There is no sex or breed predilection and horses of any age may be affected. Peaks in incidence have been reported between the ages of 2–6 years and between 9–15 years. The majority of equine lymphoma are comprised of small to medium T-cells, and involve multiple lymphoid tissues and abdominal or thoracic organs. Occasional B-cell tumours have been reported, many of which have significant T-cell content also (termed T-cell-rich B-cell lymphoma). Solitary tumours may affect extranodal sites but multicentric involvement is more common. Subcutaneous forms of lymphoma responsive to steroid hormones have been reported. Unlike other species, peripheral lymph nodes are often not involved, although may be enlarged in multicentric lymphoma. A high proportion of tumours will undergo necrosis such that peripheral signs may include pyrexia, with hyperfibrinogenaemia and neutrophilia.

Clinical signs

Weight loss and ventral oedema are the most common historical and physical abnormalities, and may be accompanied by depression, exercise intolerance and inappetance. Specific clinical signs are variable, depending on primary site of lesions. Paraneoplastic syndromes such as immune-mediated haemolytic anaemia, thrombocytopenia, mucosal ulceration, pruritus and alopecia have been reported.

- Generalized or multicentric form: most common. Generalized lymphadenopathy and ventral oedema. Other clinical signs depend on specific organ involvement. At necropsy, lesions are often observed in multiple internal organs.
- Intestinal (alimentary) form: weight loss, ventral oedema, intestinal malabsorption, intermittent abdominal pain and/or fever. Mesenteric lymph nodes may be palpably enlarged on rectal examination. Small intestinal wall thickening seen on transabdominal ultrasound examination in up to 50% of cases. Diarrhoea may or may not be observed. Hypoalbuminaemia. Occasional immune-mediated haemolytic anaemia and/or thrombocytopenia.
- Mediastinal or thymic form: adult horses with ventral thoracic oedema and respiratory difficulties due to bilateral pleural effusion. Enlarged thoracic lymph nodes. Bilateral jugular vein distension if masses occlude venous return.
- Cutaneous form: multiple subcutaneous nodules. May regress and reappear. Generalized lymphadenopathy is uncommon but individual local lymph nodes may be enlarged.

Diagnosis

Most common clinical laboratory abnormalities include hyperfibrinogenaemia, hypoalbuminaemia, anaemia, leukaemia, hyperglobulinaemia and thrombocytopenia. Diagnosis confirmed by neoplastic cells in aspirates or biopsies. Pleural effusions are more likely to contain exfoliative neoplastic cells than are peritoneal effusions, aiding in diagnosis of mediastinal lymphosarcoma. Tru-cut biopsy specimens rather than fine needle aspirates should be collected from enlarged lymph nodes or nodules for diagnostic purposes. In the intestinal form, xylose and glucose absorption tests are often markedly abnormal due to diffuse lymphosarcomatous infiltration of the small intestine. Exploratory laparotomy and biopsy of intestine or mediastinal lymph nodes is required for definitive diagnosis.

Horses with peripheral lymphocytosis or abnormal lymphocytes in the circulation generally have tumour myelophthisis which must be established via bone marrow biopsy.

Treatment and prognosis

Treatment is not generally recommended and prognosis is poor. The cutaneous form may transiently respond to corticosteroid therapy but lesions often recur when therapy is discontinued. The cutaneous form is the most benign form of this neoplasm and lesions may progress only very slowly. Most horses with multicentric lymphosarcoma deteriorate and require euthanasia within 7 months of diagnosis, even if treated with systemic steroids.

10.28 PLASMA CELL MYELOMA

The most common form of plasma cell myeloma in the horse is multiple myeloma, but all are rare. In multiple myeloma there is malignant proliferation of plasma cells within the bone marrow. Monoclonal gamma globulin peaks may be observed on serum protein electrophoresis.

Clinical signs

Weight loss, limb oedema and inappetence are commonest signs. Lameness, paresis, lymphadenopathy and epistaxis may occur depending on site of organ infiltration.

Diagnosis

Anaemia, hyperglobulinaemia and hypoalbuminaemia are present, with characteristic protein electrophoresis profile. Leukopenia, thrombocytopenia and circulating plasma cells may be present. Definitive diagnosis depends on demonstration of >10% plasma cells in bone marrow biopsy specimen.

Treatment

Most horses die or are euthanased within 4 months of treatment, although survival times may be prolonged by chemotherapeutic protocols. Poor prognosis.

10.29 SPORADIC LYMPHANGITIS ('MONDAY MORNING LEG'; 'MONDAY MORNING DISEASE')

An acute lameness with swelling of one or occasionally both hindlegs associated with inflammation of the lymphatics.

Aetiology and pathogenesis

The aetiology is uncertain in many cases. A nutritional basis is assumed in some cases, since the disease may occur in horses receiving full rations while taking little or no exercise (hence 'Monday morning leg'). Some cases may develop following an infection, e.g. from a puncture wound.

Inflammation of the lymphatic tracts and inguinal lymph nodes leads to lymph stasis and swelling of the limb. In protracted cases, subcutaneous fibrous tissue may be laid down and normal lymphatic drainage may be impeded; these cases may be affected by recurrent bouts of lymphangitis, and permanent thickening of the soft tissues of the limb may result.

Clinical signs

There is usually an acute progressive swelling of one hind limb and associated lameness. The limb appears painful, and the foot is 'pointed' or held off the ground. A pain response can be elicited by palpation over the ascending lymphatic tracts proximal to the medial hock and adjacent to the saphenous vein. In severe cases, the skin of the leg appears tight and may ooze serum. The body temperature is elevated.

Diagnosis

Diagnosis is based on the typical clinical signs. Ultrasonography may reveal numerous dilated lymphatics in the subcutaneous tissues. A wound may or may not be found in the leg.

If the horse has not already been treated with antibiotics, bacterial culture of a needle aspirate or of a swab taken from an area oozing serum may be attempted.

Treatment

The aims are to provide pain relief, and to treat the inflammatory reaction and infection, so that the leg is returned to mobility as soon as possible.

1. Systemic antibiotics. The antibiotic should be effective against *Staphylococcus aureus*, since this organism is frequently involved. The antibiotic may need to be changed depending on the results of culture.
2. Anti-inflammatory therapy with non-steroidal anti-inflammatory drugs. Some clinicians advocate the use of a single dose of corticosteroid early in the course of the disease.
3. Hydrotherapy with cold water is helpful in reducing the swelling.
4. The opposite leg should be supported with a bandage, and observed closely for laminitis.
5. Gentle exercise should be provided as soon as the horse is comfortable enough to be walked/ exercised.
6. Provide tetanus toxoid or antitoxin if necessary.

FURTHER READING

Crisman MV, Scarratt WK (2008) Immunodeficiency disorders in horses. Veterinary Clinics of North America, Equine Practice 24:299–310

De Waal DT, Van Heerden J (2004) Equine babesiosis. In: Du Plessis (ed) Infectious diseases of livestock. Oxford University Press, Cape Town, pp 425–434

Gardner RB, Hart KA, Stokol T et al (2006) Fell pony syndrome in a pony in North America. Journal of Veterinary Internal Medicine 20:198–203

Meyer J, DeLay J, Bienzle D (2006) Clinical, laboratory, and histopathologic features of equine lymphoma. Veterinary Pathology 43:914–924

Meyers KM, Menard M, Wardrop KJ (1987) Equine hemostasis: description, evaluation, and alteration. Clinical Pathology, Veterinary Clinics of North America, Equine Practice 3:485

Morris DD (1987) Cutaneous vasculitis in horses: 19 cases (1978–1985). Journal of the American Veterinary Medical Association 191:460

Russell KE, Sellon DC, Grindem CB (1994) Bone marrow analysis in the horse. Compendium of Continuing Education for the Practicing Veterinarian 16:1359–1367

Thomas GW, Bell SC, Carter SD (2005) Immunoglobulin and peripheral B-lymphocyte concentrations in Fell pony syndrome. Equine Veterinary Journal 37:48–52

Trogdon Hines M (2003) Immunodeficiencies of foals. In: Robinson NE (ed) Current therapy in equine medicine, 5th edn. Saunders, St Louis, p 692–697

APPENDIX

Differential diagnosis of conditions presenting with haematopoietic or immunological signs

1. Epistaxis

Exercise-induced pulmonary haemorrhage.
Progressive ethmoidal haematoma.
Guttural pouch mycosis.
Trauma.
Thrombocytopenia.
Disseminated intravascular coagulation.
Rhinitis/sinusitis.
Neoplasia.
Pneumonia/pulmonary abscess.
Foreign body.
Nasal amyloidosis.

2. Gastrointestinal blood loss

Parasites.
Ulcers.
Inflammatory bowel disease.
Neoplasia.
Coagulopathy.

3. Intra-abdominal haemorrhage

Parasites.
Trauma.
Neoplasia.
Abdominal abscess.
Ruptured uterine artery.
Aortic aneurysm.
Coagulopathy.

4. Intrathoracic haemorrhage

Trauma.
Ruptured pulmonary abscess.
Aortic aneurysm.
Neoplasia.
Coagulopathy.

5. Haematuria

Cystitis/urolithiasis.
Pyelonephritis.
Idiopathic renal haematuria.
Urinary tract neoplasia.
Coagulopathy.
Trauma.

6. Haemolysis

Neonatal isoerythrolysis.
Immune-mediated haemolytic anaemia.
Red maple leaf toxicity.
Equine infectious anaemia.
Piroplasmosis.
Disseminated intravascular coagulation.
Severe hepatic disease.
Hypotonic fluid administration.
Intravenous DMSO.
Phenothiazine toxicity.
Onion toxicity.
Familial methaemoglobinaemia.

7. Thrombocytopenia

Immune-mediated thrombocytopenia.
Lymphosarcoma.
Disseminated intravascular coagulation.
Bone marrow failure (pancytopenia).
Equine infectious anaemia.
Equine granulocytic ehrlichiosis.

8. Petechia/ecchymoses

Disseminated intravascular coagulation.
Thrombocytopenia.
Purpura haemorrhagica.
Vasculitis.
Equine infectious anaemia.
Equine granulocytic ehrlichiosis.

9. Pancytopenia

Myeloproliferative disease.
Aplastic anaemia.
Radiation.
Phenylbutazone toxicity.
Myelophthisis.
Metastatic neoplasia.

10. Non-regenerative anaemia

Chronic inflammatory disease.
Neoplasia.
Chronic renal disease.
Iron deficiency.
Hypothyroidism.
Bone marrow failure (pancytopenia).
Chronic hepatic disease.
Acute haemorrhage or haemolysis.
Nutritional deficiency.

11. Recurrent infections

Inappropriate therapy.
Failure of passive transfer of antibody.
Fell pony syndrome.
Combined immunodeficiency.
Selective IgM deficiency.
Transient hypogammaglobulinaemia.
Agammaglobulinaemia.

Chapter 11

Neurology

Yvette S. Nout, Leo B. Jeffcott

CONTENTS

11.1 Diagnostic approach to neurological diseases 212
History 212
Physical examination 212
Neurological examination of foals 215
Ancillary diagnostic tests 215
The brain 216
11.2 Foals 216
Anencephaly 216
Hydrocephalus 216
Perinatal asphyxia syndrome/hypoxic ischaemic encephalopathy/neonatal encephalopathy/neonatal maladjustment syndrome (dummies, barkers, wanderers) 216
Congenital cerebellar disease 216
Idiopathic seizures 216
11.3 Trauma 217
Forebrain syndrome 217
Optic nerve syndrome 217
Midbrain syndrome 217
Vestibular syndrome 217
Hindbrain syndrome 217
Treatment of traumatic brain injury 217
11.4 Toxic conditions 217
Hepatoencephalopathy 217
Plant poisons 217
Lead poisoning 218
Snake bite 218
Fluphenazine toxicity 218
11.5 Infectious conditions 219
Togaviral equine encephalitides: Alphavirus and Flavivirus species 219
Other viral encephalitides 220
Borna disease 220
Rabies 220
Brain abscess 220
Meningitis 221
11.6 Parasitic lesions 221
Verminous encephalitis 221
Parasitic cerebral thromboembolism 221
Equine protozoal myeloencephalitis (EPM) 221
11.7 Neoplasia 221
11.8 Miscellaneous conditions 221
Epilepsy 221
Narcolepsy 222
Head shaking 222
Temporohyoid osteoarthropathy 222
Intracarotid drug injection 222
The spinal cord 222
11.9 Ataxia 222
Clinical signs 222
Clinical aids to diagnosis 223
Specific conditions of spinal cord disease 225
11.10 Vertebral malformations 225
Occipitoatlantoaxial malformation (OAAM) 225
Cervical vertebral stenotic myelopathy (CVSM) 225
Other vertebral and spinal cord malformations 226
11.11 Trauma 226
11.12 Inflammatory conditions 227
Equine protozoal myeloencephalitis (EPM) 227
Equine herpesvirus 1 vasculitis/myeloencephalopathy 227
Other inflammatory conditions 227
11.13 Equine degenerative myeloencephalopathy (EDM) 228
11.14 Neoplasia 228

© 2013 Elsevier Ltd
DOI: 10.1016/B978-0-7020-2801-4.00011-0

11.15 Toxic conditions 228
Plant poisons (rye grass staggers, Bermuda grass staggers and Dallas grass staggers) 228
Sorghum and Sudan grass ataxia 228
11.16 Equine motor neuron disease (EMND) 228
Peripheral and autonomic nervous systems 229
11.17 Generalized conditions 229
Botulism 229
Tetanus ('lockjaw') 229
Organophosphorus poisoning 230
Lactation tetany and idiopathic hypocalcaemia 230
11.18 Peripheral nerve damage 231
Suprascapular paralysis 231
Radial nerve paralysis 231
Other peripheral nerve damage 231
Facial paralysis 231
Vestibular disease 231
Polyneuritis equi (cauda equina neuritis) 232
11.19 Peripheral neuropathies 233
Idiopathic laryngeal hemiplegia 233
Stringhalt 233
Shivering 233
11.20 Neoplasia 233
Peripheral nerve sheath tumours 233
Lymphoma 233
11.21 Conditions of the autonomic nervous system 233
Horner's syndrome 233
Grass sickness 233
Further reading 233
Appendix 234

11.1 DIAGNOSTIC APPROACH TO NEUROLOGICAL DISEASES

History

Important points that should be recorded include:

- Age, breed, sex, use; in Quarter Horses: HYPP status.
- Duration and progression of signs.
- History of trauma or other disease.
- Health of in-contact horses.
- Diet and recent changes in diet.
- Sudden or insidious onset of signs.
- Medication.
- General management (vaccination status, travel) and environment.

Physical examination

The objectives of the physical examination are to first identify the anatomical site of the lesion, and second to diagnose the cause of the disease (although this may not always be possible).

Examination of the head

1. Behaviour, e.g. seizures, head pressing, circling, aggressiveness, etc.
2. Mental status, i.e. level of consciousness and awareness, e.g. coma (complete unresponsiveness to normal stimuli), semicoma (partial responsiveness to stimuli), somnolence, depression, etc.
3. Head posture and coordination, e.g. head tilt, head swaying, jerking.
4. Cranial nerves. The nuclei of the cranial nerves are located along the brainstem, and lesions to the brainstem frequently also result in abnormalities of cranial nerve function. The nerves are examined by starting with the most rostral ones and proceeding caudally.
   - Olfactory nerve (I) is responsible for the sense of smell. Function of this nerve is difficult to assess, but may be evaluated by testing the horse's ability to smell the hand or food.
   - Optic nerve (II) is responsible for vision. The menace response is assessed by making a threatening gesture with the hand to each eye (avoiding air currents or touch) and observing eyelid blinking or movement of the head away. This reflex also relies on cranial nerve VII. Depressed or excited horses, and neonatal foals, may have deficient menace responses. Further assessment of vision may be made by creating an obstacle course. Unilateral vision deficits can be assessed by blindfolding each eye prior to walking through the obstacle course. An ophthalmoscopic examination should be performed in addition to assessment of vision.
   - Oculomotor nerve (III) is responsible for the control of the pupil (constriction) via parasympathetic fibres (this is opposed by dilator tone controlled by sympathetic pathways). The pupils are assessed for size and symmetry. The pupillary light reflex is performed by shining a bright light into each eye and observing immediate constriction of the pupil in the same eye (direct reflex) and in the opposite eye (consensual reflex). Strabismus (abnormal eye position) may be caused by disease of cranial nerves III, IV or VI, or damage to the extraocular muscles.
   - Trochlear nerve (IV) is responsible for normal eye position (along with cranial nerves III and VI). Moving the head slowly from side to side causes a rhythmic horizontal movement of each eyeball known as the oculocephalic reflex, doll's eye, or normal vestibular nystagmus. Extending the head on the neck causes the eyeballs to rotate ventrally within the orbit. The eyeballs return to a central position as the head is lowered. These normal eye movements require an intact vestibular system, intact cranial nerves III, IV and VI, and the connections between these structures.
   - Trigeminal nerve (V) contains motor fibres to the muscles of mastication, and sensory fibres from much of the head. There are three branches – mandibular, maxillary and ophthalmic. Sensory function is tested by assessing facial reflexes (observed flicking of ear, closure of eyelid, flaring of nostril and withdrawal of the labial commissure in response to touch of these areas); these reflexes also require an intact facial nerve. Facial sensation is tested by observing a cerebral response (e.g. head jerk) in response to pricking areas on the face. Loss of motor function of the trigeminal nerve results in a dropped jaw, and inability or difficulty in chewing, and drooling of saliva.
   - Abducens nerve (VI) is responsible for normal eye position (with cranial nerves III and IV).
   - Facial nerve (VII) supplies motor innervation to the muscles of facial expression via the auricular, palpebral and buccal branches. The function of the facial nerve is tested by facial

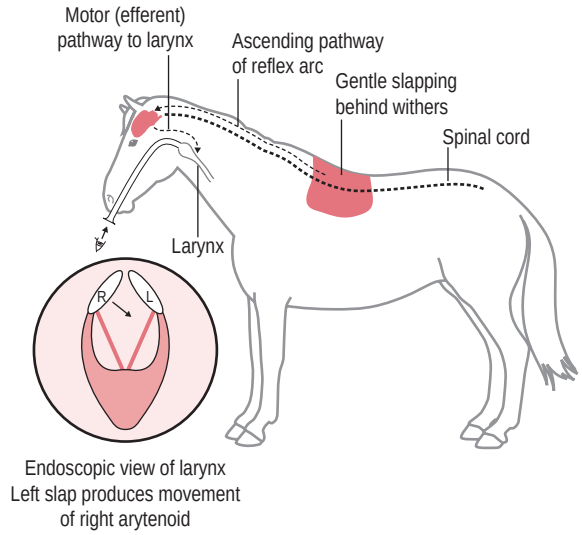


Figure 11.1 Laryngeal adductory function test ('slap test').

reflexes (which also test the function of sensory fibres of the trigeminal nerve as described above) and observation of the ability to move the ears, blink the eyelids and move the lips during feeding. The facial nerve also innervates the lacrimal and salivary glands. Facial paralysis results in drooping of the ear, ptosis, decreased tear production, and paralysis of the upper lip causing it to be pulled towards the unaffected side.

- Vestibulocochlear nerve (VIII) is responsible for hearing (cochlear division) and balance (vestibular branch). Input to the vestibular system also comes from the cerebellum and other brainstem centres. Signs of vestibular disease include a rhythmic nystagmus (fast phase usually directed away from the side of the lesion), strabismus and head tilt (towards the side of the lesion). The nystagmus may occur when the head is in a normal position (resting nystagmus) or when the head is placed in an unusual position (positional nystagmus). Central vestibular disease results in additional signs including proprioceptive deficits, ataxia, weakness and depression.
- Glossopharyngeal nerve (X), vagus nerve (XI) and accessory nerve (XI) contain sensory and motor fibres that innervate the pharynx, larynx, oesophagus and many other viscera. The normal swallowing reflex can be assessed by observing the horse eating or drinking, or by passing a nasogastric tube. The pharynx and larynx can be examined by endoscopy. The laryngeal adductor response (slap test) is tested by gently slapping one side of the chest just caudal to the dorsal scapula, while observing the larynx endoscopically. In normal horses the contralateral arytenoid adducts briefly. This reflex involves afferent pathways in the thoracic nerves and cervical spinal cord, and an efferent pathway via the recurrent laryngeal branch of the vagus (Figure 11.1). Pharyngeal paralysis results in dysphagia and nasal return of food.
- Hypoglossal nerve (XII) supplies motor innervation to the tongue. Weakness of the tongue is assessed by observing the resistance to pulling the tongue out of the mouth.

A summary of cranial nerve function tests is given in Table 11.1.

Examination of gait and posture

Abnormalities of gait may arise with either musculoskeletal or neurological problems, and the former must be ruled out before proceeding with the neurological examination.

Table 11.1 Summary of cranial nerve function tests

Cranial nerve	Major function	Reflex/response/assessment
I Olfactory	Sense of smell	
II Optic	Afferent pathway for vision and light	Menace response Pupillary light reflex Pupil size
III Oculomotor	Pupillary constriction Extraocular muscles	Pupillary light reflex Pupil size Pupil symmetry Medial movement of globe Doll's eye
IV Trochlear	Extraocular muscles	Ventrolateral rotation of globe Doll's eye
V Trigeminal	Sensory to side of face Motor to muscles of mastication	Ear, eyelid and lip (palpebral/facial) reflexes Pain perception from head Eyeball retraction Chewing, jaw tone, muscle mass Temporal/masseter symmetry
VI Abducens	Extraocular muscles	Eyeball retraction Lateral movement of globe Doll's eye
VII Facial	Motor to muscles of facial expression Facial symmetry	Ear, eyelid and lip (palpebral/facial) tone, reflexes, and movement Menace
VIII Vestibular	Afferent branch of vestibular system	Head posture Induced eyeball movement Normal vestibular nystagmus (Doll's eye) Normal gait Blindfold test
Cochlear	Sense of hearing	Response to noise
IX Glossopharyngeal	Sensory and motor to pharynx and larynx	Swallowing Gag reflex
X Vagus		Slap test
XI Accessory		
XII Hypoglossal	Motor to tongue	Tongue size and symmetry

Lesions affecting descending motor pathways result in paresis (weakness) caudal to the lesion. Such lesions can also cause overactivity of reflexes (due to damage to inhibitory pathways) resulting in spasticity (stiffness or hypometria). Damage to ascending proprioceptive fibres in peripheral nerves or white matter of the spinal cord results in ataxia (incoordination) in the limbs caudal or distal to the

lesion. Interference with the transmission of proprioceptive information to the cerebellum can result in poor control of voluntary movements and hypermetria (over-stepping).

Gait and proprioceptive deficits are assessed by observing the horse walking, trotting, turning, circling, backing, walking up and down a slope, and moving with a blindfold on. Proprioception can be assessed by walking the horse over a step or through an obstacle course, by crossing its forelegs and by forcing the horse to adopt a base-wide stance.

The degree of gait abnormality can be graded on a scale from 0 (normal) to 5 (recumbency).

- Ataxia causes an unstable, swaying gait with abnormal foot placement, which becomes worse when the horse is walked on a slope. The limbs may be circumducted, especially on turning. The horse may pivot on the affected limbs when turning or backing.
- Paresis causes dragging the feet and stumbling. These signs may be exacerbated by walking the horse in a tight circle or up a slope.
- Spasticity causes a stiff movement of the limbs with reduced flexion of the joints. Spasticity can be exaggerated by walking up and down a slope with the head elevated.
- Hypermetria results in overstepping with excessive joint movement (Figure 11.2).

A summary of the neuroanatomical localization of lesions that result in gait abnormalities is given in Table 11.2.

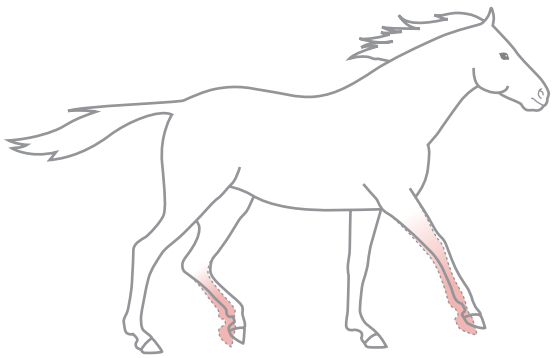


Figure 11.2 Fore- and hind limb hypermetria ('goose-stepping').

Examination of the neck and forelimbs

- The neck and forelimbs are inspected for symmetry, malformations, muscle atrophy, patchy sweating and the degree and strength of voluntary effort.
- Skin sensation and spinal reflexes over the neck and forelimbs are assessed using a pen or probe to prod the skin. This results in flinching of the cervical musculature as well as a behaviour/cerebral response such as head or body movement away from the stimulus. The cervicofacial reflex is tested by prodding caudal to the ear, which results in twitching of the ear, blinking and movement of the labial commissure on the side being tested.
- The neck should be manipulated dorsoventrally and laterally to assess the range of movement and presence of pain.
- Pushing against the shoulders (sway test) assesses the capacity of the horse to resist lateral force, and is helpful in defining paresis and/or ataxia.

Examination of the trunk and hind limbs

- The trunk and hind limbs are observed for musculoskeletal malformations, vertebral column deviations and muscle atrophy.
- Skin sensation is assessed as for the neck and forelimbs. Prominent skin flicking over the thorax and flank in response to prodding is known as the panniculus response.
- A sway test is performed by providing lateral force to the pelvis (Figure 11.3a,b). A tail pull test is performed by pulling laterally on the tail when the horse is standing still and walking (Figure 11.3c).
- A loin pressure test is performed by pressing down firmly with the fingers on the loin and dorsal hip region.

Examination of the tail and anus

- The caudal body region is assessed for asymmetry of bone or muscle. Holding the tail elevated or to one side might be normal or abnormal for an individual horse.
- Tail tone is assessed by manoeuvring the tail.
- The perineal reflex is tested by gently prodding the perineum and observing a reflex contraction of the anal sphincter and clamping down of the tail.
- Rectal palpation is performed to evaluate rectal and urinary bladder content and tone.

Table 11.2 Summary of neuroanatomical localization of lesions resulting in gait abnormalities

Lesion location	Abnormal posture	Paresis	Ataxia	Hypometria	Hypermetria
Cerebrum	+++				
Brainstem	++	++	++	++	++
Vestibular	+++		++	++	
Cerebellum	++		+++	+	+++
Spinal cord/UMN	++	++	++	++	++
Peripheral nerve/LMN	++	+++	+	(++)*	(+++)
Musculoskeletal	+	++		+	

*Usually only with very selective sensory fibre involvement.

UMN = upper motor neuron. LMN = lower motor neuron.

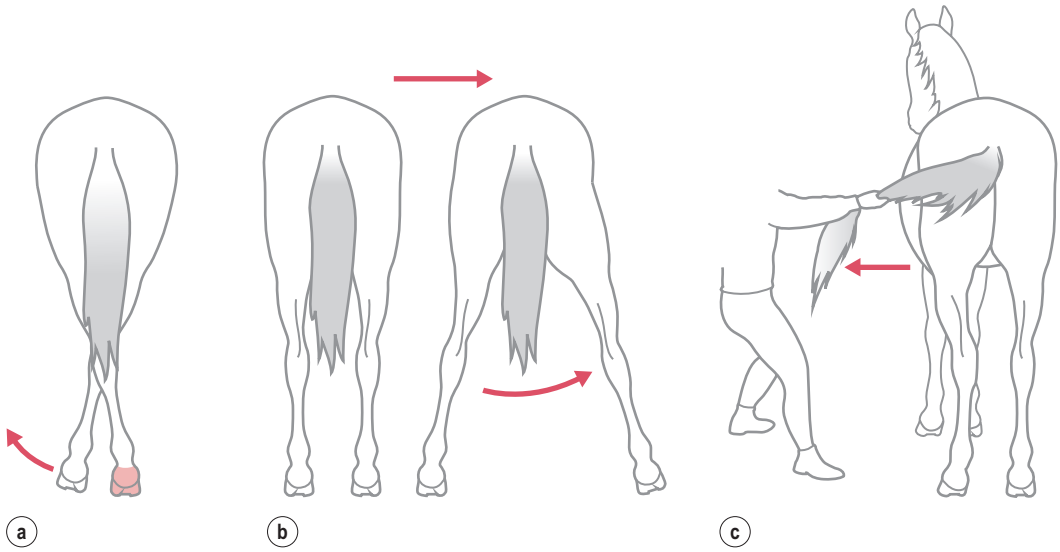


Figure 11.3 Sway response. (a) Push from quarters results in overstep of hindfoot across the midline and unsteadiness (proprioceptive deficit). (b) Response for comparison in normal horse. (c) Pulling tail laterally at walk shows weakness of hindquarters (paresis) in horses with cervical vertebral stenotic myelopathy (CVSM).

Neurological examination of foals

See also Chapter 20.

Some important differences in reflex activity and neurological function exist between foals and adult horses:

- Menace response is commonly reduced or absent in normal foals.
- Jerky head movements are commonly seen in normal foals.
- Dysmetria and mild incoordination are common in normal foals.
- Response to the slap test is variable up to one month of age.

Ancillary diagnostic tests

Cerebrospinal fluid (CSF) collection

Atlanta-occipital (AO) space. With the horse under general anaesthesia, the head is flexed at right angles to the top line of the neck. Using aseptic technique, a needle is inserted at the middle of a line drawn between the cranial borders of the wings of the atlas. The subarachnoid space is entered at a depth of 2.5 to 7 cm with an 18–20 G, 9 cm spinal needle. A palpable 'give' in resistance is felt as the subarachnoid space is penetrated. Ten millilitres of CSF can be safely withdrawn.

Lumbosacral (LS) space. Collection from the LS space can be performed in the standing horse. The site of needle insertion is the palpable depression on the dorsal midline just caudal to the sixth lumbar spinous process (between the paired tuber sacrale). This usually coincides with the highest point of the hind quarters. Using aseptic technique, a 15–20 cm, 18 G spinal needle is inserted to a depth of 11–13 cm until a change in resistance is appreciated; the horse often flinches as the subarachnoid space is entered.

CSF analysis

Bacteriological and cytological examinations can be performed. Normal CSF is clear and colourless, and has a nucleated cell count less than 6/μL, of which all are mononuclear cells. There are normally no red blood cells, and the total protein is 0.5–1.0 g/L.

Pink discoloration results from the presence of red blood cells due either to blood contamination during collection or from central nervous system (CNS) trauma. Xanthochromia (yellow discolouration) results from the presence of blood pigments or breakdown products, and is seen as a result of CNS trauma or leakage from damaged vessels (e.g. equine herpesvirus (EHV) 1 vasculitis).

Increased numbers of white blood cells occurs in traumatic, infectious, neoplastic and other diseases. Bacterial infection generally results in a neutrophilic reaction, whereas viral infection results in a mononuclear reaction. Diseases causing tissue damage may result in the appearance of macrophages (some containing phagocytic vacuoles). Parasitic infections can result in an eosinophilic response.

Leakage of blood or plasma into the CSF results in an elevation of protein concentration. This is observed in traumatic, vascular, inflammatory and some degenerative diseases.

Radiology

See Chapters 18 and 25. Diagnostic imaging techniques are increasing and improving; currently, in addition to radiography, computed tomography (CT) and magnetic resonance imaging (MRI) modalities may be useful particularly for examination of intracranial disease. In addition, the availability of these techniques is increasing.

Electrodiagnostics

Needle electromyography (EMG) can be used to detect disruption of the nerve supply to selected muscles. Damage to motor neurons in the ventral grey column or in peripheral nerves causes abnormalities of the electrical characteristics of the affected muscles.

Evaluation of the brainstem auditory evoked response (BAER) can aid in evaluating cranial nerve VIII and auditory pathways along the brainstem.

Electroencephalography (EEG) may be a useful tool for diagnosing focal of diffuse intracranial lesions.

Necropsy

A final diagnosis may be obtained through necropsy; however, this is not always the case. Precautions should be taken to protect people during procedures involving horses with neurological disease lacking

antemortem diagnoses, in particular rabies and Borna virus suspects, both zoonotic diseases. Furthermore, the CNS is heavily affected by post mortem autolysis, which can preclude definitive diagnosis of disease.

The brain

11.2 FOALS

See also Chapter 20.

Anencephaly

In this rare developmental condition, the cerebrum and cranial vault of the skull are absent. The pituitary gland may also be absent. Parturition often requires assistance. Affected foals are not viable.

Hydrocephalus

Hydrocephalus is uncommon (Figure 11.4). It may cause dystocia due to gross enlargement of the cranium. Foals are typically non viable, but mildly affected foals may survive and may be affected with a congenital dummy syndrome with slow learning ability.

Perinatal asphyxia syndrome/hypoxic ischaemic encephalopathy/neonatal encephalopathy/neonatal maladjustment syndrome (dummies, barkers, wanderers)

See Chapter 20.

Congenital cerebellar disease

Cerebellar abiotrophy of Arabs

This condition affects purebred or partbred Arabian foals of either sex. The onset of clinical signs is usually between 1 and 6 months of age, but rarely at birth. It is inherited by a recessive gene. There is a presumed deficiency of a vital trophic substance in cerebellar neurons that results in degeneration of neurons with depletion of Purkinje and granular cells. The cerebellum is usually small (less than 8% of total brain weight).

Clinical signs

- Sudden onset.
- Tremor of the head.
- Intention tremors (nodding movements of the head when trying to carry out a specific manoeuvre e.g. moving towards the mare's udder to suckle).
- Deficit of menace response but normal vision.
- Hypermetric or hypometric ataxia; some foals show a hypometric gait at a walk that becomes hypermetric at faster gaits; there is no weakness.
- Foal may rear and fall over backwards when startled.
- Neurological deficits are exaggerated by turning sharply or blindfolding.
- Ataxia may progress to inability to stand.

Sometimes, the clinical signs stabilize, but they usually progress, and euthanasia is required.

Other cerebellar conditions

A disorder very similar to the Arabian diesase is seen in Gotland ponies. Also, 1–2 month old Oldenberg foals may show signs referable to a rapidly progressive cerebellar disorder. Occasionally, degenerative cerebellar lesions are seen in newly born Thoroughbred foals when the foal begins to walk or at 2–3 days of age.

Idiopathic seizures

Young growing foals, 1–10 months old, can have periods of repeated seizures that usually abate with or without long-term treatment. This may be more frequent in adolescent Arabian foals and possibly reflects a relatively low seizure threshold that allows expression of fits in response to many temporary toxic, infectious, metabolic and physical cerebral perturbations.

Figure 11.4 Hydrocephalus in a non-viable foal. Note the gross enlargement of the cranium.

Residual behavioural changes (dementia) may occur following an episode of anoxia or hypoxia as in anaesthetic accidents, but blindness is more common.

11.3 TRAUMA

CNS injury can be caused by kicks or from running into a tree or fence post, rearing, somersaulting backwards and striking the poll. Impact is usually sustained at the level of the poll or frontal/parietal bones and can lead to open or closed head trauma. In addition temporohyoid arthropathy in which fusion between the temporal and stylohyoid bones occurs can result in skull fractures with resulting damage to the vestibular apparatus and/or cranial nerve VII.

Signs depend on the area of the cranium and brain affected. Syndromes described below may occur independently or concurrently in any combination.

Forebrain syndrome

Trauma to the frontal/parietal area is usually associated with a depressed fracture; however, closed head trauma may also result in direct cortical damage and/or indirect brain damage resulting from elevation of intracranial pressure.

Clinical signs

- Disorders of consciousness varying from normal to comatose.
- Altered behaviour.
- Wandering in circles.
- Seizures.
- Blindness.
- Decreased facial sensation on opposite side of lesion.

Optic nerve syndrome

Clinical signs

- Vision and pupillary light response impairment.
- May progressively deteriorate.

Midbrain syndrome

Clinical signs

- Coma.
- Depression.
- Ataxia and weakness.
- Miosis or mydriasis and decreased pupillary light reflex (but no blindness).

Vestibular syndrome

Seen after poll impact or temporohyoid osteoarthropathy.

Clinical signs

- Peripheral disease (otitis media or temporohyoid osteoarthropathy): head tilt, circling towards the side of the lesion, recumbency, horizontal or rotatory nystagmus with the fast phase away from the side of the lesion, facial nerve (VII) paralysis.
- Central disease: other signs of brainstem involvement, cranial nerve involvement, vertical nystagmus, depression, ataxia and weakness.
- Central lesions may result in paradoxical vestibular syndrome: lesion opposite side than expected; other cranial nerve deficits on the side opposite to the direction of circling.

Hindbrain syndrome

Signs occur due to damage to pons and medulla. Signs of cerebellar injury are rare but may include tremor, absent menace response with normal vision, and paradoxic vestibular syndrome.

Clinical signs

- Cranial nerve dysfunction.
- Depression.
- Ataxia and weakness.
- Respiratory abnormalities.

Treatment of traumatic brain injury

- Seizure control: diazepam at 5 mg for a foal and up to 25 mg for an adult should be repeated as necessary. Alternatives: midazolam, phenobarbital, pentobarbital. In some cases, general anaesthesia is necessary (guaifenesin or barbiturates).
- Anti-inflammatories: NSAIDs and/or corticosteroids.
- Decreasing intracranial pressure: hyperosmolar fluids IV (e.g. 20% mannitol, hypertonic saline (3–7%), perhaps furosemide).
- Anti-oxidative stress: dimethyl sulphoxide (DMSO), vitamin E.
- Antimicrobials may be indicated.
- Nutritional support.
- Surgical decompressive craniotomy is indicated when there are bone fragments penetrating the cerebrum from an open skull fracture.

11.4 TOXIC CONDITIONS

Hepatoencephalopathy

See Chapter 3.

Hepatoencephalopathy may be caused by any form of acute or severe hepatic disease, but is particularly associated with pyrrolizidine alkaloids in *Senecio* and *Crotolaria* spp. In foals, it can be associated with Tyzzer's disease.

Plant poisons

Locoweed and Darling pea toxicity

Locoweed (*Astragulus* and *Oxytropus* spp.) in the USA and Darling pea (*Swainsona* spp.) in Australia can produce a syndrome of dementia and periods of aggression and hyperaesthesia, along with cerebellar ataxia. The alkaloid toxins in these plants induce a lysosomal storage disease (alphamannosidosis) in affected horses.

Removal from exposure to the toxic plants can result in some alleviation of signs, but behavioural changes may be permanent. Reversal of signs with apparent permanent cure has been claimed using combinations of mood elevators (e.g. tranylcypromine and protriptyline) and reserpine.

Mouldy corn poisoning – leucoencephalomalacia

Associated with the ingestion of mouldy corn over a period of about 1 month, leucoencephalomalacia (LEM) develops from the

mycotoxin fumonisin B1 produced by *Fusarium* spp., which alters sphingolipid biosynthesis, thus affecting the subcortical white matter.

Clinical signs

- Sudden onset.
- Dementia.
- Drowsiness.
- Blindness.
- Circling.
- Ataxia.
- Muscle fasciculations over neck and withers.

Signs can be asymmetric, and there may be brainstem involvement with pharyngeal paralysis and other cranial nerve deficits. The horse may also have signs of mild liver disease (e.g. jaundice).

The disease is usually progressive, terminating in coma and death. Many cases are fatal within a few hours to days from the onset of signs. Removal from the source of toxin in mild cases leaves the horse with residual brain damage.

Yellow star thistle poisoning

Nigropallidal encephalomalacia occurs in USA, South America and Australia in horses eating yellow star thistle (*Centaurea solstitialis*) or Russian knapweed (*Centaurea repens*). The toxicity results in necrosis of the substantia nigra and globus pallidus (i.e. nigropallidal encephalomalacia). Horses must eat the weeds for several weeks.

Clinical signs

- Sudden onset.
- Weight loss, depression, yawning.
- Excessive muscle tone (dystonia) of jaws, resulting in a grinding movement of the jaws without the ability to close the mouth completely.
- Inability to prehend food, chew or drink.
- Tongue may be drawn into a longitudinal trough.
- Lips retracted.
- Fasciculations of affected muscles.
- Sometimes circling, aimless wandering or ataxia.

Signs stabilize after several days, but the condition is fatal as there is no known treatment.

Birdsville horse disease

This disease is caused by toxicity of *Indigophera enneaphylla*, which contains alkaloids (i.e. indospicine and canavanine) that are arginine antagonists. It occurs in desert areas of Australia in the spring and summer.

Clinical signs

- Weight loss.
- Progressive ataxia.
- Weakness.
- Dyspnoea.
- Exaggerated hackney-type action in front.
- Reduced flexion with toe-dragging behind.
- Head and tail are held high.
- May gallop frantically on the spot, eventually the hind limbs spreading and sinking as the animal becomes recumbent.
- May progress to convulsions and death.

In mild cases, complete recovery can occur but toe-dragging may persist.

The toxicity can be prevented by feeding a diet rich in arginine such as alfalfa or peanut meal.

Gomen disease

Gomen disease occurs in adult horses in one part of New Caledonia. Horses show progressive incoordination, debility and death. The disease is thought to be environmental in origin and likely to be caused by a toxic plant.

Lead poisoning

Lead poisoning is usually caused by the ingestion of lead acetate found in lead paint, used oil, old batteries, etc. Lead oxide and lead sheeting are less toxic. The other common source is industrial contamination or accidental feeding (e.g. boiled linseed oil). Although chronic exposure is most common, signs can be acute in onset.

Acute form

- Cerebral depression.
- Blindness.
- Partial then complete paralysis.

Subacute form

- Pharyngeal paralysis and dysphagia.
- Regurgitation of food down the nose.
- Choke.
- Aspiration pneumonia.
- Paralysis of lips.
- General muscle weakness.
- Stiff gait.
- Ataxia.

Chronic form

- Recurrent laryngeal nerve paralysis (roaring).
- Ill-thrift.

Treatment

Calcium EDTA (calcium disodium ethylene diamine tetra-acetic acid) 75 mg/kg per day IV, for 3–5 days.

Prognosis

The prognosis is guarded, and poor if pneumonia is present.

Snake bite

In North America, venomous snakes are members of the families Crotalidae (pit vipers), Elapidae and Viperidae (vipers). Rattlesnake bites are most common. Horses are usually bitten on the nose and face, and the principal clinical signs are oedematous swelling and local tissue necrosis. Neurological signs are usually slight.

In Australia, bites from the tiger snake may result in sudden onset of incoordination with dilation of the pupils, and gross muscle tremors. There is apparent foot pain, and the horse behaves as if 'walking on hot bricks', and lies down repeatedly. Tiger snake antivenom works well, but many horses would probably recover anyway. There is usually a good response in 12 hours and complete recovery in 24 hours. Brown snake bite is similar, but paralysis is usually more marked with an inability to swallow and withdraw the tongue. Treatment is by antivenom.

Fluphenazine toxicity

Fluphenazine, a potent phenothiazine, is used in humans for its antipsychotic effects and has been used in horses for its sedative effects.

Figure 11.5 Extrapyramidal signs as a result of fluphenazine toxicity.

Phenothiazines block dopamine, which is the cause of the significant extrapyramidal side-effects reported after use of fluphenazine. Clinical signs include depression, profuse sweating, pawing, striking, agitation, circling, hypermetria, and refusal to walk. Horses may respond to diphenhydramine hydrochloride; however, horses may seriously injure themselves during the course of this toxicosis (Figure 11.5).

11.5 INFECTIOUS CONDITIONS

See also Chapter 19.

Togaviral equine encephalitides: Alphavirus and Flavivirus species

The most important cause of equine encephalitides is neurotropic viral infections caused by the arthropod-borne *Togavirus* species. These include the *Alphavirus* species that cause Venezuelian equine encephalitis (VEE), eastern equine encephalitis (EEE) and western equine encephalitis (WEE), which are found in the Americas, and the *Flavivirus* species that cause Japanese B encephalitis, which occurs in the Far East, and West Nile Virus encephalitis (WNV), which has recently re-emerged in Europe and been introduced to the USA. All of these viruses can cause encephalitis in humans.

A high-titre viraemia occurs with VEE virus in the horse, and epidemics are maintained by a mosquito/horse cycle; infection of humans and other species is incidental. EEE and WEE have been recognized as separate diseases since 1933, and in the USA horses are protected by routine vaccination. Epidemics of these diseases are now uncommon. In contrast to VEE, both EEE and WEE viruses are maintained by a bird/mosquito cycle. The viraemia in the horse is generally considered insufficient to infect mosquito vectors; the horse is a 'dead end host'.

Several species of mosquito can act as vectors of VEE, WEE and EEE. The extension of other arthropod-borne diseases to areas originally outside their geographical distribution (e.g. bluetongue in sheep) serves to illustrate the potential of VEE, WEE and EEE to cause disease on other continents.

Clinically there is progressive onset of severe depression, fever, and peracute to subacute diffuse brain signs. These include dementia, head pressing, ataxia, blindness, circling and seizures. Rarely, signs of spinal cord disease are seen first.

Japanese B encephalitis (JE) and West Nile Virus encephalitis (WNV) are transmitted by mosquitoes, and have birds as the reservoir. JE is a significant problem, but the mortality rate in the horse is usually low (less than 5%). The case fatality rate for WNV has been estimated as 25–28%. The horse is probably a 'dead end host', although mosquitoes have been shown to transmit JE but not WNV between horses.

Clinical signs

Alphavirus encephalitides. These are variable:

1. Mild form.
   - Pyrexia (39.5°C, 103.1°F).
   - Anorexia.
   - Jaundice.
   - Lethargy for 2–3 days.
   - Usually recover in 4–5 days.
2. Moderate form.
   - Pyrexia (38.5–42.0°C, 101.3–107.6°F).
   - Jaundice.

- Petechiation of mucous membranes.
- Somnolence.
- Dysphagia.
- Incoordination and falling over.
- Temporary blindness.
- Rigidity of neck.
- Radial and labial paralysis.
- Usually recover in 5–10 days.

3. Severe form – as above, but with tremors, excitability and sweating. This form is fatal.

West Nile Virus encephalitis

- Ataxia.
- Weakness.
- Recumbency or difficulty rising.
- Muscle fasciculations.
- Pyrexia (39.5°C, 103.1°F).
- Droopy lip.
- Twitching face or muzzle.
- Teeth grinding.
- Hyperaesthesia.
- Blindness.

Diagnosis

Diagnosis is confirmed by serology or by transmission of the disease to a normal horse. An IgM capture ELISA is available for detection of WNV in the acute phase.

Control

Reducing insect vectors (e.g. mosquito control) and vaccination.

Other viral encephalitides

Arboviruses

These include encephalitis associated with Semliki Forest, Near East Equine Encephalitis (NEEE) and louping ill viruses. The clinical signs vary with each virus, but are essentially referable to diffuse cerebral disease. In louping ill, signs of spinal cord disease predominate.

Equine herpesvirus 1 (EHV 1)

See Chapter 19.

Borna disease

Borna disease, which takes its name from a town in Saxony, has been recognized in Germany for over 200 years. Comparatively little is known about the disease, but it is probably transmitted by ticks such as *Hyalomma anatolicum*. It is possible that the virus survives by a tick/bird cycle and infection of the horse is incidental. The incubation period of the disease is at least 1 month, and the encephalitis is indistinguishable from VEE, EEE or WEE. Mortality is 80–90%. A characteristic inclusion, the Hoest–Degen body, may be found in the brain cells of the hippocampus and olfactory lobes of horses that die.

Clinical signs

- Fever.
- Pharyngeal paralysis.
- Hyperaesthesia.
- Lethargy.
- Flaccid paralysis.
- Course of disease 1–3 weeks.

A formalin-inactivated vaccine is available.

Rabies

Epidemiology

Neurotropic rhabdovirus can cause disease in any warm-blooded species. Horses are susceptible, and are usually infected via a bite wound from a rabid wild animal.

Equine rabies has been reported from most countries where the virus is endemic; at present, Great Britain, Ireland, New Zealand, Australia and Hawaii are free from the disease.

The incubation period varies from hours to months (usually 2 to 9 weeks), because the rabies virus can remain latent at the site of wound for varying lengths of time. The incubation period, sequence of clinical signs and survival period vary with the dose and pathogenicity of the virus strain, and the site of bite wound relative to the brain.

Clinical signs

Three forms of disease: furious (cerebral) form, dumb (brainstem) form and spinal cord (paralytic) form. Dumb and spinal cord forms are most common in horses.

The clinical signs associated with the classical forms of rabies are:

Furious form

- Convulsions.
- Aggressiveness.
- Photophobia.
- Hydrophobia.
- Hyperaesthesia.
- Colic.

Dumb form

- Pharyngeal paralysis.
- Drooling.
- Depression.
- Ataxia.

Spinal cord form

- Ascending paralysis.

Clinical signs in individual cases are extremely variable, making the antemortem diagnosis difficult. Most cases show a rapid progression of disease, with death occurring in 4–5 days.

Diagnosis

- Clinical signs.
- CSF analysis – pleocytosis with predominance of lymphocytes (occurs late in the disease).
- Necropsy.
  Histological identification of Negri bodies (intracytoplasmic inclusion bodies) in hippocampus and Purkinje cells of cerebellum.
  Fluorescent antibody test on brain tissue.

Brain abscess

Brain abscesses are very rare and are usually associated with *Streptococcus equi* (strangles) or *Actinobacillus mallei* (glanders) infections.

Cerebral abscesses may be present without clinical signs, or be associated with septic meningitis, or cause signs of an expanding, asymmetric space occupying mass.

Clinical signs

These are variable, including:

- Depression.
- Head-pressing.
- Aimless wandering.

- Excitement.
- Circling.
- Contralateral blindness.
- Decreased facial sensation.

Diagnosis

- History (e.g. strangles).
- Haematology – neutrophilia and hyperfibrinogenaemia.
- CSF analysis – variable; xanthochromia and elevated protein.

Treatment

Treatment is with prolonged antibiotic therapy.

Meningitis

Meningitis may be due to bacterial, viral, protozoal or fungal infection, or rarely as an immune-mediated process. It is principally seen in young foals with poor immune status or secondary to septicaemia/suppurative polyarthritis (see Chapter 20). It may also be associated with a wound near the calvarium or vertebrae in adults.

Clinical signs

- Hyperaesthesia.
- Stiff neck.
- Depression – somnolence, seizures.
- Blindness.
- Pyrexia.
- Anorexia.
- Ataxia/paresis.
- Wandering, star-gazing, barking and seizures in foals.

Diagnosis

- Leucocytosis.
- CSF analysis – large numbers of neutrophils and increased protein concentration.
- Blood and CSF cultures.

Treatment

- Appropriate antibiotics.
- Control of seizures as necessary (e.g. diazepam, pentobarbitone).

Prognosis

The prognosis is poor.

11.6 PARASITIC LESIONS

Verminous encephalitis

Occurrence is sporadic, although endemic in some areas, e.g. Kumri in India due to *Setaria* spp. It is most commonly associated with *Strongylus vulgaris*, *Hypoderma* spp. and *Halocephalobus gingivalis* (*Micronema deletrix*). Clinical signs are extremely variable depending on the number, size and location of the parasites. Progressive forebrain, brainstem or cerebellar signs may occur.

Parasitic cerebral thromboembolism

Occurs mainly in young adults due to *Strongylus vulgaris* thromboarteritis in great vessels leading to embolic shower via carotids to cerebrum. This is rare now that *S. vulgaris* has been virtually eliminated from many regions.

Clinical signs

- Acute onset of seizures.
- Depression.
- Violent or passive behaviour.
- Circling.
- Wandering towards the side of the lesion.
- Blindness.
- Subtle gait abnormality on opposite side of the lesion.

Diagnosis

- Eosinophilia.
- CSF analysis – increased neutrophils, eosinophils, erythrocytes and xanthochromia.

Treatment

- Anthelmintics (larvicidal doses).
- Anti-inflammatory drugs.

Equine protozoal myeloencephalitis (EPM)

See section 11.12. EPM is more commonly a cause of spinal cord disease but occasionally there are cerebral signs alone. This disease can mimic many other neurological diseases of the horse.

11.7 NEOPLASIA

Tumours of the CNS are very rare in the horse. The most common tumour is pituitary adenoma, which usually causes Cushing's disease (see Chapter 9), although it may rarely induce blindness and severe depression.

Cholesterol granulomas are commonly found in the choroid plexuses of old horses, and are usually clinically silent. Large granulomas are sometimes associated with intermittent circling, blindness and depression.

11.8 MISCELLANEOUS CONDITIONS

Epilepsy

Recurrent seizures occur due to a state of excessive and abnormal nervous discharge in the brain. It may be idiopathic or occur secondary to areas of cerebral cortex damage due to ischaemic, traumatic, infectious or neoplastic diseases. The clinical signs and intervals between attacks are highly variable. In a generalized seizure, one may observe three distinct phases: the aura (anxiety, uneasiness prior to onset), ictus (seizure), and postictus (depression, blindness after seizure).

Clinical signs

Clinical signs may include some or all of the following:

- Loss of consciousness.
- Tonic and clonic muscular activity.
- Abnormal movements of head and eyes.
- Jaw clamping.
- Opisthotonos.
- Paddling.
- Urination, defecation.
- Sweating.
- Salivation.

Treatment

Treatment with diphenylhydantoin (phenytoin) or bromide may be effective in idiopathic cases. To manage acute onset of seizure activity, diazepam and/or phenobarbital are best initial treatments.

Narcolepsy

Abnormal sleep tendency characterized by periods of muscle relaxation and collapse (cataplexy). A familial occurrence has been noted in Miniature horses. Similar signs may occur secondary to sleep deprivation and reluctance to lie down, e.g. due to chronic orthopaedic pain.

Clinical signs

- Cataplexy (muscular weakness), which usually affects the head and neck (head hangs low and may rest on the ground); sometimes collapse occurs.
- Attacks last from a few seconds to several minutes.
- Trigger for attack varies between horses.

Diagnosis

- Clinical signs and absence of other diseases.
- Pharmacological testing – physostigmine salicylate IV induces narcoleptic attack in 3 to 10 minutes (this test sometimes gives inconsistent results).

Treatment

Imipramine hydrochloride IM relieves narcolepsy for 5 to 10 hours.

Head shaking (see Chapter 5)

Many possible causes have been suggested for head shaking, including photophobia and trigeminal neuralgia. Horses should undergo a thorough examination (endoscopy, radiography, otoscopy, dental examination) to find a potential cause for this problem. Trigeminal neuritis may be involved in many cases. A trigeminal nerve block should be considered in horses that have no obvious abnormalities in the rest of the examination. Cyproheptadine and carbamazapine have been used as well as management changes that include contact lenses, masks, and nose nets. When the cause is likely to be trigeminal neuritis compression of the infraorbital nerve using platinum embolisation coils that are introduced into the intraorbital canal under fluoroscopic guidance may block pain and end head shaking in some horses.

Temporohyoid osteoarthropathy (see Chapter 5)

Proliferation of the bone of the temporohyoid joint and the proximal stylohyoid bone ultimately leads to fusion of the temporohyoid joint. Fusion of the joint predisposes horses to fracture along the base of the skull, secondary to normal tongue and laryngeal movement. Cranial nerves VII and VII are most commonly involved. Diagnostics include endoscopy of the gutteral pouch and radiology. Prognosis is fair to good for return to athletic activity, although some horses retain cranial nerve deficits or can die acutely. See also the section on brain trauma.

Intracarotid drug injection

Intracarotid drug injections cause intense vasospasm and endothelial damage which may result in cortical ischaemia and necrosis.

Clinical signs are observed immediately on injection of the drug. The horse may rear, or lunge backwards, or run wildly. Some may collapse and become comatose. Some die after a variable period, whereas others recover. Residual CNS or sympathetic trunk damage may cause blindness, Horner's syndrome, head tilt, etc.

The spinal cord

11.9 ATAXIA

Ataxia is the result of reduced kinaesthesia, or lack of sense of motion or position of the body or limbs in space. This results from a loss of proprioception and is frequently accompanied by some degree of weakness (paresis). General proprioceptors (GP), which are sensitive to movement, are located in the internal mass of the body in the muscles, tendons and joints. The pathways for segmental reflex activity and transmitting proprioceptive information principally involve the spinocerebellar tracts. It may be difficult clinically to differentiate proprioceptive deficit from paresis caused by upper motor neuron (UMN) damage. The UMN and GP systems accompany each other through most of the CNS. The UMN is responsible for the initiation of voluntary movements, body support and posture regulation.

Clinical signs

The physical condition of the horse in the acute stages is usually good, and clinical signs are essentially those of proprioceptive deficits. The degree of ataxia can be graded on a scale of 0 to 5:

Grade 0: Deficit not detected.
Grade 1: Deficit not detected at normal gait or posture. Slight deficits noticeable when horse is challenged.
Grade 2: Deficit easily detected and exaggerated by challenging the horse.
Grade 3: Deficit very prominent at normal gait with a tendency to buckle or fall when challenged.
Grade 4: Signs of stumbling, tripping and falling spontaneously at normal gait, and more severe deficits.
Grade 5: Recumbent horse, unable to get up without assistance.

- When standing at rest, the ataxic horse often adopts a base-wide stance. In addition, worn off toes and abrasions around fetlock can be indications of abnormal gait.
- Examination of the neck for stiffness, pain and muscle wastage is usually helpful in cases of trauma and arthritis of the cervical vertebral bodies.
- Usually, some unsteadiness is noted even when in the stable, but no distress is seen nor any awareness of the proprioceptive deficit.
- They seem to be unaware of the position of their limbs and lack smooth coordinated movements (Figure 11.6).
- Signs are usually most noticeable at the slower paces; the horse improves at the canter, but often lacks impulsion from the hindquarters.
- They show difficulty in coming quickly to a halt, and the hindfeet seem to slip or may interfere with the heels of the forefeet. This is usually best demonstrated by observing the horse's gait when turned loose in a small paddock, or on the lunge line.
- In mild cases, signs in the hind limbs are more easily recognized.
- The signs of hind limb ataxia are usually, but not always, symmetrical and can vary in severity from day to day.
- They exhibit exaggerated lifting of the hind limbs, hesitate slightly and then stab the feet back onto the ground.
- Knuckling of the fetlock and stumbling are seen in severe cases.
- At the walk and trot, some swaying of the hindquarters and dragging of the toes are seen.

Figure 11.6 Abnormal foot placement in a horse with ataxia due to Cervical Vertebral Stenotic Myelopathy (CVSM) (proprioceptive deficit).

- There may be involvement of the forelimbs, and this is usually seen as hypermetria (or goosestepping action) due to a prolonged flexor phase of the gait.
- After observing a horse at rest and at a walk, the horse can be challenged to exaggerate gait deficits. Challenges include backing the horse up, turning the horse in wide and tight circles, walking it up and down an incline, elevating the head while walking (up and down an incline), and the sway and tail pull tests.
- On turning in a tight circle, the outer hind limb may circumduct with poor foot placement and a tendency to tread on the other toe (Figure 11.7).
- The horse often backs awkwardly due to a widening of the hindfeet position, dragging of the toes and unsteadiness (Figure 11.8a,b).
- The sway reaction, particularly on the hind limbs, is abnormal, and pressure on the loins or tail pull can cause them to nearly go down.

The clinical background of affected horses is variable, but some diagnostic clues may be obtained from a general comparison of the history, signalment and pathology of the five major causes of spinal cord diseases in horses (see Table 11.3).

Clinical aids to diagnosis

The following clinical aids to diagnosis may be helpful:

1. Radiography:
   - Lateral and oblique views in standing position (C1–T1).
   - Neutral, flexed and extended views under general anaesthesia; also ventrodorsal positions to about C4–C5 level.
   - Myelography – lateral views in neutral, flexed and extended positions.
2. Slap test: for evaluation of laryngeal adductory reflex as confirmation of cervical spinal cord damage.

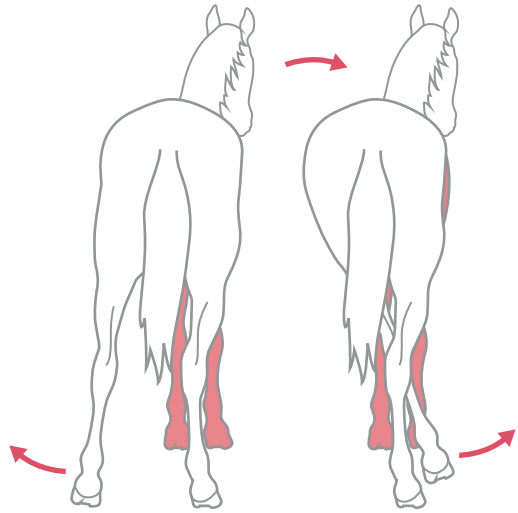


Figure 11.7 Turning horse in a tight circle with a proprioceptive deficit demonstrates circumduction of outer limb and abnormal placement.

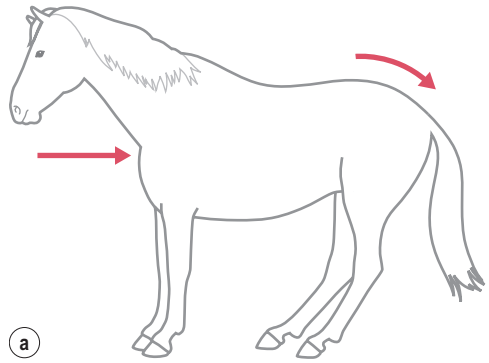


Figure 11.8 (a,b) Proprioceptive deficits. (a) Difficulty with backing is illustrated with lowering of the quarters. In (b) abnormal asymmetric hind limb positioning is shown in a horse with EPM.

Table 11.3 Some features of five major causes of ataxia in horses

	CVSM	EPM	EDM	EHV-1	Trauma
Incidence					
Sporadic	+	+	±	–	+
Age at onset	All ages	6 months–14 years	0–24 months	>24 months	All ages
Sex	Male	Either sex	Either sex	Either sex	Either sex
Breed	Tb	Stb	Arabian	None	None
Onset	Variable	Variable	Variable	Acute	Acute
Clinical signs					
Neck stiffness	±	–	–	–	+
Lameness/pain	–	+	–	–	+
Muscle wasting	±	+	–	–	–
Ataxia symmetric	±	+	–	–	–
Ataxia progressive	±	+	±	–	–
Response to DXMS	±	±*	±	–	+
HL ataxia only	+	+	+	+	+
+ FL involvement	+	+	+	–	±
+ CNS involvement	–	+	±	+	+
Other clinical signs	–	–	–	+	±
Clinical aids					
Radiological changes	+	–	–	–	+
CSF	–	±	–	+	+
Serology	–	–	–	±	–
Pathological lesions	Focal	Diffuse and multifocal	Diffuse	Diffuse Focal	
White matter	+	+	+	+	+
Grey matter	–	+	±	+	±
Site: Cervical	+	+	+	+	±
Site: TL	±	+	+	+	±
Site: Brain	–	+	+	+	±
Clinical improvement likely	±	±	–	+	variable

*Corticosteroid treatment of EPM may exacerbate the disease.

CVSM = cervical vertebral stenotic myelopathy. DXMS = dexamethasone. EPM = equine protozoal myeloencephalitis. EDM = equine degenerative myeloencephalopathy. EHV-1 = equine herpesvirus 1 myeloencephalopathy.

3. Clinical pathology.
   - CSF analysis (from AO or LS sites).
   - Haematology – WBC count and differential.
   - Biochemical profile.
   - Serology.
4. Electromyography: for detection of subtle neurological problems by evaluating the entire motor unit.

Radiological examination of the cervical spine (see also Chapter 25). Plain radiography performed with the horse standing has definite advantages over that carried out under general anaesthesia. A dynamic visualization of the cervical vertebrae is obtained, which makes evaluation of cervical stenosis easier. However, sophisticated equipment involving a powerful generator or overhead mounted tube and linked cassette holder is necessary to achieve this. Low-output portable equipment is probably inadequate for the caudal neck and always requires general anaesthesia. Rare earth intensifying screens and a cross hatch (8:1) grid are helpful in providing good radiographic quality.

A full radiographic survey from C1–T1 in the lateral plane is necessary in all cases because lesions may be present at multiple sites. Ventrodorsal views are possible with the horse placed in dorsal recumbency, although caudal to C4–5, the X-ray quality is usually very poor because of the large amount of soft tissue involved. For myelography, a suitable dose of contrast agent (e.g. metrizamide, iopamidol or iohexol) is injected slowly (i.e. over 2–3 minutes) into the

atlanto-occipital (AO) space. It is important to inject the contrast agent with the head and neck raised to prevent any going forward into the lateral ventricles of the brain. Recovery from anaesthesia may be prolonged, but serious untoward sequelae (e.g. convulsions, or meningitis) are not usually encountered.

Some experience is required in interpreting cervical radiographs because there are individual variations in radiographic anatomy and differences in age, site and neck position. There are also some incidental anomalies that cannot always be differentiated from significant radiological findings. The most important one is the appearance of ventral lipping of new bone with buttressing of the articular processes from C5–T1 that is seen in many normal adult horses as well as young ataxic Thoroughbreds.

Slap test. The presence of a laryngeal adductory reflex can be demonstrated in horses by slapping the saddle region with the palm of the hand. The normal response can be seen using an endoscope, and involves a slight adductory flicker of the contralateral arytenoid cartilage that coincides with each slap. The adductory movement may also be palpated externally as an alternative to using endoscopy.

The degree of adductory movement elicited varies among normal horses. Some horses react to gentle slapping while in others the reflex is only observed after a more vigorous stimulus. For this reason, the optimal strength of the slap administered has to be determined for each individual horse, and varies according to the type and size of the animal. If too soft a slap is used, no response is elicited even in normal horses. If the slap is too forceful, the horse adducts both arytenoid cartilages simultaneously, which is regarded as a fear response.

In tense or excited horses, fixed bilateral abduction of the larynx occurs, and this precludes assessment of the reflex. Some horses relax after the endoscope has been in situ for a few minutes, and, in some instances, removal of the twitch tends to encourage relaxation of the larynx. Chemical restraint does not appear to affect the reflex.

The slap test response is often abolished from the left arytenoid in horses with laryngeal hemiplegia. In horses with cervical spinal cord damage (e.g. compressive lesion of vertebral stenosis), the slap response on both sides is absent. The test provides valuable confirmatory evidence of a cervical spinal cord lesion in horses exhibiting forelimb as well as hind limb incoordination. In those cases where only hind limb ataxia is present, it can help differentiate cervical from thoracolumbar (TL) spinal cord damage. Finally, the slap test has been found to be considerably more reliable as a diagnostic aid than plain radiography.

Clinical pathology. CSF can be collected from both atlanto-occipital (AO) and lumbosacral (LS) sites and should be examined for colour, clarity, cytology (i.e. red and white cell counts) and total protein. Results are usually within normal limits for most conditions apart from mild xanthochromia possibly reflecting some previous haemorrhage. In equine protozoal myeloencephalitis (EPM), an LS tap is more likely to be abnormal and may show a mild increase in white cell count. The CSF of horses with EHV 1 may have raised total protein, white cell count and xanthochromia.

The haemogram and biochemical profile are largely to provide eliminative information; serology may be helpful in infectious diseases such as EHV 1, toxoplasma, and EPM.

Specific conditions of spinal cord disease

The following list of conditions should be considered in horses showing signs of ataxia:

1. Congenital lesions.
   - Occipitoatlantoaxial malformations in Arabians.
   - Vertebral synostosis with meningocele.
   - Cervical vertebral anomalies of development.
2. Trauma.
   - Fracture – vertebral body/neural arch.
   - Dislocation – vertebral subluxation.
   - Haematoma, soft tissue damage and other vascular accidents.
3. Cervical vertebral stenotic myelopathy (CVSM).
   - Cervical vertebral displacement.
   - Medial impingement of articular processes.
   - Functional stenosis of the vertebral canal.
   - Arthropathy of articular processes.
   - Synovial cyst at interneural articulations (C5–T1).
4. Inflammatory conditions.
   - Equine protozoal myeloencephalitis (EPM).
   - Equine herpesvirus 1 vasculitis/myeloencephalopathy.
   - Migrating parasite.
   - Spinal abscess.
   - Vertebral osteomyelitis.
   - Discospondylitis.
5. Degenerative conditions.
   - Equine degenerative myeloencephalopathy (EDM).
   - Fibrocartilaginous embolism.
6. Neoplasia.
   - Glioma, sarcoma, lymphosarcoma.
7. Toxic conditions.
   - Sorghum grass ataxia.
   - Rye grass staggers.

11.10 VERTEBRAL MALFORMATIONS

Occipitoatlantoaxial malformation (OAAM)

This uncommon condition, usually seen in Arabians, is probably inherited. Lesions involve developmental abnormalities of the occipital condyles, atlas and axis, with fusion and hypoplasia of the odontoid process and additional bony pieces.

Foals are either born dead, or are ataxic at birth, or show signs of progressive ataxia in the first few months of life. They may have an extended neck posture with reduced range of flexion of the atlanto-occipital joint. Vertebral abnormalities may be palpable externally. Diagnosis can be confirmed radiographically. There is no treatment.

Cervical vertebral stenotic myelopathy (CVSM)

Clinical features

'Wobbler' syndrome is seen principally in male Thoroughbreds 18–30 months old. The horses are usually well grown and in excellent body condition. The disease is also recognized in older horses that develop osteoarthritis of the cervical vertebral bodies.

Clinical signs

- Onset of signs may be sudden following some traumatic incident or may be insidious.
- Severity of signs ranges from only just detectable (Grade 1) to the horse being unable to stand (Grade 5).
- Signs may be progressive, but often plateau and then vary from day to day.
- Hind limb ataxia always seen.
- Forelimb dysmetria may be seen, especially in young horses.
- Incoordination is usually symmetric, but, when due to a caudal cervical arthropathy, can appear more unilateral.
- Slap test is usually abnormal.

The proprioceptive deficit is caused by one or more sites of compression of the cervical vertebral canal resulting from vertebral malformation or malarticulation. This stenosis may be caused by a static lesion encroaching on the vertebral canal or by dynamic compression when the neck is in flexion or extension. Cervical vertebral stenosis, when mild, causes only hind limb ataxia by interruption of general proprioception. As the degree of compression increases, the signs progress to spasticity and paresis as the deep motor neuron pathways are affected. The hind limb signs are more obvious because the hind limb spinocerebellar tracts in the lateral funiculi are more superficially positioned than those of the forelimb.

The underlying pathogenesis of the condition is unknown, but it is probably multifactorial in origin. A number of different lesions are recognized.

Pathogenesis

1. Cervical vertebral displacement.
   - This is seen radiographically as pronounced dorsal displacement of the vertebral head of C3 to C6 resulting in narrowing at the intervertebral joint space. It is not apparently a traumatic lesion, and it occurs mostly in the younger age group (9–18 months). It may be similar to the cervical instability seen in large breeds of dogs.
2. Abnormalities of vertebral development.
   - Symmetric overgrowth of the cranial articular processes of C3–C5 resulting in vertebral canal stenosis, which is exacerbated during neck flexion.
   - Narrowing of the cranial entrance to the neural canal producing severe stenosis of the vertebral canal. This deficit often accompanies the vertebral displacement lesion.
   - The dorsal prominence of the caudal epiphysis (i.e. 'ski slope' appearance) producing a ventral stenosis of the vertebral canal in some young horses (12–24 months). This is not usually a primary lesion, but is seen in association with the others listed.

These changes lead to intravertebral stenosis of the canal.

3. Functional dynamic stenosis. Narrowing of the normal vertebral canal causes spinal cord compression by flexion of the mid neck (C3–C5) or extension of the caudal neck (C5–T1). This is best seen during myelography.
4. Arthropathy of the interneural articulations. This is associated with asymmetric overgrowth of the articular processes, usually in the caudal cervical spine (C4–C7), and is essentially a static lesion. In young, rapidly growing horses (<24 months) the underlying problem may be associated with osteochondrosis, but older horses (>3 years) have degenerative joint disease whose pathogenesis is obscure. There may be associated damage to the ligamentum flavum and joint capsule, presumably caused by stretching and tearing of these structures. This results in local fibrosis, osteosclerosis of the dorsal lamina and osteophyte formation on the articular facets. Radiographically, there is new bone formation with ventral overgrowth obliterating the intervertebral space. Diagnosis should be confirmed by myelography because the radiographic changes occur quite commonly in normal horses. In this latter group, no ataxia ensues because there is no medial encroachment of the vertebral canal by the asymmetric articular processes. Clinical cases in this category can produce an asymmetric ataxia that is due to more pronounced impingement by the articular process on one side.
5. Synovial cyst. The presence of a small cyst or outpouching of the joint capsule of the interneural articulation on the roof of the vertebral canal can produce dorsal compression of the spinal cord. These lesions usually occur in the older age group in the caudal neck (C5–T1) and are exacerbated by extension. The pathogenesis is equivocal, but is assumed to be due to continued movement or overriding of the articular process leading to distension of the joint, which then protrudes as an outpouching ventrally into the roof of the vertebral canal.

Pathology

The symmetry of the clinical signs reflects damage to the ascending proprioceptive pathways causing ataxia, and the descending upper motor neuron pathways causing paresis. The gross lesions are associated with a flattening of the spinal cord at the site of compression with local haemorrhage and softening due to malacia.

Histologically at the site of compression there is malacia, usually with cavitation, haemorrhage, loss of myelin in all funiculi and variable axonal degeneration. Proximal to the compressive site, there is degeneration of myelin of the ascending tracts of dorsal white and superficial dorsolateral funiculi. There may also be neuronal degeneration in the grey columns with infiltration of gitter cells and proliferation of gemistocytic astrocytes. Caudal to the compressive site, there is loss of myelin and Wallerian degeneration of axons in the descending spinal cord tracts.

Treatment

- Glucocorticoids can provide transient relief but do not solve the underlying problem.
- Rest and neck braces with correction of overfeeding has helped in limiting progression of the disease, and may provide radiographic as well as clinical improvement.
- Surgical treatment. A modified Cloward technique of ventral fusion is successful in selected horses, and is particularly useful in early cases, involving one site, usually from C2 to C6.

Prognosis

The prognosis for athletic use is generally poor. Some horses improve with conservative treatment of rest and dietary manipulation.

Early surgical treatment usually provides an improvement of at least one grade of ataxia.

Other vertebral and spinal cord malformations

Rare cases of block vertebrae (synostosis), spina bifida, cavitated vertebrae and myelodysplasia occur. Congenital kyphosis, scoliosis and torticollis may be associated with arthrogryposis, including the contracted foal syndrome.

11.11 TRAUMA

Cervical vertebral trauma can affect all types, breeds and ages of horses.

The severity of clinical signs of proprioceptive deficits depends on the degree of trauma to the spinal cord and the site of the damage. Signs include sudden onset of ataxia, reluctance to move or recumbency. The signs are not usually progressive, and improvement often occurs unless haemorrhage, joint instability or callus formation is present. Not all cases of vertebral damage result in discernible radiographic changes, but usually the horse has a stiff painful neck with patchy sweating.

The area most frequently affected in young foals is the occipitoatlantal region. Trauma to this area causes mild signs of tetraparesis and recumbency, but the foals usually improve with time. Vertebral fractures with spinal cord involvement carry a very poor prognosis. Radiography is essential for specific diagnosis because an incomplete or

crush fracture of the thoracolumbar spine can cause hind limb ataxia and look clinically very much like a cervical lesion. Conservative treatment includes dexamethasone, dimethyl sulphoxide (DMSO), phenylbutazone or flunixin meglumine.

11.12 INFLAMMATORY CONDITIONS

Equine protozoal myeloencephalitis (EPM)

EPM is caused by *Sarcocystis neurona* infection of the CNS.

The disease is principally seen in North America. It affects horses of all ages, but most commonly horses 1–6 years old. It is non-contagious, but clusters of disease sometimes occur.

Clinical signs

- May be sudden onset of hind limb lameness and ataxia.
- Progressive over days to weeks.
- Weakness and spasticity of limbs.
- Muscle wastage from the quarters.
- Ataxia may involve forelimbs as well as hind limbs.
- Ataxia is often asymmetric.
- Pronounced alteration in the sway reaction.
- On backing, the horse tends to sink back, refusing to move the hind limbs.
- Asymmetric cranial nerve deficits may develop.
- Sensory deficits and focal sweating.

Diagnosis

- Clinical features indicate multifocal lesions with asymmetric signs.
- CSF (LS) may show slight xanthochromia, red blood cells and monocytes/PMNs.
- Antibody test (immunoblot tests, ELISA, indirect fluorescent antibody test) on serum and CSF. Positive serum indicates exposure to the organism, but not necessarily clinical disease. Positive CSF is highly correlated with clinical EPM.
- Positive response to treatment.
- Definitive diagnosis depends on histologic observation of the infectious agent within the CNS.

Pathology

Gross discoloration and softening of the CNS tissue. Lesions are usually multifocal within the spinal cord, brainstem and sometimes the cerebrum and cerebellum. Necrotic areas with haemorrhage are present in severe lesions. Inflammatory lesions consist of prominent lymphoid perivascular cuffing. Organisms are seen singly and in groups (schizonts or merozoites in the cytoplasm of macrophages, neurons, axons, neutrophils and pericytes).

Treatment

Current treatment options include:

- Ponazuril (Marquis) daily by mouth for 28 days. This can be administered at 5 mg/kg (single dose) or 10 mg/kg (double dose).
- Nitazoxanide (Navigator) daily by mouth for 28 days. This drug may be more efficacious; however, requires close monitoring of horse weight and weekly recalculation of the dose because of the narrow safe dose range and potential of adverse side effects. At the time of writing, this drug is not commercially available.
- Pyrimethamine (Daraprim) daily by mouth, and sulfonamide (with or without trimethoprim) by mouth, twice daily for 90–120 days.
- Corticosteroids are contraindicated because they may exacerbate the condition.

Equine herpesvirus 1 vasculitis/ myeloencephalopathy

See Chapter 19.

Usually acute onset, and several horses may be affected. Pyrexia, respiratory disease and abortion may be observed either in the neurologically affected horses or other horses on the premises.

Clinical signs

- Fever.
- Ataxia (most severe in pelvic limbs).
- Tetraplegia/tetraparesis.
- Urinary incontinence and bladder distension.
- Flaccid anus and tail.
- Variable areas of perineal desensitization.
- Stallions and geldings may develop paraphimosis.

The progression of signs is variable. Many cases stabilize rapidly and improve after a few days; others show a gradual deterioration until death. Complete recovery can take up to 18 months.

Diagnosis

- Serology to show a rising antibody titre.
- Antibodies in CSF.
- Virus isolation from CSF/nasopharyngeal swab.
- Post-mortem examination and histopathology.

Treatment

Treatment is mainly supportive. Many horses respond to large doses of corticosteroids. Antiviral drugs (acyclovir) have been used in outbreaks; however, to date, data do not show convincing beneficial results. Supportive care includes provision of adequate bedding for recumbent horses and catheterization to relieve bladder distension.

The pathological changes are principally haemorrhage and malaria. Histologically, there is a diffuse non-suppurative necrotizing leptomeningeal vasculitis with multifocal ischaemia, myelopathy and encephalopathy. The vasculitis with resultant vascular insufficiency causes the infarcts, and areas of ischaemic or haemorrhagic malacia.

Other inflammatory conditions

Migrating helminths occur occasionally in all ages and types of horses. Signs are usually acute and progressive, but this varies according to the movement of the parasite. The signs reflect the tortuous, usually asymmetric, random migrations of the parasites (*Strongylus* spp., *Hypoderma* spp., *Habronema* spp., *Setaria* spp.) or diffuse brain and/or spinal cord involvement (*Setaria* spp., *Halocephalobus gingivalis*). Thus, progressive forebrain, brainstem, cerebellar or spinal cord signs may predominate. Diagnosis may be assisted by circulating eosinophilia or abnormal CSF tap. Treatment consists of antiparasitic and anti-inflammatory medication. Prognosis depends on the damage incurred, but some good recoveries from acute syndromes have been recorded. The gross lesions involve swelling of the site with haemorrhage, red-brown discoloration and soft focal areas of malacia. Histologically, there may be haemorrhagic malacic tracts in acute cases with macrophages and perivascular cuffing of lymphocytes, neutrophils and eosinophils. In older lesions, macrophages, astrocytes and proliferating capillaries predominate.

Spinal abscess is rare. Vertebral osteomyelitis (see Chapter 20) is occasionally seen in foals, and usually involves the caudal cervical/cranial thoracic spine (C6–T3). Signs include sudden onset of pain or stiffness. Progressive paresis occurs. Diagnosis can be confirmed by radiological examination and clinical pathology. There is neutrophilia, left shift, abnormal protein electrophoresis, and raised fibrinogen. Treatment is by appropriate antimicrobial medication, but prognosis is usually poor.

11.13 EQUINE DEGENERATIVE MYELOENCEPHALOPATHY (EDM)

EDM is characterized by degenerative lesions and demyelination of the spinal cord. The clinical picture is very similar to CVS because it causes symmetrical ataxia, weakness and spasticity of the limbs. Can affect most breeds (but especially Arabians). An age range from birth to 24 months is usually seen (most commonly 6–8 months), and there is no sex predilection. More than one horse on a stud or premises may be affected.

The cause of the condition is unknown, but there may be a congenital neuronal metabolic deficit, or a nutritional deficiency (e.g. vitamin E deficiency), or plant poisoning, or familial tendency.

Clinical signs

- Symmetric ataxia: knuckling, stumbling, circumduction of limbs, abduction, interference, etc.
- Spasticity and 'stabbing' with the limbs as they are placed on the ground.
- Inability to turn sharply.
- May be unable to back.
- May fall or stumble with light pressure over the withers or tuber coxae.
- The neurologic deficit is often equal in both fore- and hind limbs.
- Clinical signs are usually slowly progressive.

Diagnosis

- Clinical features.
- Plain and contrast radiography of cervical spine (to rule out CVS).
- CSF analysis is normal.
- Plasma vitamin E may be low.
- Absence of slap test reflex.

Lesions are characterized by neuronal fibre degeneration with loss of myelin throughout the cord, but especially in the dorsolateral and ventromedial funiculi. There is also neuroaxonal degeneration of the proprioceptive relay areas in the cervical and medullary nuclei. The lesions are usually bilaterally symmetrical, but most severe in the thoracic areas.

The signs are irreversible, and there is no effective treatment.

11.14 NEOPLASIA

Spinal cord neoplasms in horses are rare, but lymphoma, haemangiosarcoma, melanoma, undifferentiated sarcoma and glioma have been recorded causing progressive para- or tetraparesis. Signs can begin suddenly and can progress rapidly.

11.15 TOXIC CONDITIONS

Plant poisons (rye grass staggers, Bermuda grass staggers and Dallas grass staggers)

Tremogenic diseases associated with feeding certain fields of ryegrass, Bermuda grass, and Dallas grass. Ryegrass staggers and Dallas grass staggers have been recognized in animals in New Zealand, Australia, USA and Great Britain. Bermuda grass staggers reported mainly in the USA. Dallas grass staggers is associated with ingestion of grass infected with the ergot fungus *Claviceps paspali*. Clinical disease is more commonly observed in cattle and sheep.

Clinical signs

- Stiff, goose-stepping gait.
- Ataxia.
- Tremors of all major muscle groups.
- Intentional head tremor.
- Truncal sway.
- Base-wide stance.
- Collapse.

Affected horses should be removed from the pasture. Most animals gradually recover, but full recovery may take several months.

Sorghum and Sudan grass ataxia

Sorghum grass cystitis and ataxia occurs uncommonly in horses in USA and Australia. Because of toxicosis occasionally associated with sorghum pasture or hay it is generally recommended that horses not be fed forage from the sorghum family.

Clinical features

- Symmetric hind limb incoordination.
- Urinary incontinence.
- Hypoalgesia of perineum.
- Flaccid tail.
- Weakness and stiffness of hindquarters.
- On examination per rectum, the urinary bladder may be distended.

The lesion is due to fibre degeneration throughout the spinal cord with degenerative neuropathy of spinal nerves. Secondary cystitis often occurs.

Signs may improve following removal from affected pastures but in many cases, signs persist.

11.16 EQUINE MOTOR NEURON DISEASE (EMND)

Equine motor neuron disease was first recorded in 1990. The disease is most prevalent in northeastern USA and Canada, but cases have also been recorded in South America, Japan and Europe.

Clinical signs

- Weakness without ataxia.
- Muscle tremors and fasciculations.
- Shortened length of stride.
- Occasional stumbling.
- Abnormal positioning of the feet under the body.
- Excessive periods of recumbency.
- Weight loss despite normal appetite.
- Muscle atrophy.
- Condition is usually progressive necessitating euthanasia, but in some cases, the clinical signs stabilize.

Diagnosis

- Clinical signs.
- May be moderate elevations of CK and AST.
- CSF protein concentration is elevated.
- EMG shows widespread denervation of muscles.
- Ophthalmic examination may show retinal hyperpigmentation.
- Muscle biopsy of the sacrocaudalis dorsalis medialis.
- Biopsy of the ventral branch of the spinal accessory nerve.

Pathology

Motor nerve cell bodies in the ventral horn grey matter of the spinal cord and in some brainstem nuclei become swollen, and there is loss

of Nissl substance. This is followed by cell shrinkage and development of intracytoplasmic inclusions. Damaged nerve cell bodies are eventually phagocytosed by microglia. There is denervation atrophy of muscles with a high percentage of type I fibres.

Aetiology

Aetiology is uncertain. Possibly vitamin E deficiency. Most affected horses are stabled with no access to grass.

Peripheral and autonomic nervous systems

11.17 GENERALIZED CONDITIONS

Botulism

Botulism occurs in adult horses of any type, and onset of signs may be associated with some stress (e.g. racing, foaling, weaning). In foals botulism is not uncommonly seen in the Mid-western and North-eastern USA. Due to the dramatic shivering foals with botulism display, the disease in foals is also referred to as 'shaker foal syndrome'.

Clostridium botulinum produces a neurotoxin that blocks the neuromuscular junction. In adult horses, the toxin is usually preformed and ingested, and the disease is commonly known as forage poisoning. *C. botulinum* grows in neutral or alkaline conditions, and produces toxin in anaerobic environments such as decaying vegetable matter and animal carcases. Silage may provide a suitable medium for toxin production if the organism is present before fermentation reduces the pH to 4.5 or less. Contamination of silage by animal carcases or spoiled big bale silage are recognized sources of toxin. Cases may occur sporadically or as outbreaks. Toxicoinfectious botulism involves the ingestion of spores that germinate and produce toxin within the gastrointestinal tract. The presence of gastric ulcers may predispose to the disease. This form of botulism is the usual cause of the shaker foal syndrome (see Chapter 20). Wound infection by the organism with the subsequent production of toxin is an unusual cause of the disease. The toxin type varies in different areas and different countries, but is usually type B, C or D.

Clinical signs

Clinical signs in adult horses include sudden death or acute onset of flaccid quadriplegia with no history of illness or trauma.

- Violent trembling.
- Progressive weakness with normal sensation.
- Pharyngeal paralysis resulting in dysphagia.
- Drooling saliva.
- Flaccid tongue.
- Flaccid tail.
- Sluggish pupillary light reflexes.
- Recumbency.
- Intercostal and phrenic nerve paralysis may result in dyspnoea.
- Death may arise from respiratory failure or complications of recumbency (e.g. pneumonia).

Diagnosis

- Clinical signs.
- Identification of toxin in serum, gastrointestinal contents or food. In most cases attempts to identify the toxin are unsuccessful.
- Electrophysiologic testing (EMG).

Treatment

- Botulism antitoxin.
- Penicillin (not procaine), metronidazole.
- Neostigmine.
- General nursing care and nutritional support.

Prognosis for adults is good when the onset of signs is gradual, but poor when onset of signs is rapid. Complete recovery in non-fatal cases takes weeks to months. The prognosis for shaker foals is poor if left untreated. A recent report shows a survival rate of 96% in treated foals, however 50% of these required oxygen therapy and 30% of required mechanical ventilation. Vaccination of mares with *C. botulinum* type B toxoid twice during the last trimester is practised in some areas where the shaker foal syndrome is common.

Tetanus ('lockjaw')

Tetanus is caused by neurotoxins produced by *Clostridium tetani*. The organism produces spores and grows in anaerobic sites to produce its toxins. The disease has a worldwide distribution, and *C. tetani* spores are found in the soil, especially soils heavily contaminated by faecal matter.

C. tetani neurotoxin interferes with interneurons in the CNS resulting in hypertonia and hyperreflexia. Affected interneurons are often inhibitory; therefore there is a lack of inhibition and consequently stimulation of lower motor neuron function. Toxin is bound to gangliosides in the CNS, and the effects wear off as these are replaced.

The most frequent sites of infection are soft tissue-injuries (including surgery) and puncture wounds to the foot. Tetanus is also seen in young foals through umbilical cord infection and in mares after foaling. Under anaerobic conditions (especially in the presence of necrotic tissue and pus), *C. tetani* spores germinate into the vegetative form, which produces toxins. At least three toxins are produced. Tetanolysin increases the local tissue necrosis. Tetanospasmin binds to nerves, and is transported to the CNS where it inhibits the action of inhibitory interneurons in the ventral horn of the spinal cord. Non-spasmogenic toxin causes overstimulation of the sympathetic nervous system.

The incubation period varies between 7 days and one month after bacterial inoculation.

Clinical signs

- Sudden onset of stiff gait leading to generalized spasticity.
- There may be signs of colic initially.
- Extended head.
- Limbs placed in 'sawhorse' stance.
- Flared nostrils.
- Ears erect and stiff.
- Lips retracted.
- Prominence of the nictitating membranes.
- Eyeball retracts readily and nictitating membrane 'flicks' when the head is tapped or in response to a loud noise.
- Stiff jaw and firmness over the masseter muscles.
- Elevated tail head.
- Dysphagia and drooling saliva.

All signs are exaggerated with stimuli, especially a hand-clap, or tapping the head. Mildly affected horses may only show a stiff gait. Severely affected horses become recumbent with the head and legs in full extension. Severe tonic muscular activity results in pyrexia and sweating. Spastic paralysis of the respiratory muscles results in hypoxia, and death occurs as a result of hypoxaemia or aspiration pneumonia. Horses frequently die while in a terminal convulsion. Horses that recover usually start to show improvement after 2 weeks, but complete recovery may take 1 month or longer.

Diagnosis

Diagnosis can be difficult to confirm because demonstration of circulating neurotoxin, and isolation of the organism from wounds, is often not possible. Differential diagnosis includes fractured cervical vertebrae, cervical osteomyelitis, colic, pleuritis, laminitis, meningitis and myopathy.

Treatment

1. Sedation and muscle relaxation. Keep the horse in a quiet, dark environment, and undisturbed. Sedate with acetylpromazine, chlorpromazine or promazine at 4–6 hour intervals. Stronger sedation may be achieved, if necessary, using chloral hydrate or sodium pentobarbitone. Diazepam, used alone or in addition to other sedatives, reduces severe muscle spasms. Other muscle relaxants can be used in combination with sedatives, including guaifenesin (given to effect by slow IV drip) and methocarbamol. A balance between the amount of sedation and the degree of muscle relaxation is important. An intravenous catheter may be left in place to minimize stimulation when administering drugs. Packing the ears with cotton wool minimizes auditory stimulation.
2. Provide adequate bedding for recumbent horses to minimize decubital ulcers. Standing horses should have adequate footing. Consider the use of slings to support horses that tolerate them.
3. Eliminate infection by surgical debridement of wounds and parenteral administration of penicillin at double the usually recommended dose.
4. Antitoxin. Administer a large dose of antitoxin (5000–10 000 IU) intravenously at the onset of clinical signs. Local infiltration of any wound with up to 9000 IU of antitoxin has also been suggested. Tetanus toxoid may be administered simultaneously with antitoxin, but at a separate site.
5. Fluid and nutritional support. Intravenous fluid and electrolyte therapy may be necessary. Dysphagic horses may be fed through an indwelling nasogastric tube.

Prognosis

If the horse can still drink, the prognosis is good with nursing care. For recumbent horses, the mortality rate approaches 80%. Horses that survive for more than 7 days have a fair chance of survival.

Recovery from tetanus does not protect against the disease; therefore, the horse must still be vaccinated. Other horses at risk should be vaccinated. Decubital lesions, fractured bones and scoliosis can be lethal complications of the disease.

Organophosphorus poisoning

Organophosphates and carbamates are used as insecticides or may be accidental contaminants in food and water. Overdosing with antiparasitic preparations by owners is also a hazard. The organophosphates include trichlorfon, dermeton, malathion, dichlorvos, ronnel, rulene, parathion and diazinon. The carbamate group of insecticides includes carbaryl.

Both the organophosphorus and carbamate insecticides have their effect and cause clinical signs by binding acetylcholinesterase, thereby permitting continuous cholinergic stimulation and excessive autonomic and muscular activity.

Clinical signs

Effects are manifested within 1 hour of exposure.

- Anxiety and uneasiness.
- Frequent urination and defecation.
- Colic due to increased peristalsis.
- Patchy sweating, particularly on the neck, shoulders and chest.
- Excessive salivation.
- Bradycardia.
- Laboured breathing due to bronchoconstriction, pulmonary oedema and weakness of respiratory muscles and laryngeal paralysis.
- Stiff gait.
- Muscle tremors of the face, neck and other body muscles.
- Hyperactivity of skeletal muscles is followed by weakness, incoordination, ataxia and prostration.
- Respiratory failure occurs in severe toxicity.
- Death may occur within minutes to several hours after the initial signs develop.

Treatment

- Atropine to counteract the parasympathetic signs. Use to effect, with mydriasis and absence of salivation used as indicators of an effective dose. Repeated partial doses given subcutaneously every 2 hours as needed.
- Oximes (e.g. protopam chloride) to bind organophosphorus and release the inhibited acetylcholinesterase. Oximes are not indicated in cases of carbamate poisoning; so, if the precise toxic agent is unknown they should not be used.
- Fluid and electrolyte therapy as required.

Prognosis

The prognosis is quite good providing therapy is rapidly instituted and there is no severe respiratory embarrassment. In cases of oral poisoning, continuing absorption prolongs the clinical signs. In other cases, horses that survive 12 hours after exposure have a good chance of spontaneous recovery.

Lactation tetany and idiopathic hypocalcaemia

This condition is also known as eclampsia and transit tetany.

This is rarely seen today; it used to be more common when draught horses were more popular. It occurs in heavily lactating mares in the first 3 days postpartum on lush pasture, especially at around the foaling heat (i.e. approximately 10 days), or 1–2 days post-weaning; it can also arise in mares during mid gestation and during the last month of gestation. It can be seen in foals, geldings and stallions exposed to stress during transport or in rounding up feral ponies for sale. It can also occur due to alkalaemic conditions, hepatitis, urea poisoning, blister beetle (catharidin) toxicosis and pancreatic atrophy.

Clinical signs

- Rapid violent respirations with thumping sounds from the chest (spasmodic contraction of the diaphragm).
- Sweating.
- Dilation of the nostrils.
- Stiff/stilted gait, hind limb ataxia.
- Muscle fasciculations or tremors of temporal, masseter and triceps muscles.
- Trismus (with no prolapse of the third eyelid nor hypersensitivity to sound).
- Handling and excitement exacerbate the signs.
- Pulse is normal initially, but later becomes rapid and irregular.
- Slight temperature rise.
- May be inability to swallow and salivation.
- After about 24 hours, recumbency and tetanic convulsions.
- Death after about 48 hours.

Diagnosis

The diagnosis can be confirmed by low serum calcium (4–6 mg/100 mL; 1–1.5 mmol/L). There may be a low serum magnesium in transit cases.

Treatment

Calcium borogluconate IV (given to effect) results in a rapid recovery. The heart should be monitored during therapy.

11.18 PERIPHERAL NERVE DAMAGE

Suprascapular paralysis

This is now uncommon (used to be a common condition in draught horses). It is usually caused by kicks or collisions against objects that damage the suprascapular nerve.

Clinical signs

- Lateral subluxation ('popping') of the shoulder when weight bearing.
- Progressive atrophy of infraspinatus and supraspinatus muscles resulting in prominence of the scapular spine ('sweeney').

Diagnosis

The diagnosis can be confirmed by EMG.

Treatment

- Controlled exercise.
- Physiotherapy (e.g. faradism).
- Surgical exploration (if no spontaneous improvement after 3 months) to free the nerve from any entrapment or to perform anastomosis if a neuroma is present.

Radial nerve paralysis

The radial nerve is not commonly damaged alone. It can be involved in humeral or first rib fractures. Lesions near the elbow result in 'high radial nerve paralysis':

- Dropped elbow.
- Failure of limb protraction with toe scuffing.
- Flexion of distal limb joints with foot knuckled over.
- Inability to bear weight on the limb.

Some cases arise due to compression of the brachial plexus and radial nerve roots between the scapula and the ribs. This is then often accompanied by sweeney. Post-recumbency 'radial paralysis' is associated with a post-anaesthetic myopathy and is usually only a transient problem.

Diagnosis

The diagnosis is based on typical clinical signs. EMG is helpful to detect involvement of more than just the radial nerve. Sensory loss over the limb is variable.

Other peripheral nerve damage

The femoral nerve is rarely damaged alone. Clinical signs include profound loss of weight-support, with inability to extend and fix the stifle. There is a sensory deficit on the medial thigh.

Sciatic nerve damage may arise from iatrogenic injection damage or encasement of the nerve by abscesses, especially in foals. The limb is dragged with the stifle dropped and extended, and the foot is constantly knuckled.

Peroneal nerve damage can also arise from injections, but also kicks to the lateral stifle and recumbency. The limb is held slightly caudal with hyperextension of the hock; the fetlock and pastern are flexed, and the limb is jerked caudally when the horse moves.

Tibial nerve injury is uncommon and usually traumatic. The hock is held flexed and the fetlock is knuckled. The leg is raised higher than normal on moving.

Facial paralysis

The facial nerve emerges from the skull through the stylomastoid foramen and travels across the dorsal aspect of the guttural pouch where branches leave to supply the caudal ear muscles (caudal auricular nerve), eyelid muscles and cranial aspect of the ear (auriculopalpebral branch). The remainder of the nerve becomes superficial as it courses across the lateral aspect of the mandibular ramus and the masseter muscle, and divides into dorsal and ventral buccal nerves, which supply the cheeks, nose and lips.

The facial nerve may be damaged centrally or peripherally, and the site of damage determines which muscles are affected. Damage to the upper motor neuron pathways controlling the facial nerve (i.e. supranuclear paralysis) (e.g. cerebral haematoma, EPM and abscesses) can result in abnormal facial expression without paralysis. There is still muscle tone and the facial (V–VII) reflexes remain, but the expression may be bland or grimacing on one or both sides. The EMG of the facial muscles is normal.

Facial nerve lesions proximal to the vertical ramus of the mandible cause proximal facial nerve paralysis, characterized by:

- Deviation of the nostril towards the contralateral side.
- Reduced inspiratory flaring of the ipsilateral nostril.
- Ipsilateral drooping of the lip, eyelid and ear.
- Exposure keratitis.

This can arise from extension of fractures of the petrous temporal bone, otitis interna/media, arthritis of the temporohyoid joint, guttural pouch mycosis, parotid gland neoplasia/abscess, or fracture of the vertical mandibular ramus.

In distal peripheral nerve trauma, usually one or two branches of the nerve, not all three, are involved. Pressure on the side of the face (halter, recumbency) damages the buccal branches, paralysing the nares and lips. Ear droop may occur with direct auricular trauma.

The prognosis for peripheral trauma is quite good, with most cases recovering in 1–10 days. Central facial nerve lesions carry a much poorer prognosis.

Vestibular disease

The vestibular system is a proprioceptive system that functions to maintain the orientation of the horse in its environment. The system helps to maintain the position of the eyes, trunk and limbs in relationship to movements of the head. The receptor organs are contained within the membranous labyrinth in the petrous part of the temporal bone. Impulses from the receptors are transmitted via the vestibular nerve to the vestibular nuclei, which lie adjacent to the facial nucleus in the medulla. There is also a direct afferent connection to the cerebellum.

Signs of vestibular disease vary depending on whether the central or peripheral part of the system is damaged, and whether there is unilateral or bilateral disease.

Clinical signs

- Staggering.
- Leaning.

- Circling.
- Drifting sideways when walking.
- Head tilt.
- Change in eye position (strabismus).
- Nystagmus.

Causes

- Otitis interna.
- Head trauma.
- Temporohyoid osteoarthropathy (often with facial paralysis).
- Idiopathic.

Treatment

Treatment depends on the cause. Prolonged antibiotic and anti-inflammatory drug treatment can be effective in otitis media/interna although residual signs are common in chronic cases. Ceratohyoidectomy can be effective for treatment of temporohyoid osteoarthropathy.

Prognosis

The prognosis is good in otitis interna if early treatment is provided. Prognosis for osteoarthrosis and temporohyoid fractures is poor. Idiopathic cases often recover.

Polyneuritis equi (cauda equina neuritis)

This condition is seen in horses and ponies of many breeds and both sexes, and has a wide age range. In addition to the nerves of the cauda equina, other peripheral nerves are also commonly involved. The trigeminal nerve is frequently affected, but there is no definite pattern of peripheral nerve involvement (other than cauda equina nerves).

Aetiology and pathogenesis

The cause of the polyneuritis is often undetermined but immunological factors are probably important. The original lesions involve neuritis with a component of demyelination. This evolves into a polyneuritis and pachymeningitis involving the sacrocaudal roots (rarely lumbar) of the cauda equina and some cranial nerves, which can extend out to involve peripheral nerves. Sometimes diffuse involvement of many spinal nerve roots is seen. Severe, chronic, granulomatous inflammation occurs, involving lymphocytes, macrophages, eosinophils, fibroblasts, giant cells and neutrophils. Early lesions resemble allergic neuritis. Affected horses have been found to have circulating antibodies directed against P2 myelin protein.

Clinical signs

The onset and progression may be acute and fulminating, or insidious.

Acute form

- Hyperaesthesia around the head and perineum.
- May be head-tilt and ataxia.
- With time, the hyperaesthesia lessens, progressing to hypoaesthesia or anaesthesia.

Insidious form

- Gradual onset of progressive paralysis of tail, urinary bladder and sphincter, rectum and anal sphincter.
- Urinary and faecal incontinence.
- Mares develop urine scald of the perineal area and medial parts of the hind limbs, and the vaginal mucosa may become hyperaemic.

Figure 11.9 Right-sided gluteal muscle atrophy in a horse with polyneuritis equi.

- In males, the penis may be relaxed and protruding.
- Weakness or paralysis of the tail.
- Constipation.
- Anaesthesia of the perineum (and other areas reflecting other affected nerves).
- Ptosis.
- Drooping lips.
- Loss of tongue-tone.
- Drooling.
- Atrophy of gluteal, temporal and masseter muscles (Figure 11.9).
- Weakness and sometimes ataxia of hind limbs.

Diagnosis

- Typical clinical appearance (especially if cranial nerve involvement).
- Lumbosacral (and occasionally cisternal) CSF is often xanthochromic with moderately elevated protein and prominent mononuclear pleocytosis (especially chronic stage).
- Epidural biopsy.
- Serum anti-P2 antibody levels.

Differential diagnosis includes other causes of hind limb ataxia, EHV 1 infection, sorghum grass ataxia and fracture of the sacrum.

Treatment

Treatment involves evacuation of the bladder and rectum, but no specific therapy is available. Antibiotics and corticosteroids are rarely helpful.

Prognosis

The prognosis is poor because the disease gradually progresses.

11.19 PERIPHERAL NEUROPATHIES

Idiopathic laryngeal hemiplegia

See Chapter 5.

Stringhalt

This is a motor disorder of unknown aetiology.

Clinical signs

- Normal at rest.
- Involuntary hyperflexion of the hock when moving.
- Unilateral or bilateral.
- Signs are worse on turning or backing.
- May be worse in cold weather or after rest.

There appear to be three syndromes:

1. Sporadic.
2. Epidemic. Australian stringhalt refers to a syndrome that occurs in outbreaks with involvement, often profound, of both rear legs and often the forelimbs and even the neck. There is an unproven association with dandelion and flatweed intoxication. Muscle atrophy can occur in severe cases.
3. Intoxication with sweet pea plants (lathyrism).

These syndromes may be the result of a sensory neuropathy, a myopathy or spinal cord disease. In lathyrism and Australian stringhalt, there is a Wallerian degeneration of fibres and evidence of neurogenic muscle atrophy in severe cases.

Diagnosis

The diagnosis is based on the clinical signs and ruling out other diseases. Mild forms need to be differentiated from upward fixation of the patella, fibrotic myopathy and other musculoskeletal lamenesses. Occasionally EPM and other spinal cord diseases result in stringhalt.

Treatment

Treatment is to remove the horse from any toxic plant or area. Many cases improve slowly with time. Tenotomy or tenectomy of the lateral digital extensor tendon often helps, even in cases of lathyrism, although this does not cure severely affected horses. For Australian Stringhalt treatment with oral phenytoin (15 mg/kg PO q12hrs) has been helpful in improving clinical signs and getting these cases back into exercise.

Shivering

A condition of mature horses (especially draught breeds) characterized by very mild muscle tremors of the hindquarters and tail occurring with movement. The pathogenesis is unknown, but it probably involves genetic predisposition and peripheral neuromuscular pathology. The disease is usually slowly progressive. No relationship between shivers and polysaccharide storage myopathy has been found, although some horses may have concurrent disease. Although there is no specific treatment for shivers, dietary management such as done for polysaccharide storage myopathy (high fat, low carbohydrate) might benefit horses with shivers.

11.20 NEOPLASIA

Peripheral nerve sheath tumours

Neurofibromas are uncommon. Localized pain in association with nerve or nerve root (sometimes multiple) deficits are recognized when sensory nerves are affected.

Lymphoma

Lymphoma can encompass peripheral nerves.

11.21 CONDITIONS OF THE AUTONOMIC NERVOUS SYSTEM

Horner's syndrome

Horner's syndrome can occur as a sequel to guttural pouch mycosis and surgery (i.e. postganglionic), trauma to the basisphenoid area, deep cervical injuries and perivascular injections, and space occupying lesions in the cervical area and thoracic inlet (i.e. preganglionic). The sympathetic supply for the eye and blood vessels of the head is carried in the cervical sympathetic trunk adjacent to the cervical vagus nerve. These fibres pass up the neck to the cranial cervical ganglion on the wall of the guttural pouch adjacent to the internal carotid artery, and the postganglionic fibres follow other vessels and nerves to all parts of the head.

Clinical signs

- Ipsilateral ptosis of the upper eyelid.
- Ipsilateral enophthalmos.
- Ipsilateral mild miosis.
- Slight protrusion of the ipsilateral nictitating membrane.
- Congestion of nasal and conjunctival membranes.
- Sweating and regional hyperthermia over the ipsilateral side of the face.
- Vision and pupillary light reflexes are normal.

Grass sickness

See Chapter 3.

FURTHER READING

Mayhew IG (2009) Large animal neurology, 2nd edn. Wiley-Blackwell, Oxford, UK

Reed SM, Bayly WM, Sellon DC (2010) Equine internal medicine, 3rd edn. Elsevier, St Louis, MO

Furr MO, Reed SM (2008) Equine neurology. Blackwell Publishing, Ames, IA

APPENDIX

Differential diagnoses of conditions producing neurological signs

1. Differential diagnosis of conditions that produce cortical/basal ganglia signs, i.e. changes in behaviour, mental status, etc.

Hepatoencephalopathy.
Brain abscess/meningitis.
Viral encephalomyelitis.
Rabies.
Parasite migration.
Leucoencephalomalacia.
Trauma.
Neoplasia.
Idiopathic epilepsy.
Narcolepsy.
Lead poisoning.
Fluphenazine toxicosis.
Neonatal maladjustment syndrome.
Intracarotid drug injection.

2. Differential diagnosis of conditions that produce brainstem and cranial nerve dysfunction

EHV-1.
EPM.
Brain abscess.
Neoplasia.
Polyneuritis equi.
Parasite migration.
Botulism.
Lead poisoning.
Vestibular disease.

3. Differential diagnosis of conditions that result in spasticity/tremors

Equine motor neuron disease.
Cerebellar abiotrophy.
Grass staggers.
Locoweed/Swainsonia poisoning.
Botulism.
Tetanus.
West Nile encephalitis – other encephalitides.

4. Differential diagnosis of ataxia/spinal cord disease

A. Spinal cord diseases

Cervical vertebral stenotic myelopathy.
EPM.
EDM.
EHV-1.
Trauma.
Occipitoatlantoaxial malformation.
Vertebral disease – abscess/discospondylitis.
Parasite migration.
Neoplasia.
Grass staggers.
Sorghum grass ataxia.

B. Brain and brainstem lesions

Viral encephalitides (VEE, WEE, EEE, rabies, Borna disease).
Cranial trauma/fracture.
Cerebellar abiotrophy or other disease.
Meningitis.
Cerebrospinal nematodiasis.
Space occupying lesion (tumour, cholesteatoma).
Middle ear infection.

C. Peripheral nervous system disease

Tetanus.
Botulism and shaker foal syndrome.
Myotonia.
Polyneuritis equi/cauda equina neuritis.
Myopathies – rhabdomyolysis, polysaccharide storage myopathy.

D. Systemic disease

Liver damage.
Acute renal disease.

E. Toxicity

Lead.
Monensin.
Plant poisons (yellow star thistle).
Mycotoxins.
Others (OPs).

In addition, many other conditions need to be differentiated from genuine ataxia:

A. Causes of stumbling and toe-dragging

Navicular disease.
Hind limb lameness (e.g. bone spavin).
Upward or partial fixation of the patella.
Hind limb arthrosis (e.g. OCD, degenerative joint disease).
Weakness and debility.
Low back problem.
Scirrhous cord.

B. Bilateral forelimb lameness

Carpal lameness/sore shins.
Navicular disease.
Stiff neck due to soft tissue injury.

C. Poor performance

Gait asymmetry or 'leggedness'.
Fatigue.
Back problem.

Chapter 12

Ophthalmology

Kristopher Hughes

CONTENTS

12.1 Examination of the eye and adnexa 235
Ophthalmic equipment and examination techniques 236
12.2 Techniques in ocular therapeutics 239
Local therapy 239
Systemic medication 242
Surgery 242
12.3 Globe and orbit 243
Congenital abnormalities 243
Acquired abnormalities 243
12.4 Eyelids 244
Congenital disorders 244
Acquired disorders 244
12.5 Conjunctiva, limbus, episclera, sclera 246
Congenital disease 246
Acquired disease 246
12.6 Nasolacrimal system 247
Obstructive disorders 247
Keratoconjunctivitis sicca (KCS) 247
12.7 Cornea 247
Congenital diseases 248
Acquired diseases 248
12.8 Uveal tract 252
Congenital diseases 252
Trauma 253
Iridocyclitis (uveitis) 253
Acute iridocyclitis 254
Chronic/end-stage iridocyclitis 254
Neoplasia 255
12.9 Glaucoma 255
12.10 Lens 256
Cataract 256
Subluxation and luxation of the lens 257
12.11 Vitreous 257
Congenital vitreal anomalies 257
Acquired diseases of the vitreous 257
12.12 Fundus 257
Normal variants of fundus appearance 257
Pathological variants of fundus appearance 258
12.13 Disorders of the optic nerve 259
Optic atrophy 259
Optic neuritis 259
Traumatic optic neuropathy 259
Proliferative optic neuropathy 259
Optic nerve neoplasia 259
12.14 Differential diagnoses for common presenting complaints 259
Further reading 260

12.1 EXAMINATION OF THE EYE AND ADNEXA

Disease of the eye, adnexal tissues and/or orbit is common in Equidae due to several factors, including animal behaviour, responsiveness of ocular tissues to injury, and occasionally as a manifestation of systemic disease. Horses are often presented for veterinary examination due to corneal opacity, abnormal eye position, ocular pain, ocular discharge and abnormalities in vision (see section 12.14 for differential diagnoses for common ocular presenting complaints). In addition, examination of the eyes is an integral component of a 'prior to purchase' examination. Ocular examination is an important component of a physical examination and requires a systematic approach that can be adapted to the individual case.

© 2013 Elsevier Ltd
DOI: 10.1016/B978-0-7020-2801-4.00012-2

History

While diagnosis of some ophthalmic conditions is achieved readily by clinical examination alone, an accurate history is invaluable and should include:

- Presenting complaint.
- Duration of signs and rapidity of onset, i.e. acute, subacute or chronic.
- Progression of signs.
- Evidence of ocular pain: lacrimation, blepharospasm, photophobia and avoidance of examination of the affected eye. Pain is usually associated with a lesion in the anterior segment and is seldom a feature of posterior segment involvement.
- Evidence of vision loss: inability to avoid inanimate objects, change in temperament, reluctance to ambulate freely in reduced lighting and shying. Acute vision loss is often overt; however, gradual and/or partial loss is difficult to assess.
- Known/suspected history of trauma. Trauma may predispose to a variety of conditions including keratitis and uveitis.
- Concurrent/previous ocular disease in cohorts and/or related horses. Breed predisposition occurs for several diseases, including cataracts, glaucoma, uveitis, periocular neoplasia and a variety of congenital disorders. A genetic predisposition may exist for some conditions.
- Previous treatment and response.
- Evidence of systemic disease, e.g. uveitis due to extra-ocular *Rhodococcus equi* or *Streptococcus equi* infection, hyphaema secondary to a coagulopathy and ocular involvement in systemic lymphosarcoma.

Ophthalmic equipment and examination techniques

Thorough ophthalmic examination necessitates examination in both bright light and a darkened room. Bright conditions are used to assess neuro-ophthalmic responses and reflexes, periocular tissues and head for any asymmetry, and to examine the adnexa and anterior segment for any obvious abnormalities. The use of an artificial light source in the dark increases the ability to detect abnormalities via illumination of the structure(s) of interest.

Focal light source

A pen torch may provide some illumination; however, a bright focal light source such as a Finoff transilluminator is superior. Maximal benefit is achieved if various angles and distances between the light source, examiner and the eye are used. The fundic reflection can be used for retroillumination and detection of opacities present in the transparent ocular tissues.

Magnification

A form of magnification is useful alone or in conjunction with a focal light source. Methods include:

- Otoscope head: a simple method that allows a magnified examination of the external structures of the eye and periocular tissues.
- Head loupe.
- Condensing lens: a +20 to +30 dioptre (D) lens used in combination with a focal light source for indirect ophthalmoscopy permits rapid examination of the fundus. The image obtained is inverted and magnified. Monocular indirect ophthalmoscopes provide an erect and mildly magnified image and a greater field of view than do direct ophthalmoscopes; however, expense precludes widespread availability. Indirect ophthalmoscopy allows the fundus to be surveyed rapidly under low magnification, and any lesions identified can be examined in greater detail by direct ophthalmoscopy.
- Direct ophthalmoscopy – close direct ophthalmoscopy – can be used for examination of the adnexa and eye from the cornea to the fundus. The image is real, upright and magnified. Manipulation of the dioptre setting permits focused examination of different ocular tissues without movement of the ophthalmoscope. Distant direct ophthalmoscopy is achieved by selecting 0D, holding the ophthalmoscope at arm's length and examining the tapetal reflection; any opacity present in the light-conducting tissues of the eye will appear as a silhouette.
- Slit lamp biomicroscopy provides superior magnification of external ocular structures, the anterior chamber, iris, lens and anterior vitreous. Determination of the depth of lesions and detection of subtle abnormalities including exudation of protein and/or cells into the anterior chamber are possible. However, equipment is expensive, and experience is required for effective use, limiting the application of this technique in equine practice.

Restraint

Dependent on the temperament of the horse and specific requirements of examination, one or more methods of restraint may be required:

- Nose twitch.
- Topical anaesthesia. Desensitization of the cornea and conjunctiva facilitates examination, collection of tissue/fluid samples and cannulation of the nasolacrimal duct. Proxymetacaine hydrochloride or tetracaine hydrocaine solution can be applied by administration directly from a single use ampoule, by spraying the solution using a 1-mL syringe and hub of a 25-gauge needle with the needle removed or by application of an anaesthetic-soaked sterile cotton bud.
- Intravenous sedation:
  - an α_2-adrenergic agonist; xylazine 0.2–0.5 mg/kg, detomidine; 0.01–0.02 mg/kg, or romifidine 0.04–0.12 mg/kg usually suffices.
- Motor and/or sensory nerve blocks (Figure 12.1).
  - 1–2 mL of prilocaine or mepivacaine is injected via a 25-gauge needle.
  - A palpebral nerve block provides eyelid akinesia.
  - Various sensory nerve blocks provide anaesthesia of the palpebral tissues.

Protocol for ophthalmic examination

A logical approach should be performed using examination in both light and darkened conditions. The sequence of examination and diagnostic tests is important as one procedure may influence the results of subsequent examinations. Both eyes should be examined, and the findings should be compared.

Examination in daylight

Important components of examination in daylight/artificial light are:

- Orbit and globe. The general appearance of the eyes and adnexa and ocular/facial symmetry is assessed, noting any abnormalities in structure and function of the eyelids (Figure 12.2). The orientation of the cilia (eyelashes) on the superior eyelid should be nearly perpendicular to the corneal surface. Palpation of the orbital rim and supraorbital fossae will assist in detection of disruption of the integrity of the orbit and space-occupying

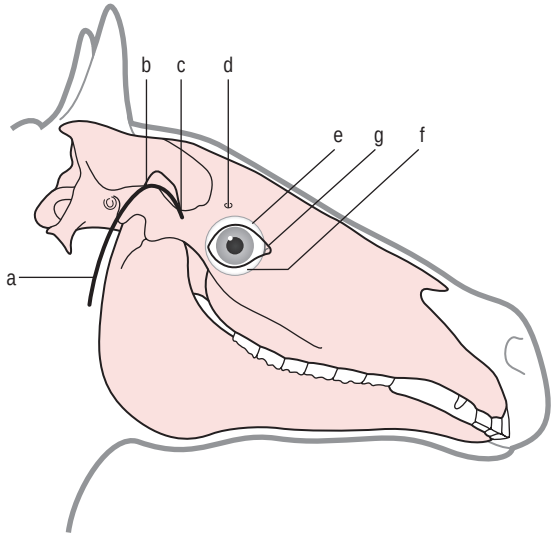


Figure 12.1 Schematic drawing of nerve blocks useful for ophthalmic procedures. Akinesia of the eyelids can be achieved by blockage of the auriculopalpebral nerve (a) or palpebral nerve as it traverses the zygomatic arch (b or c). Densensitization of palpebral tissues can be achieved by blocking the supraorbital (frontal) nerve as it exits the supraorbital foramen (d), the lacrimal nerve (e), the zygomatic nerve (f) and the infratrochlear nerve (g). At each site 1–2 mL of prilocaine or mepivacaine is administered subcutaneously with a 25-gauge needle.

Figure 12.2 Appearance of a normal equine eye and adnexa.

lesions. Eye movement should be assessed bilaterally. In addition, symmetry of pupil size should be assessed because difference in pupil size (anisocoria) reflects a functional (asymmetry of autonomic tone) or structural (e.g. synechia) abnormality.

- Neuro-ophthalmic assessment. Ocular disease, disruption of neural pathways associated with vision and/or intra-ocular/extra-ocular muscle dysfunction may result in loss of ocular reflexes/responses that can facilitate localization of a lesion.
  - Menace response: crude assessment of vision manifested by blinking. An absent menace response may reflect an intra-ocular lesion, damage to visual neural pathways, facial nerve dysfunction, reduced mentation or immaturity (the menace response may not fully develop until 2–3 weeks of age).
  - Dazzle reflex results in retraction of the globe and blinking on response to bright light. The response is subcortical and may help to differentiate cortical blindness from disease of the eye, optic nerve or optic tracts.
  - Palpebral and corneal reflexes. Assess the integrity of the trigeminal nerve (sensory), facial nerve and neural connections in the brainstem. Degree and rapidity of eyelid closure after periocular/corneal stimulation are assessed prior to sedation or regional anaesthesia.
  - Pupillary light reflex (PLR). Assesses the integrity of the retina, subcortical visual pathways, efferent parasympathetic function of the oculomotor nerve (motor) and the iris constrictor muscle. Both the direct and consensual PLR should be assessed (before sedation or use of mydriatics); however, the consensual reflex is weak in the horse. An intact PLR does not indicate vision because the reflex is subcortical.
- Examination of adnexa including the nasolacrimal system. The margins and internal and external surfaces of the superior and inferior eyelids should be examined. The meibomian glands and their openings on the margin of the upper and lower eyelids are visible. Presence and position of the nictitating membrane should be assessed; the palpebral surface can be examined by manual retropulsion of the globe, while examination of the bulbar surface requires application of gentle traction on the free edge with forceps after application of topical anaesthesia. The nasolacrimal drainage apparatus is assessed by confirming the presence of the upper and lower puncta and the nasal ostium. The lacrimal caruncle located at the medial aspect of the palpebral fissue is prominent in the horse.
- Ocular surface. Examination of the cornea, conjunctiva and limbus can be performed at this stage to detect alterations in structure. The degree of pigmentation of the conjunctiva is variable.

Examination in a darkened area

Once an initial examination in daylight has been completed, a more detailed examination can be performed in a darkened environment such as a stable.

- Examination of the cornea by use of focal illumination with/without magnification. Slit-lamp biomicroscopy permits increased acuity of examination, detection of subtle lesions and determination of lesion depth. The normal cornea is translucent, horizontally elongated and slightly wider medially than laterally and devoid of vasculature and pigmentation. The pectinate ligament is appreciated readily as a grey line at the medial (lacrimal) and lateral (temporal) limbus and should not be confused with corneal oedema or cellular infiltration (Figure 12.2).
- Examination of the anterior chamber. The depth and clarity of the anterior chamber should be assessed by shining a slit beam of light across the chamber from medial to lateral. Abnormal depth may reflect a change in size or position of the lens or volume of aqueous humor. Opacities indicate the presence of protein (aqueous flare) and/or cells. The ventral aspect of the chamber should be examined for sedimentation of leukocytes (hypopyon) or erythrocytes (hyphaema).
- Examination of the iris. The irises of most horses are heavily pigmented; however, pale brown, grey, gold, blue or white coloration may occur and multiple colours may occur within (heterochromia iridis) or between irises. The division between the ciliary and papillary zones of the iris (collarette) is not

always distinct, particularly if the iris is heavily pigmented. True albinism is rare; the white/pink iris is frequently very thin and may allow transillumination of the underlying lens equator and ciliary body. The corpora nigra (granula iridica) are heavily pigmented uveal bodies prominent on the dorsal papillary margin and less developed ventrally. The pupil in adults is horizontally ellipse in shape, slightly wider medially, while in foals the pupil is ovoid. Remnants of the embryonic vasculature (persistent pupillary membranes) emanate from the collarette and are common. The iridiocorneal angle can be visualized nasally and temporally.

- Examination of the lens. Thorough examination of the lens requires mydriasis achieved by topical application of 1% tropicamide. The lens is examined for abnormalities in clarity, size and position. The normal lens is optically clear; however, in older horses it is yellow, and nuclear sclerosis may be present. Opacities should be described according to size, location and density. Some lens opacities are normal variations (e.g. point of attachment of the hyaloid artery to the posterior capsule (Mitterdorf's dot), suture lines on the anterior and posterior surfaces and refractile concentric 'onion rings'). Cataracts are abnormal; however, the degree of vision impairment and likelihood of progression is heavily dependent on cataract type and aetiology. The equator of the lens is not normally visible, and detection indicates lens luxation/subluxation, microphakia (congenital hypoplasia of the lens) or lens coloboma.
- Examination of the posterior segment: examination is facilitated by mydriasis and distant and close techniques of direct ophthalmoscopy (vitreous body and fundus), focal light source (vitreous body and fundus), slit-lamp biomicroscopy (anterior vitreous body) and indirect ophthalmoscopy (fundus). The vitreous body is optically clear. The equine fundus comprises a tapetal zone (tapetum lucidum) located dorsal to the optic papilla and a non-tapetal zone. The tapetum is roughly triangular, reflective and often blue/green/yellow in colour. The non-tapetal fundus contains the optic disc and is often heavily pigmented. The optic disc is horizontally ovoid in shape, salmon pink in colour and approximately 40–60 retinal blood vessels radiate from the periphery and extend 1.5–2 disc diameters into the fundus (Figure 12.3). Despite these generalizations, considerable variability exists in the appearance of normal fundus, and differentiating between a normal variant and pathological change can be challenging.

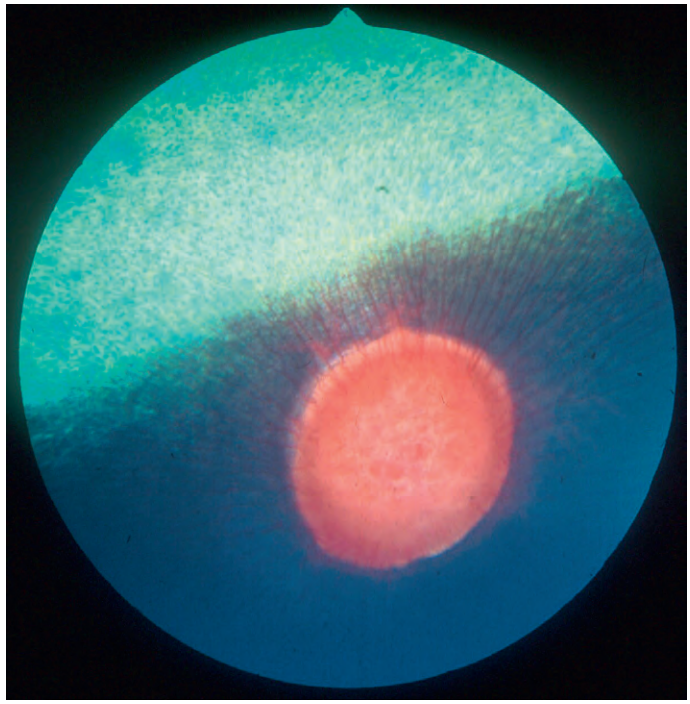


Figure 12.3 Direct ophthalmoscopic appearance of the equine fundus. The distinction between the tapetal and non-tapetal (pigmented) fundus is distinct. The optic disc is located in the non-tapetal fundus, and numerous retinal blood vessels are evident crossing the margin of the disc and radiating into the fundus. Picture courtesy of Dr John Mould.

Ancillary diagnostic techniques

- Schirmer tear test (STT). Inadequate tear film production is rare in horses; however, disorders of the preocular tear film (PTF) should be considered when corneal disease of undefined aetiology is present. The Schirmer I tear test is used to assess the aqueous component of the PTF. Stimulated (reflex) tear formation is assessed, and thus the test must be performed prior to application of topical local anaesthetics. A commercially available test strip is bent at the marker notch, and the tip is placed within the conjunctival sac to the level of the bend. The aqueous fraction of the PTF 'wicks' along the strip, and the length of wicking from the notch is measured immediately after removal; values of 20–30 mm/min are expected from normal horses while repeatable values of <10 mm/min are consistent with inadequate tear production.
- Collection of samples for cytology and microbiology. Samples can be collected from the eyelids, conjunctivae, 3rd eyelid, cornea and adnexal tissues. The method of sample collection is dependent on the lesion.
  - Tissue swabbing and scraping. The conjunctivae and palpebral margins can be sampled without local anaesthesia; however, sampling of the cornea is facilitated by topical anaesthesia. Corneal ulcers can be sampled with a moistened swab or by scraping the ulcer with a Kimura spatula or a scalpel blade handle. Bacteria are most likely to be sampled from the periphery of a lesion, while collection of corneal tissue by deep scraping or partial keratectomy is recommended if mycotic keratitis is suspected. Cytological examination of samples obtained from ulcerative keratitis may reveal leukocytes, and microorganisms, and microbiological culture may identify the causative organism(s). Isolation of microorganisms is not affected by prior use of topical anaesthesia.
  - Impression smears can by made from lesions of the adnexa by pressing a clean slide directly onto the site of interest. Squamous cell carcinomas and, rarely, other neoplasms, can be diagnosed in this manner.
  - Fine-needle aspiration of space-occupying lesions involving the external globe and adnexa may harvest cellular tissue suitable for cytological examination. Material aspirated should be transferred immediately to a clean slide.
  - Collection of corneal biopsy samples by sharp dissection, preferably during general anaesthesia, for cytology, microbiology and histopathology. Corneal biopsy is best undertaken where suitable facilities and expertise are available.
- Conjunctival/palpebral biopsies may be obtained by incisional or excisional techniques. Samples are most appropriate for histopathology, however, they may be submitted for microbiology also. Conjunctival tissue can be collected by sharp

dissection after provision of topical anaesthesia. Palpebral lesions can be sampled following local/topical anaesthesia or during general anaesthesia.

- Ophthalmic stains:
  - Fluorescein sodium. This orange dye changes to green in the alkaline environment of the PTF and is used to assess the integrity of the corneal epithelium. The dye does not stain epithelium or Descemet's membrane (hydrophobic); however, it is rapidly absorbed by exposed stroma (hydrophilic) facilitating diagnosis of ulcerative keratopathies. After application of the dye, the corneal surface should be lavaged with sterile, isotonic saline solution to disseminate the stain and avoid erroneous diagnosis of corneal ulceration. Weak staining, as occurs with corneal microerosions, is best appreciated with cobalt-blue light. Fluorescein can also be used to check for leakage of aqueous humor from the anterior chamber (Seidel test); after application of the dye the corneal surface is examined for the presence of a clear/light green rivulet within the yellow-green fluorescein.
- Rose Bengal is a red dye used to assess the integrity of the mucin layer of the PTF, as retention of stain indicates staining of epithelial cells in the absence of a mucin covering. In addition, Rose Bengal stains devitalized corneal and conjunctival cells. The dye is an irritant and should be reserved for the detection of subtle defects, including superficial punctate epithelial lesions.
- Tonometry. Measurement of intraocular pressure (IOP) is achieved most reliably with a portable tonometer (e.g. Tonopen® XL, Tonovet®). Abolishment of palpebral tone appears to be unnecessary; however, application of topical anaesthesia is required to ensure accurate readings. Sedation may reduce IOP, while a lowered head position may increase IOP. The normal IOP in horses is 23.3 ± 6.9 mmHg (range 7–37).
- Nasolacrimal drainage assessment. The upper and lower lacrimal puncta and nasal ostium can be located and inspected for size, position and discharge. Patency can be tested most simply by applying fluorescein stain to the eye; drainage of green stain should be evident at the nasal ostium within 5 minutes. If samples of nasolacrimal discharge are required for bacteriology/cytology or no patency is evident with fluorescein stain, a puncta or the nasal ostium can be cannulated and irrigated after applying local anaesthesia locally.
- Diagnostic imaging:
  - Radiology. Osseous lesions of the orbit and surrounding skull may be identified radiographically; lesions may include fractures, osteomyelitis and neoplasia. Contrast radiography of the nasolacrimal duct (dacrocystorhinography) is useful for assessment of congenital and acquired disorders; the upper lacrimal punctum is cannulated, 5–10 mL of an iodine-based contrast agent is injected, and lateral and oblique radiographs of the head are obtained.
  - Ultrasonography is a useful, non-invasive technique for the examination of the globe, adnexa and orbit. Superior image resolution is achieved by use of a high-frequency (7–16 MHz) linear transducer. Indications include any condition that impedes direct examination of part or the entire globe (e.g. blephoedema, diffuse corneal disease, uveitis, cataracts or intra-ocular masses), determination of globe size, assessment of the peri-ocular and retrobulbar soft tissues and examination of the orbit. Identifiable lesions include: structural consequences of uveitis, uveal cysts, cataracts, lens luxation or rupture, vitreal opacities, retinal detachment, enophthalmitis, abnormalities in globe size/shape, intraocular/periocular/retrobulbar masses and orbital fractures.
  - CT/MRI. Advanced diagnostic imaging techniques are not used commonly for examination of the eye and adnexa; however, they may permit detection of periocular/retrobulbar masses and osseous lesions.
  - Nuclear scintigraphy may detect active disease of the bony orbit.

12.2 TECHNIQUES IN OCULAR THERAPEUTICS

Treatment of ocular disease in the horse presents several challenges, principally anatomical and physiological in nature. Barriers to the corneal penetration by topically administered drugs are the PTF, lipid-dense hydrophobic corneal epithelium and the hydrophilic stroma. Loss of one or more of these barriers may facilitate uptake of topically applied medications and enhance efficacy of treatment. Intraocular distribution of systemically administered pharmacological agents is influenced by the vascular supply to the eye, the avascularity of the majority of the paurangiotic equine fundus, cornea, lens and vitreous body, and an almost complete blood–ocular barrier in the uveal tract. Selection of medication, route of administration and dosage are influenced by lesion aetiology and location. In general, local administration (topical, subconjunctival, intraocular and intra/perilesional) is used for conjunctival and anterior segment disease, while systemic administration is used for diseases of the eyelid, conjunctiva, sclera, uveal tract, posterior segment and orbit.

Local therapy

Topical medication

Topical administration of medications is the most common and important method in equine ophthalmology (Table 12.1). However, effective use is influenced by:

- Corneal/scleral anatomical barriers. Less important for ulcerated cornea or conjunctival disease; however, deep corneal disease (e.g. corneal stromal abscess) and intraocular disease require selection of agents that are able to penetrate the cornea effectively.
- Residence time. The majority of aqueous solutions are removed rapidly from the ocular surface by reflex tearing and nasolacrimal drainage. The latter may result in systemic absorption and is of most concern with atropine due to gastrointestinal side-effects.
- Frequency of administration. Provided the medication has low tissue toxicity or side-effects, frequent treatment is recommended to maintain therapeutic concentrations.
- Volume. Due to reflex tearing and the vertical orientation of the equine cornea, increasing the volume of topical solutions does not improve treatment efficacy, and small volumes (0.1–0.2 mL) are adequate.
- Drug vehicle. The formulation influences retention time and available methods of administration. Drugs may be available in solution, suspension or ointment. Ointments generally have the longest retention time and are easiest to instil; however, not all ophthalmic drugs are available in this form.
- Drug concentration. The use of fortified solutions can help to maintain therapeutic concentrations at the target site.

Techniques for topical application

- Solutions/suspensions can be administered easily via a 1-mL syringe with the hub of a 25-gauge needle (with the needle

Table 12.1 Topically administered medications used commonly in Equidae with ocular disease

Category/drug	Dosage	Comments
Antibiotics		
Neomycin /polymyxin B/ bacitracin (or gramicidin)	Solution/ointment; q2–6 hr	Broad-spectrum bacteriocidal. Use for superficial ulcers.
Gentamicin	Solution/ointment; q2–6 hr	Bacteriocidal; G– spectrum. *Staphylococcus* spp.
Tobramycin	0.3% solution; q2–6 hr	Bacteriocidal; G– spectrum, use reserved for severe infections e.g. with *Pseudomonas aeruginosa*
Chloramphenicol	0.5–1% solution/ointment; q2–6 hr	Bacteriostatic; G+ spectrum
Ciprofloxacin	0.3% solution; q2–6 hr	Bacteriocidal; G– spectrum, use reserved for severe infections by sensitive organisms
Antifungals		
Natamycin	5% solution; q1–8 hr	Limited corneal penetration. Good efficacy against filamentous fungi
Amphotericin B	0.15% solution in 5% glucose q2–6 hr	Irritant to cornea. Most active against yeasts
Itraconazole + DMSO	1% with 30% DMSO. q4–6 hr	Excellent corneal penetration. Requires compounding.
Fluconazole	1% solution; q4–8 hr	Fungal isolates from equine keratomycosis often demonstrate poor susceptibility
Miconazole	1% solution; q2–6 hr, 2% cream; q4–6 hr	Good corneal penetration, broad-spectrum and non-irritant. Solution only available from compounding pharmacies. Use vaginal creams only; dermatological creams contain ethyl alcohol that is a corneal irritant
Silver sulfadiazine	1% cream; q4–6 hr	Broad-spectrum
Voriconazole	1% solution; q4–6 hr	Excellent corneal penetration and broad spectrum of fungicidal activity against filamentous fungi
Anticollagenolytics		
Potassium EDTA	0.05–1.0% solution; q2–4 hr	Add 1–5 mL of sterile water to a commercial blood collection tube. Potent inhibitor of MMPs
Endogenous serum/ plasma	q2–4 hr	Contains several α-globulins that inhibit MMPs and serine proteases. Samples should be kept refrigerated and sterile and replaced every 5 days.
Tetanus anti-toxin	q2–4 hr	Commercial source of α-globulins
Doxycycline	0.1–0.3%; q4–6 hr	Inhibitor of MMP function. Must be compounded.
Acetylcysteine	5–10% solution; q1–4 hr	Inhibitor of MMP function. Mucolytic action may compromise the tear film with prolonged use.
Mydriatics		
Atropine	1% solution; to effect	Potent mydriatic and cycloplegic. Frequent use may predispose to reduced faecal output and/or colic.
Tropicamide	1% solution; once	Short-acting mydriatic used to facilitate examination of lens and posterior segment
Phenylephrine HCl	10% solution	May be combined with atropine to improve mydriasis in refractory cases
Anti-inflammatories		
Prednisolone acetate	1% solution; q1–6 hr	Potent corticosteroid with excellent ocular penetration. Immunosuppressive and delays healing.
Dexamethasone HCl	0.05–0.1% solution; q1–6 hr	Potent corticosteroid with excellent ocular penetration. Immunosuppressive and delays healing.
Flurbiprofen	0.03% solution; q1–6 hr	NSAID, good ocular penetration. Delays corneal epithelialization
Cyclosporine A	0.2% ointment; q6–12 hr	Potent inhibitor of T-cell function. Poor corneal penetration and weak direct anti-inflammatory effect.
Suprofen	1% solution; q4–6 hr	NSAID. Good ocular penetration. Low concentrations do not affect epithelial migration.

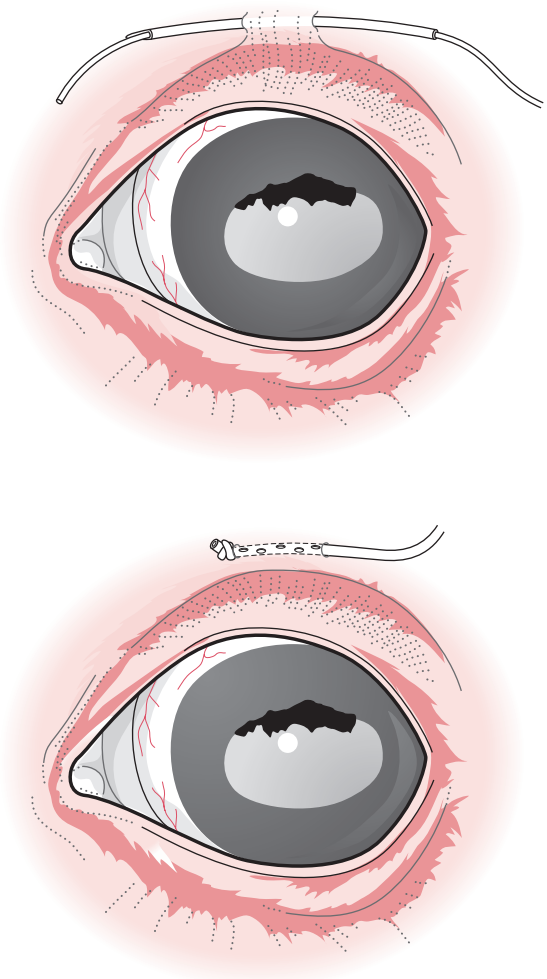


Figure 12.4 Placement of narrow-bore silastic tubing into the superior fornix as a sub-palpebral lavage apparatus. Holes are placed in the tubing prior to placement to correspond to the sub-palpebral segment. After placement of the tube using a metal trochar, one end is tied, while the other end is attached to the mane and used to administer medications.

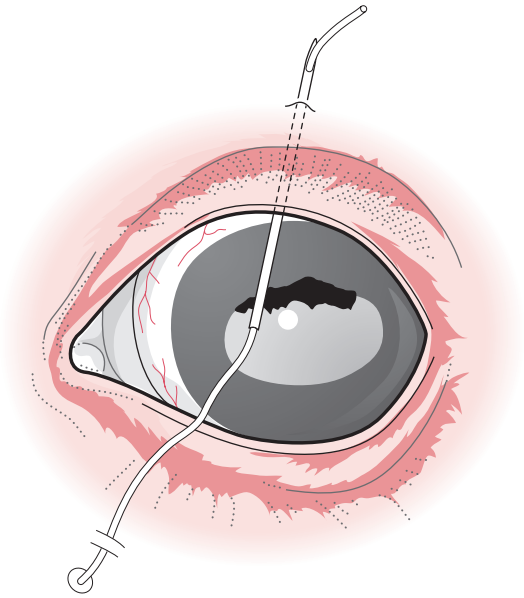


Figure 12.5 Placement of a commercially available subpalpebral lavage tube. A metal trochar is used to provide an exit point for the tube in the superior fornix. The tube is pulled through until the end plate is located in the fornix adjacent to the bulbar conjunctiva. The free end of the tube is located at the level of the horse's neck and is used to administer medications.

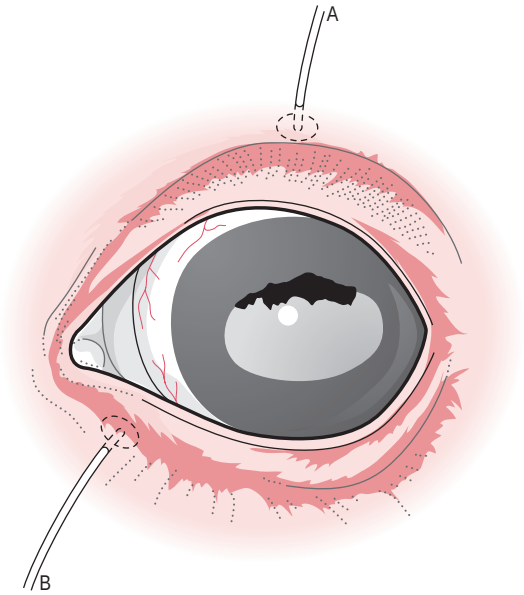


Figure 12.6 Commercially available subpalpebral lavage devices can be placed in the superior (A) or inferior (B) fornix.

removed); the syringe is steadied by placing the hand on the face of the horse, and the contents are sprayed gently onto the cornea.

- Ointments are instilled into the ventral palpebral fornix.
- Subpalpebral lavage; commercial (e.g. Mila International) or home-made lavage systems (silastic tubing) provide an effective method of administration of solutions/suspensions, particularly when frequent treatment is required. Placement can be facilitated by sedation of the horse and the use of local motor and sensory anaesthesia or under general anaesthesia. The tubing is placed in the superior or inferior palpebral fornix by use of a stainless steel trocar, ensuring that the tubing does not lie on the cornea (Figures 12.4–12.6). The tubing is attached by plastic guides or glued to the temporal region and then threaded through a series of plaits in the horse's mane. The free end of the tube should be fitted with a Luer adapter and an injection port and fixed to a plaited piece of mane. The free end should extend to the level of the withers to ensure safety of personnel, as horses with painful ocular disease can be refractory to treatment and exhibit avoidance responses.
- Nasolacrimal lavage using a commercially available nasolacrimal lavage catheter or a canine urinary catheter placed via the nasal ostium and maintained in the nasolacrimal duct. The catheter may be passed through a surgical opening in the false nostril and secured to the adjacent skin. Nasal irritation and traumatic removal of the catheter by the horse and retrograde flow of inflammatory debris onto the cornea are limitations.

- Continuous perfusion devices are available and may eliminate the need for frequent manual drug administration. However poly-pharmacy requires drug compatibility to avoid precipitation of drugs and to preserve treatment efficacy.
- Contact lenses; therapeutic contact lenses impregnated with medication have received limited attention in horses, however may provide high topical drug concentrations.
- Other recent advances in topical drug delivery (e.g. high-viscosity gels, degradable and non-degradable inserts and collagen shields) have yet to be assessed in horses.

Subconjunctival injection

Solutions and suspensions can be injected under the temporal or dorsal bulbar conjunctiva (Table 12.2). Horses should be sedated and an auriculopalpebral nerve block performed. A cotton bud soaked in local anaesthetic can be applied to the conjunctiva prior to injection. A 25 gauge needle should be used and no more than 1 mL injected. The drug vehicle will influence rapidity and duration of effect; solutions provide rapid and high concentrations in the ocular tissues, while suspensions/emulsions are less potent but have a longer duration of effect. Antibiotics, corticosteroids and atropine can be injected. Solutions for topical use should not be injected; formulations for parenteral use are most appropriate. Occasionally oily solutions can induce granulomatous inflammation.

Subconjunctival micro-osmotic pumps

Recently, implantation of a micro-osmotic pump subconjunctivally was found to be an effective method of administration of atropine in horses. This technique has the potential for continuous administration of medications for 7 days. Limitations are the limited volume capacity, requirement for fortified medications and restriction in the number of medications that can be administered to individual horses.

Table 12.2 Concentrations and doses of medications that can be administered topically as 'fortified' solutions and by subconjunctival injection (volume injected should be <1 mL)

Medication	Concentration for topical use	Dosage for subconjunctival injection
Cephalothin	65 mg/mL	
Cephazolin	33–50 mg/mL	100 mg
Ticarcillin	6 mg/mL	100 mg
Penicillin G	1×10^6 IU/mL	0.5×10^6 IU/mL
Gentamicin	10–15 mg/mL	20 mg
Amikacin	6.7–15 mg/mL	25–75 mg
Tobramycin	15 mg/mL	20–40 mg
Chloramphenicol	5–10 mg/mL	50–100 mg
Atropine		1–2 mg
Triamcinolone acetate		1–2 mg q7–10 days
Methylprednisolone acetate		20 mg q3–4 days

Intracameral injection

This procedure is performed rarely; indications are limited and include anti-microbial treatment for endophthalmitis and treatment for fibrinous adhesions in the aqueous humor with recombinant tissue plasminogen activator. General anaesthesia and use of a muscle-relaxing agent are recommended.

Intralesional medication

Peri-ocular tumours can be injected directly with immunomodulating agents (Bacillus Calmette Guérin [BCG]), radioactive implants and cytotoxic drugs (cisplatin, 5-fluorouracil). Squamous cell carcinoma (SCC) and sarcoid are the most common periocular neoplasms and often respond well to intralesional medication, with preservation of anatomical and functional integrity.

Intravitreal cyclosporine A implant

An intravitreal sustained delivery device containing the immunosuppressive agent cyclosporine A is a safe and effective treatment for chronic recurrent uveitis in horses; continuous delivery of cyclosporine A is provided over a protracted period. Recently, suprachoroidal implantation has been reported and may be associated with fewer complications than the intravitreal technique.

Systemic medication

Anti-inflammatory/analgesic medications are important for treatment of many diseases of the eye, adnexa and periocular/retrobulbar tissues as pain and inflammation are common features. Corticosteroids are useful when immune-mediated mechanisms are involved (e.g. uveitis) or inflammation is severe. Systemic corticosteroid administration has been associated with development of laminitis and should be avoided in horses with evidence of this disease. The NSAIDs are effective analgesics and anti-inflammatory agents. Flunixin meglumine affords greater analgesia than other NSAIDs; however, phenylbutazone and suxibuzone are useful when medium-grade analgesia is required. Occasionally, systemically administered antimicrobial agents are indicated for some conditions, including infections of the adnexa and periocular/retrobulbar tissues, infectious endophthalmitis/panophthalmitis and where vascularization of infected cornea exists.

Surgery

Numerous surgical techniques have been described for ocular and periocular disease. Several require specialized equipment and considerable expertise (e.g. phacoemulsification of cataracts and corneal transplantation). Those that can be performed without specialized equipment include:

1. Repair of eyelid lacerations: minimal debridement of wound margins should be performed to preserve the anatomy and function of the eyelids. The eyelids have an excellent blood supply, and even tissue of questionable viability may have the capacity for healing.
2. Nictitans flap. Superficial corneal ulcers and conjunctival grafts can be protected by attaching the 3rd eyelid to the upper eyelid by the placement of 2–4 horizontal mattress sutures. However, the corneal surface is obscured preventing monitoring of the lesion, and the flap does not provide a direct vascular supply to the cornea.
3. Tarsorrhaphy. Surgical apposition of the eyelids can be performed by the use of horizontal mattress sutures placed through the eyelid margin. Indications and limitations are

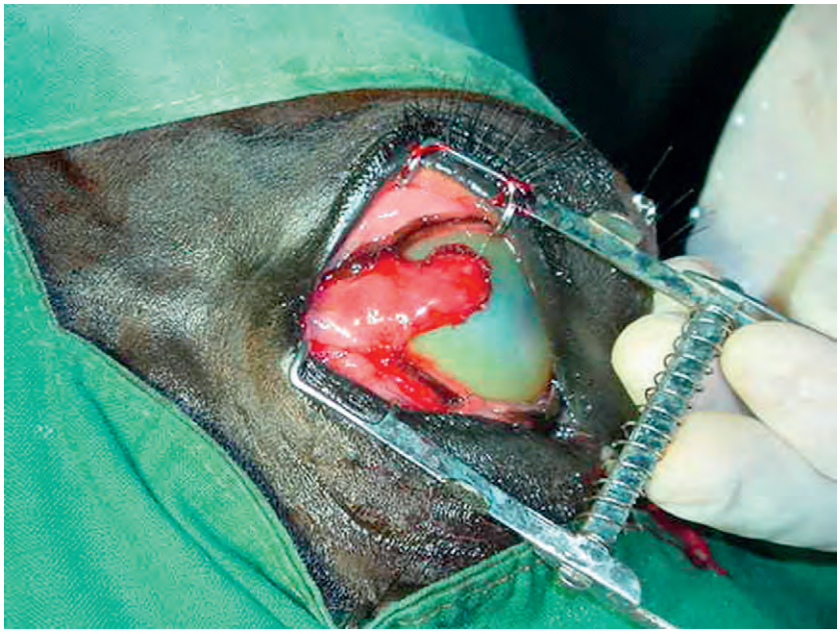


Figure 12.7 Conjunctival pedicle graft: after harvesting the graft from the conjunctiva the graft is sutured to the cornea using 6-0 to 8-0 absorbable suture material, ensuring that it is not under tension and is larger than the ulcer. The bulbar conjunctiva at site of graft collection is closed with the same type of suture material. Grafts are usually trimmed at the level of the limbus when no longer required.

similar to those for a nictitans flap. Partial tarsorrhaphy is useful for improving the retention time of a soft contact lens.

4. Conjunctival pedicle graft. A thin piece of bulbar conjunctiva can be used as a pedicle graft for treatment for deep corneal ulceration, corneal stromal abscess, descemetocoele and corneal perforation (Figure 12.7). Provision of a direct blood supply to the lesion and structural support of the cornea facilitate healing. General anaesthesia of the horse is required.
5. Enucleation may be performed during general anaesthesia of the horse or standing after sedation and use of local blocks, including a retrobulbar block.

12.3 GLOBE AND ORBIT

Congenital abnormalities

- Microphthalmos (small and imperfectly formed eye) may be unilateral or bilateral and can be associated with abnormalities of orbital development. Severity varies from subtle microphthalmos and normal eye function to extreme forms where only a small cyst occupies the orbit. While certain breeds (e.g. Thoroughbreds and Clydesdales) appear predisposed, no hereditary basis has been identified. There is no treatment.
- Anophthalmos (complete absence of the globe) is rare.
- Buphthalmos describes congenital glaucoma with globe enlargement associated with a developmental disorder of the iridocorneal drainage angle (goniodysgenesis).
- Strabismus: rarely, deviation of the position of the eye can occur congenitally.

Acquired abnormalities

Phthisis bulbi

Phthisis bulbi is an irreversibly shrunken and blind globe that can occur following trauma, uveitis, intraocular haemorrhage and occasionally glaucoma. Prominence of the 3rd eyelid is common. The affected eye is often painless, although corneal irritation and ocular discharge may occur. If ocular irritation occurs, enucleation is recommended.

Exophthalmos

Anterior displacement of the globe can occur subsequent to a retrobulbar or retro-orbital space-occupying lesion. Exophthalmos is often accompanied by prominence of the 3rd eyelid, ocular discharge, chemosis and horizontally-orientated cilia on the upper eyelid. Due to impaired eyelid function, exposure keratopathy is common. Causes include:

- Blunt trauma; retrobulbar/retro-orbital haemorrhage and oedema. Supportive treatment involves systemic anti-inflammatory therapy and prevention of exposure keratopathy (artificial tears/topical ocular lubricating creams).
- Cellulitis/abscess (e.g. penetrating trauma, foreign bodies and neoplasia). Orbital pain, blepharoedema, supraorbital fossa swelling, ocular discharge and pyrexia are common. Treatment involves systemic anti-inflammatory and antibiotic therapy, addressing any exposure keratopathy and possibly drainage of a retrobulbar abscess. Prognosis is guarded.
- Neoplasia; numerous neoplasms including melanoma, squamous-cell carcinoma, haemangiosarcoma and lymphoma have been associated with exophthalmos of gradual onset. Additionally, strabismus may occur. Ultrasonography, CT and MRI are useful for assessment and can guide diagnostic and surgical procedures. Treatment may be attempted by surgical excision and/or intralesional radiotherapy/chemotherapy; however, the prognosis is often poor to grave.
- Hydatid cysts; orbital and retro-orbital (e.g. within paranasal sinuses) localization of hydatid cysts is rare. Due to slow expansion of the cyst, gradual onset of exophthalmos may mimic neoplastic involvement. Ultrasonography may help diagnosis. Surgical evacuation can be attempted and diagnosis confirmed by histopathological examination of the brood capsule.
- Ethmoidal haematoma; rare cause of exophthalmos.

Endophthalmos

Posterior displacement of the globe within the orbit may occur due to loss of retrobulbar fat or dehydration. Specific treatment is seldom indicated as re-hydration usually corrects the condition; however temporary entropion may occur in foals and requires treatment.

Prolapse of orbital fat

Rarely, the orbital fat pad (corpus adiposum) can prolapse into the subconjunctival space at the ventral fornix behind the 3rd eyelid, often subsequent to trauma. Lesions appear as static, smooth, soft masses. Differentiation from other soft tissue masses (neoplasia, granuloma) is important and achieved easily by fine-needle aspiration. If treatment is required, surgical removal of the subconjunctival fat is curative.

Glaucoma

See section 12.9.

Endophthalmitis and panophthalmitis

Endophthalmitis (intraocular inflammation contained within the fibrous tunic [sclera and cornea] of the globe) and panophthalmitis (intraocular inflammation that involves the sclera, episclera and possibly the orbital tissues) are encountered infrequently and usually result from penetrating injury to the globe or as complications of ocular surgery.

Clinical signs

- Blepharospasm, epiphora.
- Cloudy or opaque ocular media and corneal oedema.
- Chemosis and conjunctival and episcleral vessel congestion.
- Systemic signs may occur; pyrexia, inappetance and lethargy.
- Panophthalmitis may also manifest with blepharoedema, superorbital swelling, exophthalmos and prominence of the 3rd eyelid.

Treatment and prognosis

Referral for specialist attention is appropriate; therapy with anti-inflammatories and antibiotics (based on bacteriology results of aqueous centesis samples). However, prognosis is guarded, and enucleation or exenteration (removal of the globe and retrobulbar tissues) may be required.

Strabismus

Strabismus is rare and may be congenital, or developmental (trauma, exophthalmos or cranial nerve dysfunction).

12.4 EYELIDS

Congenital disorders

Entropion

Entropion (inward rolling of the eyelid margin) may result in corneal irritation or ulceration from contact with eyelid hairs. The condition is common in neonatal foals and may occur congenitally or post-natally (dehydration, loss of peri-orbital fat, disproportionate growth or ocular discomfort). Entropion occurs rarely in adults, as a cicatricial form subsequent to trauma.

Clinical signs and diagnosis

- The lower eyelid is usually involved.
- Excessive tearing.
- Blepharospasm.
- Conjunctivitis and keratitis.

Diagnosis is achieved readily by clinical examination. Occasionally, entropion may occur as a result of spasms of the orbicularis oculi muscle and globe retraction and may necessitate further ophthalmic examination.

Treatment and prognosis

Surgery is rarely required in foals. Underlying predisposing factor(s) should be addressed and the eyelid repositioned manually or fixed in position temporarily by subconjunctival injections or pleating sutures. Acquired entropion requires blepharoplasty.

Ankyloblephron

This condition is a failure of the eyelids to open at birth. Persistent ankyloblepharon is corrected by manual separation.

Dermoid

Focal congenital masses of displaced skin tissue (dermoids) may involve the eyelid margin, conjunctiva, nictitating membrane or cornea. Conjunctivitis, keratitis or physical disruption of blinking may occur, dependent on the location of the dermoid. Treatment involves surgical removal.

Agenesis and coloboma

Coloboma (dysgenesis) of the eyelid is a full-thickness absence of portions of the normal eyelid or mucocutaneous junction. Severity varies from small defects to complete absence of the eyelid (agenesis). If eyelid dysfunction or ocular disease is present, reconstructive blepharoplasty is necessary.

Acquired disorders

Trauma

Traumatic injury to the eyelids is common due to prominent lateral eye position, equine temperament and the environment. Ophthalmic examination is warranted as concurrent damage to the globe and/or orbit may occur. All eyelid injuries (bruising or lacerations) should be examined and treated as corneal/conjunctival injury may occur if eyelid anatomy and function are compromised.

Clinical signs and diagnosis

Clinical signs and diagnosis are usually obvious, although the inflicting object is seldom identified.

Treatment and prognosis

Eyelid contusions:

- Topical compresses.
- Topical antibiotic ointments to soften and protect the tissues.
- Systemic anti-inflammatories may help reduce palpebral swelling.
- If eyelid function is abnormal, temporary tarsorrhaphy to protect ocular surfaces from dessication.

Full-thickness lacerations require surgical repair with meticulous re-apposition of the eyelid margin.

Ectropion

Ectropion usually is a complication of eyelid trauma that heals with cicatrization (unilateral) or rarely due to ageing (bilateral). Cicatricial ectropion is treated surgically (excision or V-to-Y blepharoplasty).

Prominence of the nictitating membrane (3rd eyelid)

Non-pigmented nictitating membranes may appear prominent while maintaining a normal position. True prominence of the nictitating membrane (pathological) can occur in situations of:

- Altered globe position or size; enophthalmos, exophthalmos, globe trauma, phthisis bulbi, marked ocular pain, microphthalmos and cystic globe.
- Inflammation of the nictitans.
- Neoplasia of the nictitans; most commonly SCC and rarely lymphoma, haemangiosarcoma, lipoma, neurofibroma, adenocarcinoma or melanoma.
- Tetanus; bilateral prominence and flicking of the 3rd eyelid.
- Horner's syndrome; prominence of the nictitans is rare in horses; ptosis, localized sweating and increased skin temperature are most obvious.
- Prolapse of the orbital fat pad.
- Conjunctival amyloidosis.

Clinical signs and diagnosis

The clinical appearance is often diagnostic; however, additional signs may be representative of the underlying disorder. Masses should be sampled by needle aspiration, biopsy or surgical excision, and samples should be submitted for cytology and/or histopathology.

Treatment and prognosis

Treatment and prognosis are dependent on the underlying cause. Neoplasms of the 3rd eyelid are often treated with surgical removal, while tetanus requires supportive nursing and use of anti-toxin. Prolapse of the orbital fat pad may not require treatment if function of the eyelids or cornea is not compromised. Treatment for retrobulbar and retro-orbital masses is described in section 12.3.

Inflammatory diseases of the nictitating membrane

Involvement of the nictitans is frequent in conjunctivitis; follicular hyperplasia may be observed on the palpebral and ocular surfaces of the 3rd eyelid. In horses with a lack of conjunctival pigment, solar-induced inflammation can occur and is a potential precursor of SCC.

Inflammatory diseases of the palpebrae

Inflammatory conditions of the eyelids are uncommon but include:

- Bacterial blepharitis; usually *Moraxella equi*.
- Parasitic disease; e.g. oncocerciasis, habronemiasis, mites.
- Inflammatory dermatological conditions; dermatophytosis, dermatophilosis, eosinophilic granulomas.
- Immunologic dermatological conditions; e.g. pemphigus foliaceus, cutaneous lupus erythematosus, hypersensitivity reactions.
- Juvenile Arabian fading syndrome.
- Photosensitization.
- Abscess; penetrating trauma, complication of sub-palpebral lavage tube.
- Meibomianitis; inflammation of the meibomian glands, likely immune-mediated and often secondarily infected.
- Neoplasia.

Clinical signs and diagnosis

- Swelling, crusting/scaling and alopecia of the eyelid.
- Conjunctivitis.
- Ocular discharge.

Diagnosis is by consideration of history, clinical appearance and use of ancillary aids (skin scrapings, aspirate/biopsy and microbiology).

Treatment and prognosis

Treatment is dependent on the condition. Most can be resolved or controlled with appropriate therapy; however, prognosis for eyelid neoplasia is guarded.

Neoplasia

The three most common eyelid neoplasms are SCC, sarcoid and melanoma. Viral papillomas may be found in the periocular region of young horses. Other neoplasms that may involve the eyelids include basal cell tumour, mast cell tumour, lipoma, fibroma, fibrosarcoma, haemangiosarcoma, adenocarcinoma or lymphoma.

Both intrinsic and extrinsic factors may be involved in the development of periocular neoplasms. Age, breed (draft, Appaloosa/Paint horses, Quarter horses and Thoroughbreds), lack of ocular/periocular pigmentation, gender (geldings) and genetics are intrinsic factors for development of SCC, while UV radiation exposure and chronic irritation are extrinsic factors. For sarcoids, strong evidence exists for a role of bovine papillomavirus in the aetiopathogenesis, and risk factors include breed (Appaloosas, Arabians and Thoroughbreds), age (young horses) and genetic predisposition.

Clinical signs and diagnosis

Clinical appearance is variable, and diagnosis should be based on histopathological examination. Lesions are often proliferative, but SCC may be ulcerative (Figure 12.8).

Treatment and prognosis

Treatment of palpebral neoplasia presents certain challenges. Except for discrete neoplasms involving the 3rd eyelid (usually SCC), complete surgical excision is seldom feasible as disfigurement and loss of eyelid function is likely. Treatment options include:

Figure 12.8 Squamous cell carcinoma involving the 3rd eyelid and inferior eyelid. Note the absence of pigmentation in the affected regions.

- Surgical debulking prior to use of adjunctive therapy. Sole use of conventional or CO_2 laser surgery is associated with the highest recurrence rates of SCC and sarcoids.
- Cryotherapy: 2 or 3 freeze-thaw cycles using liquid nitrogen for small superficial tumours.
- Intralesional chemotherapy (cisplatin, 5-fluorouracil); this is often effective, but precautions for use of a cytotoxic drug are required.
- Topical chemotherapy (5-fluorouracil) is useful for superficial dermal neoplasms.
- Intralesional radiotherapy is often very effective, but its use is limited by availability and expense.
- Intralesional BCG; effective treatment for sarcoids.
- Piroxicam is an NSAID with anti-neoplastic effects that, anecdotally, is effective for treatment of periocular SCC.
- Benign neglect. Some tumours (e.g. occult sarcoids) may be observed and treated only if required. Viral papillomas are self-limiting and seldom require treatment.

Prognosis for most periocular neoplasms (excluding papilloma) depends on recurrence, local invasion or metastasis.

12.5 CONJUNCTIVA, LIMBUS, EPISCLERA, SCLERA

Congenital disease

Dermoids

Dermoids often involve the conjunctiva adjacent to the limbus. Some are asymptomatic, while others cause corneal irritation and require surgical removal.

Acquired disease

Conjunctivitis

Inflammation of the conjunctiva is common and may be a primary or secondary to an underlying disease.

Causes of primary conjunctivitis include:

- Infectious agents: e.g. *Moraxella equi*, *Onchocerca cervicalis*, *Habronema* spp. and fungi.
- Trauma.
- Irritant, allergic and chemical stimuli – environmental (smoke, dust, pollen, vegetation), iatrogenic (ophthalmic preparations, fly sprays etc.).

Causes of secondary conjunctivitis include:

- Ocular diseases; keratitis, disorders of tear production and drainage, eyelid disease, uveitis, endophthalmitis and neoplasia.
- Trauma.
- Foreign bodies.
- Respiratory viral infections (e.g. equine influenza, equine herpesvirus).

Clinical signs and diagnosis

Common signs:

- Blepharospasm.
- Ocular discharge (serous, mucoid, purulent or haemorrhagic).
- Conjunctival reddening (hyperaemia).
- Conjunctival swelling (chemosis).

Follicular inflammation of the caruncle, 3rd eyelid and peri-limbal conjunctiva may occur. Occasionally, granulomatous inflammation develops in response to chronic antigenic stimulation (e.g. habronemiasis, foreign material) or subconjunctival deposition of medications.

Gross characteristics of the conjunctiva and investigation of primary causes or underlying disease are used for diagnosis. An appreciation of the normal conjunctival flora is important for interpretation of swabs/scrapings. In normal horses, Gram-positive bacteria predominate; however, climatic variation may influence results. Fungi are often isolated from normal horses, particularly when climatic (e.g. subtropical/tropical) and environmental conditions are favourable. Gram-negative organisms are more frequently isolated in conjunctival disease. Biopsy of granulomatous lesions is indicated for differentiation from neoplasia.

Treatment and prognosis

Once the cause of conjunctivitis has been established, appropriate therapy can be instituted. Bacterial disease is treated with ophthalmic preparations of antibiotics, and ointments formulations are useful (prolonged retention time). Irritant/allergenic inflammation often resolves with change of environment; anti-inflammatory therapy may be required. Granulomatous lesions may require anthelmintic and/or anti-inflammatory therapy administered locally or systemically dependent on aetiology. In general, prognosis for conjunctivitis is good to excellent, if the inciting cause is removed. Neoplastic disease has a guarded to poor prognosis.

Trauma

Blunt and penetrating trauma to the orbit can injure the conjunctiva and cause haemorrhage/haematoma, conjunctivitis and orbital fat pad prolapse. Rarely, orbital fractures that communicate with the paranasal sinuses can result in conjunctival emphysema. Specific therapy is seldom necessary, although systemic anti-inflammatory and antibiotic therapy and occasionally surgery may be required. Chemosis and dessication of exposed conjunctiva can be treated with topical hypertonic ointments/solutions (saline solution or dextrose/glucose); however, prolonged use can be irritant.

Conjunctival foreign bodies

Most foreign bodies are usually plant matter entrapped in the superior or inferior fornix or behind the 3rd eyelid. Ocular discomfort and excessive lacrimation are typical. Careful examination is required to identify foreign material (facilitated by a palpebral nerve block and topical anaesthesia). Foreign body removal using a cotton wool bud is usually curative, unless ulceration of the cornea is present.

Neoplasia

Squamous-cell carcinoma (SCC) is the most common primary neoplasm of the conjunctiva and limbus. A predilection for zones of epithelial transition exists, including the mucocutaneous junction of the eyelid, limbus, 3rd eyelid and medial canthus.

Clinical signs and diagnosis

Clinical appearance varies, dependent on location, duration and type of SCC. Limbal SCCs are proliferative, pink, raised and 'cobblestone/cauliflower' in appearance and bleed easily, while lesions involving the nictitating membrane or conjunctiva may be proliferative and/or ulcerative. Ulcerative lesions often have mucopurulent discharge and a fetid odour, reflective of bacterial infection. Early 3rd eyelid lesions may present as slightly raised, roughened lesions; however, substantial involvement of the eyelid may develop, including cartilage erosion and extension to the medial canthus and orbit. Diagnosis of SCC is

by histopathological examination of a biopsy sample; however, cytological examination of scrapings obtained from lesions may be useful.

Treatment and prognosis

Treatment options for SCC are described in section 12.4. The prognosis of SCC is guarded as local invasion of the orbit, globe and regional tissues may occur. Additionally, local recurrence is common, and lesions may develop in adjacent tissue.

Other neoplasms of the conjunctiva and sclera infrequently encountered include lymphoma, haemangioma, haemangiosarcoma and melanoma. Haemangiosarcoma and lymphoma warrant a poor prognosis due to local spread or systemic involvement.

Conjunctival pseudotumour

Ocular pseudotumours are proliferative inflammatory lesions affecting the bulbar conjunctiva or limbus that mimic neoplasia. Lesions may have a smooth surface or a granulomatous appearance. Differentiation from neoplasia is important; biopsy and histopathological examination are required. Lymphocyte-dominated granulomatous inflammation is characteristic and suggests an immune-mediated pathogenesis. Treatment with surgical excision and/or intralesional corticosteroids is usually curative.

Conjunctival amyloidosis

This is a rare condition that may present with diffuse thickening of the conjunctiva and eyelids or discrete nodules. Histopathological examination of a biopsy is required for diagnosis and differentiation from neoplasia. Prognosis is guarded, although some response to treatment with corticosteroids may be expected.

12.6 NASOLACRIMAL SYSTEM

Obstructive disorders

Dysgenesis

Abnormal development usually involves absence of the nasal ostium and a variable component of the distal duct. Conversely, dysgenesis of the proximal punctae and duct is rare.

Clinical signs and diagnosis

- Signs develop early in life.
- Marked epiphora or mucopurulent ocular discharge.
- Signs of ocular irritation and discomfort.

Close examination of the nares reveals an absence of the punctum. Absence of a patent nasolacrimal duct can be confirmed by lack of fluorescein stain at the ipsilateral naris after placement onto the eye, normograde cannulation of the duct or dacryocystorhinography. The later technique allows determination of the extent of the atresia prior to surgical intervention.

Treatment and prognosis

A urinary catheter (6–10F) is passed into the duct via the upper punctum until it reaches the obstruction. A distal ostium of the duct is then created by incising the nasal tissues at the tip of the catheter. The distal portion of the catheter is pulled through the incision, and both the proximal and distal ends are fastened using sutures and the catheter is retained in situ for 2–4 weeks.

Acquired stenosis or occlusion

Partial or complete obstruction of the nasolacrimal duct can occur subsequent to inflammatory, neoplastic or traumatic processes.

Internal conditions include:

- Dacryocystitis.
- Foreign bodies.
- Neoplasia (especially SCC, sarcoid).
- Parasitic granuloma (especially habronemiasis).

External conditions include:

- Trauma.
- Neoplasia.
- Disease of the maxillary dental arcade.
- Sinusitis.

Clinical signs and diagnosis

Epiphora is common; however, if infection occurs, mucopurulent discharge is typical. Additionally, signs of an underlying disease may be present. Diagnosis is usually obtained by careful examination and the techniques described for dysgenesis.

Treatment and prognosis

Treatment is reliant on an accurate diagnosis and is aimed at specific treatment for the underlying cause and re-establishing patency of the nasolacrimal system. In some horses, an indwelling catheter may be required for 2–4 weeks. If bacterial dacryocystitis is present topical antibiotics are indicated. If an inflammatory cause is suspected topical corticosteroids can be used. Topical medications should be solutions rather than ointments.

Keratoconjunctivitis sicca (KCS)

Deficiency of the aqueous portion of the PTF is uncommon. Possible causes include:

- Trauma to the lacrimal gland.
- Immune-mediated damage to the lacrimal gland.
- Toxic damage (locoweed) to the lacrimal gland.
- Parasitic dacryoadenitis.
- Traumatic damage to the innervation of the lacrimal gland (parasympathetic fibres of the facial nerve).

Clinical signs and diagnosis

- Ocular pain.
- Dull, lacklustre cornea.
- Corneal oedema, keratitis.
- Mucopurulent ocular discharge.

Use of the Schirmer tear test (STT) helps confirm the diagnosis; readings <10 mm (often 0 mm) are characteristic of KCS.

Treatment and prognosis. Ocular discharge should be removed and artificial tears or ocular lubricants applied regularly. Topical cyclosporine A may reduce lacrimal gland inflammation and directly stimulate lacrimation. Prognosis is guarded, although lacrimation may improve in some horses. Facial nerve injury-induced KCS can be exacerbated by an inability to blink causing exposure keratopathy.

12.7 CORNEA

Corneal disease is the most frequent reason for ophthalmological examination in horses. Many corneal diseases are the result of trauma, subsequent to the prominent lateral location of the eyes, large corneal surface and the behavioural nature of horses. Corneal clarity is

essential for normal vision and can be disrupted by any pathological process involving the cornea, including neovascularization, oedema, scarring, melanosis, lipid or mineral or cellular infiltration. The influence on vision is dictated by lesion location and area, corneal reparative responses and presence of intra-ocular disease.

Congenital diseases

Congenital disorders of the equine cornea are rare. Those reported include:

- Microcornea; can occur in association with a globe of normal size or microphthalmos.
- Megalocornea.
- Sclerocornea.
- Corneal dermoids.
- Pannus.
- Congenital melanosis or leukoma.

Acquired diseases

Acquired corneal disorders are common and may be either traumatic or non-traumatic in origin, inflammatory or non-inflammatory and ulcerative or non-ulcerative.

Corneal degenerative disease

Dry eye syndromes

Dry eye can result from tear deficiency, evaporative disorders or both.

- Keratoconjunctivitis sicca (discussed in section 12.6).
- Evaporative dry eye/exposure keratopathy:
  - Eyelid problems; inadequate blinking (facial nerve palsy +/– corneal anaesthesia), incongruity of the eyelid margins and blepharitis.
  - Meibomianitis.
  - Exophthalmos/buphthalmos.

Clinical signs are as described for KCS. Investigation of dry eye should include a STT, assessment of blinking, examination of the eyelid margins (including the meibomian glands) determination of corneal sensitivity and assessment of the corneal surface using slit-lamp biomicroscopy and staining (fluorescein and Rose Bengal). Treatment is dependent on the underlying condition.

Corneal mineralization

Accumulation of minerals (usually calcium) in the cornea occurs subsequent to ocular inflammation (usually iridocyclitis). Affected areas are gritty and appear as crystalline or shiny white regions, often with oedema and neovascularization of the cornea. In addition to addressing underlying disease, mineralization can be treated by keratectomy; however, recurrence may occur. More recently topical chelation therapy using EDTA has achieved good results.

Other degenerative disorders of the cornea:

- Fibrosis.
- Atrophy.
- Oedema.
- Corneal sequestration.
- Staphyloma.

Non-inflammatory corneal diseases

Linear keratopathy

Linear keratopathy is non-oedematous, horizontal, striate corneal opacities in the posterior cornea. An occasional incidental finding in otherwise normal eyes, it is of no significance. Lesions are non-progressive and do not affect vision. No treatment is required. Linear keratopathy should be differentiated from the striae commonly observed in glaucoma.

Primary corneal oedema

This is a rare idiopathic condition involving dysfunction of the endothelium and movement of water into the stroma. The oedema is typically non-painful; however, turgidity of the cornea may result in superficial bullae (bullous keratopathy) that can result in painful shallow erosions. Treatment is difficult; however, hyper-osmotic solutions can be used short-term, or cyclosporine A/corticosteroids can be administered if an inflammatory cause is suspected.

Keratitis

Traumatic keratitis

Traumatic keratitis varies from mild erosions to penetrating lesions (Figure 12.9) and causes are diverse. Disorders of the adnexa (e.g. ectopic cilia, entropion/ectropion, eyelid laceration), foreign bodies and mild external trauma are common causes of superficial ulceration, while more substantial trauma can result in partial- or full-thickness lacerations. Traumatic sequelae may include iridocyclitis, hyphaema, tearing of granula iridica, synechiae, lens luxation, cataracts, blindness and phthisis bulbi. Full-thickness lacerations usually result in leakage of aqueous humor, and iris prolapse frequently seals the wound.

Diagnosis and treatment

- Blepharospasm and lacrimation.
- The extent of the corneal injury can usually be determined with a thorough examination, but any involvement of deeper structures may be obscured; ultrasonographic examination is useful, provided full thickness corneal injury is absent.
- Fluorescein dye will delineate the ulcer and can provide evidence of a desmetocoele (deep ulcer, often bulging of Descemet's membrane that doesn't stain, and a halo of stain uptake at the periphery of the lesion) and full-thickness injury (Seidel's test).
- Loose flaps of corneal tissue should be removed.
- Superficial erosions can be treated conservatively using topical antimicrobials.
- Deeper lacerations often require symptomatic therapy for iridocyclitis and topical broad-spectrum antimicrobial therapy. Anti-collagenase therapy can be instituted if keratomalacia is suspected. Selection of antimicrobial therapy can be dictated by bacteriological examination of corneal swab/tissue or on the basis of suspected pathogens.
- The adnexa should be checked for foreign bodies.
- Injuries involving more than a third of the corneal thickness require support by use of a conjunctival pedicle graft.
- Full-thickness lacerations require surgical intervention to promote healing and minimize complications; in acute cases, prolapsed iris can be carefully returned to the anterior chamber, while in more protracted cases, necrotic/compromised iris tissue requires debridement before attempts to repair the corneal defect. The anterior chamber is reformed using saline solution, hyaluronic acid or air, and the corneal laceration can then be sutured under magnification using 7/0–10/0 suture material, ensuring good apposition of the wound edges.

Ulcerative keratitis

A corneal ulcer is characterized by loss of epithelium, and with progression the stroma becomes involved. Ulceration can occur from traumatic insults, foreign bodies and/or inadequate corneal protection

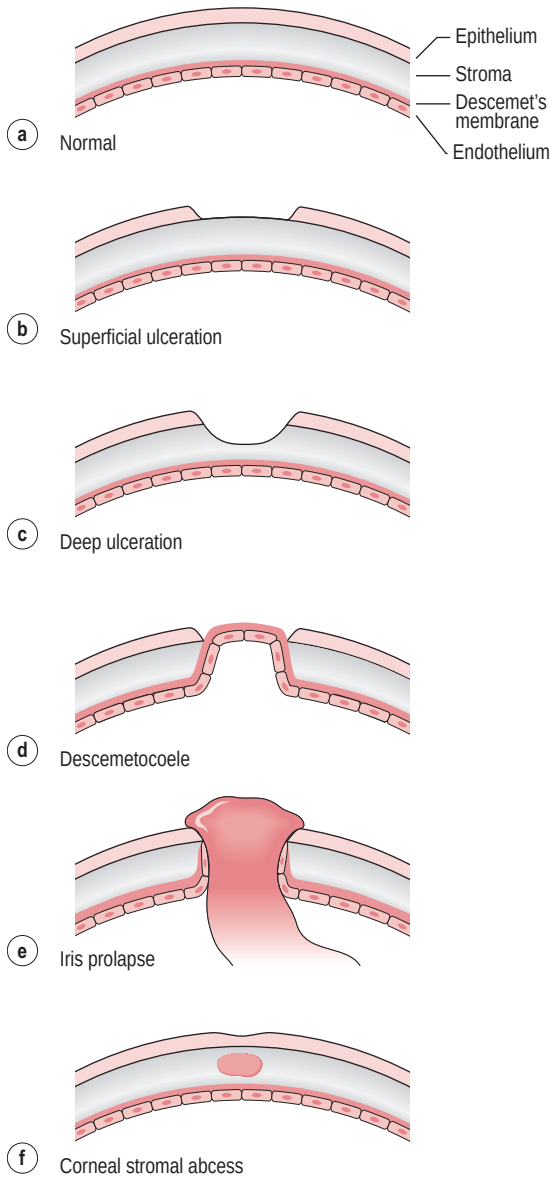


Figure 12.9 Schematic diagram of various types and severity of traumatic corneal disease. The normal cornea is comprised of five principal layers: epithelium, basement membrane, stroma, Descemet's membrane and endothelium (a). In superficial corneal ulceration, epithelium and basement membrane are lost (b). More severe ulceration includes loss of stroma (c) and, if progressive loss of stroma occurs, exposure of Descement's membrane may result in development of protrusion of the membrane through the corneal defect (descemetocoele) (d) or corneal perforation (e). Re-epithelization over a focus of stromal inflammation/infection may result in a corneal stromal abscess (f).

(e.g. KCS, eyelid dysfunction). While external trauma is the most common cause, it is a diagnosis of exclusion upon elimination of other possibilities. Most ulcers are presumed to be infected; however, bacteria do not initiate ulceration, but rather involvement is secondary. With this in mind, therapy is devised to address any underlying cause, infection and sequelae of infection. The aetiopathogenesis of ulcerative keratitis is complex, and in addition to the initial breach of the epithelial barrier, infection with bacteria or fungi, release of proteases from microorganisms, leukocytes and/or corneal cells and

Figure 12.10 Mycotic keratitis in a horse: a large, deep ulcer in the temporal cornea and dense corneal vascularization from the dorsal, temporal and ventral limbus is present.

anti-proteases may be involved. In ulcerative keratitis Gram-positive bacteria (e.g. *Streptococcus* spp., *Staphylococcus* spp.), Gram-negative bacteria (e.g. *Pseudomonas* spp., Enterobacteriaceae, *Acinetobacter* spp.) and/or fungi (e.g. *Aspergillus* spp., *Fusarium* spp., *Alternaria* spp., *Cladosporium* spp.) may be involved. Sterile ulceration may occur also. Mycotic keratitis exhibits geographical and seasonal variability in occurrence; it is common in southern states of the USA, while it is less common in Europe. Causative fungi are usually opportunistic saprophytic organisms that invade the cornea when the epithelium is compromised. Mycotic keratitis can progress rapidly if untreated or when treated with antibacterials or corticosteroids.

Clinical signs and diagnosis

Signs may include:

- Blepharospasm and photophobia.
- Serous ocular discharge that may become mucoid or purulent.
- Conjunctival hyperaemia.
- Corneal oedema.
- Iridocyclitis (miosis, aqueous flare/hypopyon).
- Raised edges of the ulcer (under-running of the epithelium) that may trap purulent exudate.
- Corneal neovascularization.
- Mycotic keratitis can present in various forms, including:
  - Superficial ulceration.
  - Deeper ulceration with a raised border of dry white exudate ('cake-frosting') with/without perilesional infiltrates ('satellite lesions') (Figure 12.10).
  - Keratomycosis with surrounding furrow.
  - Corneal abscess.

Diagnosis is achieved by direct examination of the cornea and fluorescein staining. Unless ocular discharge is prominent, a STT should be performed. The adnexa should be examined for any abnormalities including foreign bodies. More advanced ulcers, ulcers poorly responsive to therapy or ulcers that could be mycotic warrant further investigation including:

- Swabs/scrapes/corneal tissue for cytology and microbiology.
- Corneal biopsies for histopathological examination.
- Examination of the cornea, anterior chamber and iris by biomicroscopy and direct ophthalmoscopy.

Treatment and prognosis

Prior to treatment, every corneal ulcer should be characterized, based on the depth and chronicity:

- A simple ulcer is superficial (only the epithelium is involved) and heals rapidly (within 7 days)
- Complicated ulcers involve the corneal stroma and/or are present for >7 days.

Treatment options include:

- Antimicrobial therapy (Table 12.1). Empirical selection of a broad-spectrum antimicrobial treatment will frequently suffice, particularly in management of simple ulcers. When a complicated ulcer is present, especially with gross evidence of stromal infection, or when ulcer progression or poor response to therapy exists, antimicrobial selection should be based on results of cytological examination and culture results. In such cases, an antimicrobial protocol effective against Gram-negative organisms is often required as *Pseudomonas* species are commonly involved in aggressive corneal ulcers. Further, fortified solutions and possibly subconjunctival administration of antimicrobials (Table 12.2) may be used. Frequency of administration is dictated by lesion severity and antimicrobial preparation used. Ointments have a longer resident time on the cornea than solutions, permitting less frequent administration. However, solutions have enhanced bioavailability and are generally preferred, although frequent application may necessitate the placement of a lavage system (see 'Techniques in ocular therapeutics').
- Anti-fungal therapy (Table 12.1) when fungal keratitis is suspected or confirmed on the basis of cytological and mycological examination of corneal samples. Response to treatment can be slow, and prolonged treatment may be required; in many cases with stromal involvement a minimum of 6 to 8 weeks of treatment is necessary.
- Anti-collagenase therapy can be instituted if keratomalacia is suspected.
- Mydriatics; reflex uveitis commonly accompanies corneal ulceration, and 1% atropine should be applied topically until mydriasis is achieved. Thereafter atropine should be applied as required to maintain mydriasis.
- Systemic NSAID therapy is useful to treat reflex uveitis. Topical NSAID therapy should be avoided as further corneal irritation can result.
- Surgical therapy may be warranted for treatment of complicated ulcers when progression of the ulcer, loss of more than a third of the stromal thickness or delayed healing is present. Goals include corneal stabilization and/or protection during healing, and techniques include:
  - Superficial keratectomy to remove compromised epithelium/stroma.
  - Temporary tarsorrhaphy.
  - 3rd eyelid flap.
  - Conjunctival graft techniques; pedicle/360°/bridge.
  - Corneal grafts.
  - Transplantation of equine amniotic membrane.

Prognosis is guarded and dependent on the speed of corneal re-epithelialization, ability to remove any predisposing factor(s) and response of pathogens to antimicrobial therapy. Progressive ulceration may result in keratomalacia, descemetocoele formation and corneal perforation, while re-epithelialization over corneal detritus can result in a stromal abscess. Corneal neovascularization, fibrosis and melanosis cause permanent opacification of healed cornea. Prognosis of mycotic keratitis is always guarded as response to treatment is unpredictable, particularly when stroma adjacent to Descement's membrane is involved.

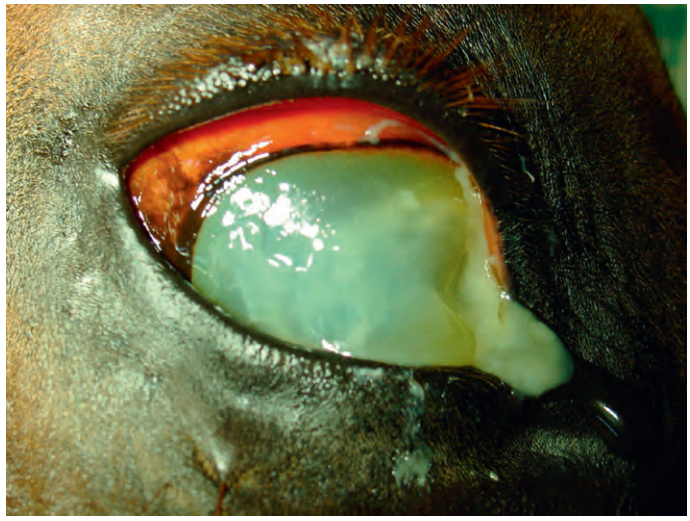


Figure 12.11 Extensive keratomalacia (melting cornea) involving the right eye of a horse. Marked liquefactive necrosis of the corneal stroma is present, due to the effects of endogenous and exogenous proteases. A *Pseudomonas* species was cultured from debrided corneal tissue.

Keratomalacia ('melting cornea')

Keratomalacia reflects liquefactive necrosis of the corneal stroma and is an ocular emergency. Keratomalacia results from exuberant production of proteinases by micro-organisms, leukocytes, corneal epithelial cells and fibroblasts. When the counteractive effects of endogenous anti-proteinases are overwhelmed, dramatic destruction of the stroma occurs and may progress rapidly to corneal perforation. Causative organisms include *Pseudomonas* species, *Streptococcus* species and fungi; however, sterile keratomalacia can occur.

Clinical signs and diagnosis

- Greyish, gelatinous (liquefactive) corneal opacity that often protrudes from the corneal surface is characteristic and diagnostic of keratomalacia (Figure 12.11).
- Marked blepharospasm and photophobia.
- Profuse serous to purulent ocular discharge.
- Corneal neovascularization in subacute/chronic cases.
- Signs of iridocyclitis are common.
- Fluorescein staining reveals the extent of ulceration.

Treatment and prognosis

Aggressive medical therapy is paramount and may be supplemented with judicious use of surgical procedures:

- Topical treatment with fortified antimicrobials are indicated (Table 12.2), and anti-fungal therapy is required when fungi are suspected/confirmed. Treatment should be instigated immediately and repeated every 1–2 hours via a subpalpebral lavage system. Treatment frequency can be reduced after improvement is evident.
- Treatment for reflex uveitis is warranted.
- Anti-proteinase therapy (Table 12.1) is of paramount importance. Combination therapy (e.g. endogenous serum, EDTA and acetylcysteine) may have an additive or synergistic effect. Preparations should be applied hourly until improvement is noted, after which treatment every 4–6 hours is sufficient.
- If corneal perforation is possible or healing is poor, corneal stabilization and provision of blood supply via a conjunctival pedicle graft are useful. Other adjunctive surgical techniques

Figure 12.12 Corneal perforation and prolapse of the iris subsequent to keratomalacia. Hyphaema, hypopyon and a constricted pupil are reflective of marked intra-ocular inflammation.

include superficial keratectomy, corneal graft or amniotic membrane graft.

Prognosis is guarded to poor and depends on successful restoration of the balance of proteinases and anti-proteinases and removal of contributory pathogens. When keratomalacia is halted quickly, corneal fibrosis may be limited and visual outcome good. However, more extensive and poorly responsive lesions can result in extensive fibrosis and impaired vision. Further, keratomalacia can result in corneal perforation (Figure 12.12), enophthalmitis, glaucoma or phthisis bulbi that may require enucleation.

Corneal stromal abscess

A corneal abscess may develop after re-epithelialization over a micropuncture or purulent debris in a stromal lesion. Gram-positive cocci and Gram-negative rods are the most common bacteria in corneal abscesses. Fungal involvement can occur and should be suspected when the abscess involves deep stromal tissues.

Clinical signs and diagnosis

- Focal, yellow/white/pink opacity of variable depth in the stroma (Figure 12.13).
- Corneal neovascularization is common.
- Moderate to marked iridocyclitis and signs of ocular pain are common.

The corneal appearance is usually diagnostic. Debrided corneal tissue can be submitted for cytological and microbiological examination.

Treatment and prognosis

Because of cornea avascularity, stromal abscesses do not heal until vascularization occurs centripetally. Healing of superficial or mid-stromal abscesses may be accelerated by use of antimicrobials with good corneal penetration (e.g. chloramphenicol, ciprofloxacin) and systemic antimicrobials may be useful for treatment of perilimbal abscesses. Surgical debridement of diseased stroma can hasten healing and promote penetration of topical antimicrobials. Deep abscesses often respond poorly to medical therapy and may not be accessed by the neovascularization response. Such cases often benefit from penetrating keratoplasty and corneal transplantation. Treatment of iridocyclitis is necessary. The minimum effective dose of NSAIDs should be used to minimize impediment of corneal neovascularization. Prognosis is guarded as some lesions are refractory to treatment and can compromise the integrity of the globe and vision.

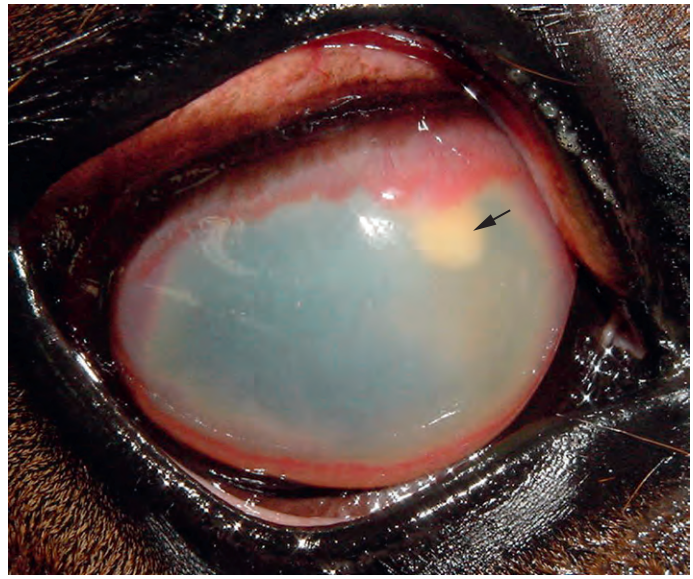


Figure 12.13 Corneal stromal abscess (arrow) and dense neovascularization response in the dorsal region of the cornea in a horse that had sustained an injury to the cornea several weeks previously.

Viral keratitis

Equine herpesvirus type 2 has been implicated as a cause of keratitis; however, diagnosis is made from response to treatment and not viral isolation. For unknown reasons the disease is reported most often in the UK and rarely in the USA.

Clinical signs, diagnosis and treatment

Putative viral keratitis may be ulcerative or non-ulcerative:

- Superficial punctate keratitis is most common; multiple superficial circular opacities, accompanied by blepharospasm and lacrimation.
- Shallow ulcers.
- Generalized epithelial irregularity causing an 'orange peel' appearance.
- Reticulated or dendritic patterns of opacities in the anterior stroma.

Diagnosis is made on appearance and rapid response to anti-viral medications: idoxuridine, trifluridine, vidaribine or acyclovir can be used; however, availability of commercial ophthalmic preparations is limited.

Parasitic keratitis

Keratitis or keratoconjunctivitis can result from the migration and death of *Onchocercia cervicalis* microfilaria in the corneal and conjunctival tissues. An immune response to dead microfilaria likely initiates disease, and the onset may be preceded by administration of ivermectin. Onchocerciasis is now uncommon in many geographical areas. Signs include ocular pain, conjunctival hyperaemia, corneal opacities and/or vascularization. Depigmentation of the perilimbal bulbar conjunctiva may develop in quiescent cases. Diagnosis is based on clinical signs and supported by demonstration of microfilaria and

inflammatory cells (especially eosinophils) in corneal/conjunctival biopsies. Treatment is directed towards removal of microfilaria using systemically administered ivermectin and control of inflammation using topical and/or systemically administered corticosteroids (Tables 12.1 and 12.3). Prognosis is good; however, recurrence is possible

Other inflammatory conditions

Several miscellaneous rare inflammatory or ulcerative conditions of the cornea are beyond the scope of this chapter, including:

- Eosinophilic keratitis.
- Suspected immune-mediated non-ulcerative keratopathies.
- Superficial non-healing ulcerative keratopathy (refractory ulceration).
- Pigmentary keratopathies.
- Corneal pannus.

Neoplasia

Neoplasms of the equine cornea are rare, usually epithelial in origin and result from direct extension of primary neoplasms involving the limbus or conjunctiva. Three forms of corneal neoplasia are reported, but all are likely stages in the development of SCC.

Clinical signs, diagnosis and treatment

- Corneal keratosis is benign pink papillomatous proliferation of corneal epithelium.
- Intraepithelial carcinoma in situ is manifested by thickening, vascularization and roughening of the epithelium.
- SCC is characterized by malignant transformation of epithelial cells with extension to involve stroma and rarely intra-ocular tissues. Lesions are proliferative, usually peri-limbal and often with conjunctival involvement.

Diagnosis is achieved by clinical and histopathological findings. Keratectomy with/without adjunctive therapy (cryotherapy, radiation therapy, CO_2 laser ablation) is preferred for treatment in most cases; however, cryotherapy/radiation therapy may suffice for lesions <2 mm in thickness.

12.8 UVEAL TRACT

The uveal tract is the vascular and pigmented layer of the eye and comprises of the iris, ciliary body and choroid. Variation in the degree of pigmentation results in differing iris colour between horses and even within eyes (heterochromia). Uveal tract disease is common in horses and may be primary or secondary.

Congenital diseases

Aniridia

Congenital absence of the iris is an occasional isolated finding or may occur with other lesions e.g. cataracts or dermoids. In Belgian horses the condition is hereditary. Affected horses may exhibit photophobia and have large, unresponsive pupils.

Coloboma

Coloboma (congenital absence of a portion of the eye) of the uveal tract may involve the iris or choroid and are classified as either typical (i.e. midline) or atypical if located in any other position.

Clinical signs and diagnosis

Partial or complete holes in the iris are characteristic of coloboma, however they should be differentiated from acquired iris atrophy and

Table 12.3 Doses of systemic medications used commonly for treatment for ocular disease

Category/drug	Dosage	Comments
Antimicrobials		
Sodium/potassium Penicillin G	20000–40000 IU/kg IV, IM q6hr	Bacteriocidal, efficacy against G+
Procaine penicillin	20 mg/kg IM q12hr	Bacteriocidal, efficacy against G+
Gentamicin	6.6 mg/kg IV, IM once daily	Bacteriocidal, efficacy against G– bacteria and *Staphylococcus* spp.
Trimethoprim-sulphonamide	30 mg/kg PO q12hr 15 mg/kg IV q12hr	Bacteriocidal in combination, broad spectrum
Doxycycline	10 mg/kg PO q12hr	Bacteriostatic, broad spectrum.
Antifungals		
Itraconazole	3 mg/kg PO q24hr	Triazole antifungal. Excellent efficacy against *Aspergillus* spp. Poor efficacy against *Fusarium* spp. Very expensive
Fluconazole	5 mg/kg PO q24hr	Triazole antifungal. Poor efficacy against *Aspergillus* spp. Expensive.
Voriconazole	4 mg/kg PO q24hr	Triazole antifungal. Excellent oral bioavailability and good distribution to tear film and aqueous humor. Broad spectrum of efficacy against filamentous fungi.
Anti-inflammatories		
Flunixin meglumine	0.5–1 mg/kg IV q12–24hr 0.5 mg/kg PO q12–24hr	Potent NSAID. Also provides analgesia. Long-term use or high dosages may result in gastrointestinal and/or renal toxicity.
Phenylbutazone	2.2–4.4 mg/kg IV/PO q12–24hr	NSAID. Also provides analgesia. Long-term use or use of high dosages may result in gastrointestinal and/or renal toxicity.
Dexamethasone	0.05–0.1 mg/kg IV, IM q12–24hr	Potent corticosteroid. Use should be avoided in horses with a history of laminitis
Prednisolone	1–2 mg/kg PO q12–24hr	Use should be avoided in horses with a history of laminitis

iris hypoplasia. Colobomas may be single or multiple and vary in thickness. A fundus refection can be appreciated through full-thickness defects. Colobomas of the choroid appear as well defined areas of the fundus that are lighter in colour than surrounding normal tissue. They are most evident when the non-tapetal fundus is involved as they affect the pigmented retinal epithelium. Most horses with a coloboma of the uveal tract, are asymptomatic; there is no treatment.

Persistent pupillary membranes

The pupillary membrane is the temporary anterior vascular tunic present on the surface of the lens and iris during embryological development. When segments of the membrane fail to undergo atrophy they remain as permanent remnants. The strands or filaments extend from the mid iris collarette and project freely into the anterior chamber or attach to the corneal endothelium or anterior surface of the lens resulting in focal opacities. There is no evidence for a hereditary basis, and lesions usually do not interfere with vision. No treatment is necessary.

Hypertrophy of the granula iridica

The granula iridica (corpora nigra; black bodies) are solid, pigmented irregular bodies present on the upper and lower edges of the iris. Occasionally they hypertrophy to varying degrees; in severe cases the pupil may be partially or completely obstructed, particularly in bright light. However, vision is affected rarely, and no treatment is required.

Uveal cysts

Cysts are common incidental findings and rarely affect vision.

Clinical signs, diagnosis and treatment

- Smooth, spherical structures of varying size originating from the pigmented posterior iris epithelium and may be attached to pupillary edge or granula iridica.
- Amount of pigment varies, but most can be transilluminated with a focal light source, allowing differentiation of cysts from granula iridica hypertrophy or neoplasia.
- Occasionally cysts can become free floating in the anterior chamber; their size may fluctuate, or they may rupture.

No treatment is required.

Iris hypoplasia

Zones of hypoplasia involving the mid-dorsal stroma of the iris are not uncommon in heterochromatic or sub-albinotic pale blue irides, most often in Welsh ponies. Affected areas bulge into the anterior chamber, the dorsal margin may be distorted and fundus reflection may be visible through the lesion.

Trauma

Trauma to the iris can occur from direct injury, whiplash-type injury or penetrating corneal lesions. Whiplash injuries occur consequent to blunt trauma or sudden and violent head movement.

Clinical signs and diagnosis

Clinical findings are usually diagnostic, including:

- Penetrating corneal lesions are usually obvious with prolapse and incarceration of pigmented iris through the lesion and variable accompanying inflammatory exudate.
- Whiplash injuries result in partial or complete avulsion of the granula iridica that hangs down in front of the pupil or floats freely in the anterior chamber.
- Foreign bodies can be difficult to visualize as corneal oedema and/or anterior uveitis may be present.
- Hyphaema is common.
- Signs of anterior uveitis may be present.

Treatment

- Irrespective of the aetiology of iris trauma, medical treatment for anterior uveitis should be commenced.
- Treatment of iris prolapse is described in 'Traumatic keratitis'.
- Foreign bodies involving the iris should generally be removed; however it is important to determine whether injury to the lens has occurred as leakage of lens protein can result in phacoclastic uveitis which is impossible to treat medically.
- Trauma to the granula iridica usually does not require specific treatment.
- Hyphaema usually resolves without treatment.

Iridocyclitis (uveitis)

Iridocyclitis has many potential causes (Table 12.4) and is the most common cause of blindness in horses. Importantly, in some horses repeated episodes of iridocyclitis occur, termed equine recurrent uveitis (ERU, moon blindness, periodic ophthalmia, recurrent iridocyclitis). In many cases the cause remains undetermined, and the likelihood of ERU unpredictable. Of the organisms implicated in the development of ERU, *Leptospira* serovars have received the most attention; in particular, serovar *pomona* in the USA, serovars *pomona*, *grippotyphosa*, *autumnalis* and *ictero-haemorrhagiae* in Europe and serovar *serjoe* in the UK. Association between *Leptospira* serovars and ERU has been based on detection of antibodies in blood or the vitreous, culture of organisms from the aqueous/vitreous or detection of leptospiral DNA in aqueous humor.

Iridocyclitis may also accompany primary corneal disease, including ulcerative keratitis, corneal stromal abscess and non-infectious keratopathies. Accurate diagnosis is vital as resolution of iridocyclitis can

Table 12.4 Causes of iridocyclitis in horses

Traumatic injury	Blunt Penetrating
Keratitis	Non-infectious Infectious
Phacoclastic uveitis	
Ocular/Systemic infection	
Bacterial	*Leptospira interrogans* serovars *Rhodococcus equi* *Streptococcus* spp. *Brucella* spp. *Salmonella* spp. *Borrelia burgdorferi*
Viral	Equine herpes virus type 1 Equine influenza Equine viral arteritis virus
Parasitic	*Onchocerca cervicalis* *Toxoplasma* spp. *Strongylus* spp.
Neoplasia	
Systemic inflammation	Endotoxaemia Septicaemia
Abscess	

be hastened with appropriate treatment for the underlying condition, and corticosteroids are contra-indicated when corneal ulceration and infection are present. Phacoclastic uveitis is a severe and refractory inflammation subsequent to leakage of lens protein into the anterior chamber.

The pathophysiology of iridocyclitis is complex and likely influenced by aetiology, immune-mediated responses and genetic factors. Definition of risk factors and investigation of the complex aetiopathogenesis of iridocyclitis/ERU will continue as searches for a greater understanding and more effective treatments for the disease continue. The aetiopathogenesis of ERU likely involves a non-specific immune-mediated process resulting in persistent or recurrent intra-ocular inflammation. Infectious agents, antigens or antigen-antibody complexes are thought to be localized in the vitreous or uveal tract, inciting periodic inflammation. Over time, sensitized T_H1 lymphocytes in the uveal tract mount responses specifically to the original antigen or non-specifically to ocular auto-antigens (molecular mimicry) or other exogenous antigens. Irrespective of the underlying antigen(s), the predominance of T_H1 lymphocytes is consistent with a delayed cell-mediated hypersensitivity response.

Classification of uveitis has been made on the basis of aetiology, anatomical involvement (anterior, posterior or panuveitis), type of reoccurrence (classic or insidious) and stage of chronicity (acute, quiescent, chronic or end-stage). However, such divisions can be difficult to define clinically, and the practical usefulness is limited. Of note, however, is the tendency of low-grade iridocyclitis that doesn't manifest as overt ocular pain to develop in certain breeds, including Appaloosas and draft horses.

Clinical signs and diagnosis

Clinical findings of iridocyclitis are variable, often dependent on disease chronicity and whether any pre-existing ocular disease is present. Diagnosis is based on clinical signs as collection of aqueous or vitreous humor for detection of aetiological agents is seldom practical unless vitrectomy is performed. One or both eyes may be affected and predisposed to ERU.

Acute iridocyclitis

Acute disease may feature variable combinations of the following:

- Signs of anterior segment pain: blepharospasm, lacrimation and photophobia.
- Miosis: a characteristic finding and is often marked.
- Impaired vision.
- Corneal oedema (Figure 12.14).
- Corneal vascularization (Figure 12.14).
- Conjunctival and episcleral hyperaemia.
- Palpebral oedema and chemosis.
- Reduced intra-ocular pressure (occasionally increased intra-ocular pressure is present).
- Dullness, change in colour and hyperaemia of the iris.
- Early synechiae formation.
- Cloudiness of the anterior chamber from aqueous flare, fibrin deposition, hypopyon or hyphaema (Figure 12.14).
- Peripapillary oedema.
- Vitritis.
- Optic neuritis.

Chronic/end-stage iridocyclitis

Chronic iridocyclitis is often reflected by structural changes and development of vision-threatening sequelae:

- Corneal vascularization and refractory corneal oedema.
- Calcific band keratopathy.

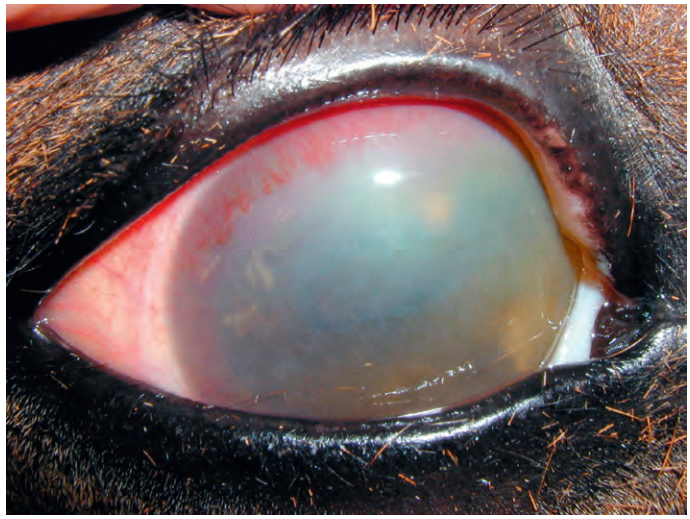


Figure 12.14 Uveitis. Oedema and vascularization of the cornea and hypopyon are present.

- Rupture or atrophy of the granula iridica.
- Posterior synechiae.
- Iris hyperpigmentation and loss of detail.
- Irregular pupil border.
- Impaired/loss of iris mobility (fixed pupil).
- Pigment deposition on the anterior lens capsule (iris 'rests').
- Cataract formation.
- Lens subluxation/luxation.
- Vitreal liquefaction and 'floaters' (inflammatory products).
- Vitreal fibrous traction bands.
- Retinal detachment.
- Peripapillary chorioretinopathy.
- Optic atrophy.
- Impaired or loss of vision.
- Phthisis bulbi.
- Hydrophthalmos/glaucoma (rarely).

Treatment and prognosis

Careful assessment is required prior to commencement of treatment, as any underlying condition requires specific management. Because vision loss is possible, particularly in ERU, immediate therapy is warranted. The goals of treatment are to control/reduce inflammation, reduce discomfort and prevent permanent damage to the eye and vision loss. Another goal is the prevention of more episodes of iridocyclitis.

Management practices

- Provision of an environment free of irritants including dust and insects.
- Reduce sun-exposure (darkened stable).
- Reduction of ocular trauma; removal of exposed nails, hooks etc. in stables and low tree branches in pastures.
- The placement of an indwelling subpalpebral/nasolacrimal lavage system may facilitate administration of topical treatments.

Medical treatment

- Topical mydriatics-cycloplegics (Table 12.1) to achieve mydriasis and reduce ocular pain (from ciliary spasm). They should be

administered only as required to maintain mydriasis to avoid development of colic. A poor response to mydriatics is suggestive of marked intra-ocular inflammation, which must be addressed.
- Topical anti-inflammatory medications (Table 12.1) with good penetration to the intra-ocular tissues. Topical administered corticosteroids are of great benefit; however, they should be used only when the corneal epithelium is intact. Alternatively, or when the epithelium is breached, topical NSAIDs can be used judiciously (as they delay corneal re-epithelialization).
- Systemic anti-inflammatory therapy (Table 12.3) is the most potent method of control of intra-ocular inflammation and pain and can compliment topical therapy. Flunixin meglumine, dexamethasone or prednisolone are effective in acute cases; however, combination therapy should be avoided due to the increased risk of gastro-intestinal side-effects. Oral flunixin meglumine or phenylbutazone are useful alternatives when intense signs of disease have regressed and ongoing anti-inflammatory therapy is required.
- Subconjunctival injection of corticosteroids (Table 12.2) is useful in severe cases. Triamcinolone acetamide is the steroid of choice as it does not result in granuloma formation. Limitations include an inability to stop therapy once commenced and predisposition to infectious keratitis.
- Topical antimicrobial therapy, when indicated.
- All anti-inflammatory therapies, irrespective of route, should be tapered in relation to clinical response.

Surgical procedures

The principal limitation of all conventional medical treatments is that they do not prevent reoccurrence of iridocyclitis. Recently, two procedures have been developed to provide long-term control of ERU and prevent ongoing ocular damage.

- Intravitreal cyclosporine. A sustained-release device. Cyclosporine A (CsA) is a valuable immunomodulator for intra-ocular inflammation; however, poor ocular penetration precludes topical application. Sustained intra-ocular concentrations can be obtained by intravitreal or suprachoroidal implantation of a sustained-delivery device. Success rates have been promising, but availability is currently limited.
- Core vitrectomy. Removal of the vitreous can be performed to remove T cells, antigens and organisms from the vitreous. Goals are to prevent recurrent inflammation and ocular pain and preserve the globe, but preservation of vision is not expected.

The prognosis for iridocyclitis is guarded to poor, particularly for cases of ERU. An accurate prognosis for ERU is not possible due to the unpredictability of timing and severity of further episodes, and owners should be counselled accordingly. Prognosis for iridocyclitis secondary to corneal disease may be more favourable, particularly if the underlying condition can be treated successfully. However development of ERU remains possible.

Neoplasia

Uveal tract neoplasia is rare; melanoma is the most common, and grey horses are most frequently affected. Most melanomas involve the iris; however any portion of the uveal tract can be involved. Melanomas usually present as a dark pigmented mass that slowly enlarges in the anterior chamber and distorts the iris and pupil. Focal corneal opacity occurs if the mass contacts the endothelium. Signs of ocular pain and discharge are absent. Most do not exhibit malignancy and sector iridectomy or enucleation is usually curative.

12.9 GLAUCOMA

The glaucomas (pathological increase of intra-ocular pressure) are an uncommon group of diseases that result from an imbalance between aqueous humor production and drainage. Aqueous humor is produced by the ciliary body, flows through the pupil into the anterior chamber and drains circumferentially through the iridocorneal angle (conventional pathway). In addition, an extensive and low-resistance uveoscleral (unconventional) pathway exists and may be the most important method of drainage in the horse and likely explains the low risk of glaucoma in Equidae.

Congenital glaucomas occasionally seen in foals are the result of developmental anomalies of the iridocorneal angle (goniodysgenesis) and may occur with other ocular congenital disorders. Primary glaucomas are not associated with any overt ocular anomaly, are often bilateral and possibly heritable and are exceedingly rare in horses. Most cases of glaucoma in horses are secondary to iridocyclitis, intra-ocular neoplasia or lens luxation.

Clinical signs and diagnosis

Signs are subtle in many cases, and early disease is often missed due to a low index of suspicion. Signs include:

- Mild ocular pain (occasionally is obvious).
- Mydriasis.
- Corneal oedema and/or striae.
- Subluxation/luxation of the lens.
- Synechiae.
- Decreased/absent menace response and PLR.
- Shallow anterior chamber.
- Decreased vision/blindness.
- Mild to moderate iridocyclitis.
- Hydrophthalmos/buphthalmos.
- Vitreal inflammation/opacities/liquefaction.
- Retinal degeneration and optic nerve cupping.

Diagnosis can be achieved through careful interpretation of clinical findings and documenting increased intra-ocular pressure.

Treatment and prognosis

Response to medical treatment and prognosis for vision are usually poor. Effective treatment of glaucoma in horses is difficult to achieve, contributed by an incomplete understanding of disease aetiopathogenesis. Glaucoma in Appaloosas is aggressive and difficult to control. Aims of treatment are to maintain vision, reduce ocular pain and reduce production and/or increase drainage of aqueous humor. If iridocyclitis is present, anti-inflammatory treatment should be instituted. Atropine may increase unconventional drainage; however, increases in IOP occur in some horses, and this drug should be avoided in the absence of iridocyclitis. Pilocarpine and synthetic prostaglandins (lantanoprost) may decrease IOP; however they may exacerbate ocular inflammation. Most success has been achieved using drugs that reduce aqueous humor production, including topical (dorzolamine) or systemic (acetazolamide) carbonic anhydrase inhibitors, topical β-blockers (timolol) or combinations (dorzolamide and timolol). Surgical treatment (cyclophotoablation) may be of value to control IOP in medically refractory glaucoma. Blind glaucomatous eyes do not require treatment provided pain or excessive globe size is not present; if necessary, enucleation or cyclophotoablation/cyclocryoablation can be performed.

12.10 LENS

Disorders of the lens can be either congenital or acquired. Because the horse lens is large, small refractile errors are of no significance. Many of these minor opacities are derived from remnants of the hyaloid system and tunica vasculosa lentis; occasionally these remnants result in an obvious opacity, e.g. focal capsular cataract at the attachment of a persistent papillary membrane (PPM). While equine nuclear sclerosis has yet to be described definitively, old horses often have yellow discolouration of the lens.

Cataract

Opacities of the capsule and/or cortex are the most common lens abnormality in horses (Figure 12.15). Broadly, cataracts can be either:

- Developmental (including congenital forms).
- Acquired/secondary.

Cataracts may also be described on the basis of:

- Cause: e.g. hereditary, inflammation, trauma.
- Age of onset: congenital, juvenile, senile.
- Location: capsular, cortical, nuclear, polar, equatorial, posterior, anterior (Figure 12.16).
- Stage of development: incipient, immature, mature, hypermature.

Some have the propensity to progress with implications for vision due to increases in size and/or density. Conversely, other cataracts are non-progressive, and some congenital lesions may become less obvious over time. Interpretation of the biological characteristics of a cataract can be difficult; with few exceptions it is impossible to predict the progress with a single examination. The impact on vision is variable; many cataracts do not cause appreciable visual deficits. However, dense cataracts located in the visual axis and complete cataracts are expected to reduce or abolish sight.

- Developmental cataracts are the most common form of cataracts in horses, and most are congenital. Most developmental lesions are related to a stage of embryogenesis, and many are non-progressive. Disruption of vision is variable. Developmental lesions include extralenticular (e.g. Mitterdorf's dot, PPM remnant), capsulolenticular and lenticular opacities. Congenital cataracts occur in several breeds including Arabians, Appaloosas, Thoroughbreds, Quarter horses, Belgian Draft horses and Morgans. A hereditary basis has been confirmed in Morgan and Belgian horses and is suspected in Quarter horses. Other postulated causes of congenital cataracts are in utero infection, malnutrition, exposure to toxins and radiation. Congenital cataracts may occur with other ocular defects e.g. aniridia in Belgian horses and anterior segment dysgenesis and retinal dysplasia in Rocky Mountain Spotted horses.
- Acquired/secondary cataracts occur when the initiating factor for development arises from outside of the lens. The most common is uveitis which may result in capsular and cortical opacities of varying size and density. When a cataract is found, both eyes should be examined carefully for evidence of active or previous inflammation. Cataracts associated with inflammation are not suitable for surgical treatment. Other causes include trauma, retinal detachment, glaucoma and intraocular neoplasia. Very rarely, systemic disease, UV light, ionising radiation, metabolic disease and ingested toxins are incriminated. Acquired cataracts are potentially progressive, dependent on the persistence or recurrence of the underlying aetiological factor.

Diagnosis

Detection and classification of the opacity provides a diagnosis of a cataract.

Treatment and prognosis

Currently, no medical treatment for cataracts is available. If iridocyclitis is present, appropriate treatment may prevent or delay cataract progression. Mydriatic therapy may also improve vision in eyes with focal cataracts. Cataract surgery is the only specific treatment option; however, careful patient selection is paramount, and post-operative complications may occur. Complete congenital cataracts unassociated with additional ocular disease are most suitable for surgery, although cataracts of traumatic and post-inflammatory origin have been successfully treated.

Figure 12.15 Anterior polar cataract.

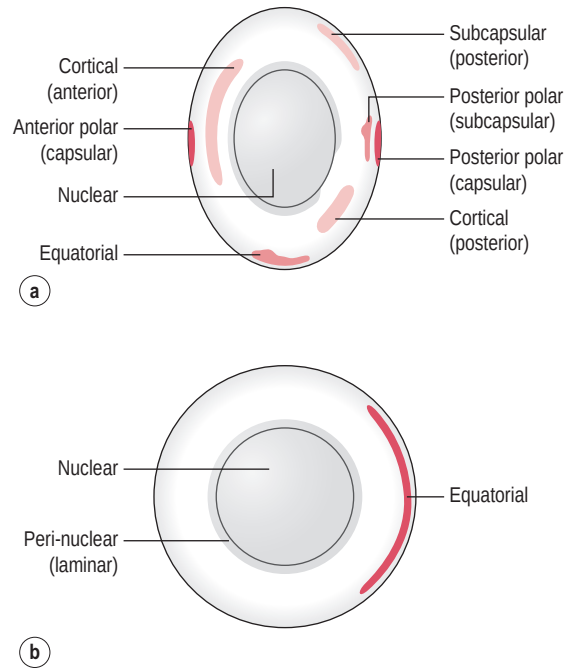


Figure 12.16 Schematic diagram of possible locations of cataracts in horses, in cross sectional (a) and frontal views (b).

Subluxation and luxation of the lens

Primary lens luxation has not been reported; however, anterior or posterior luxation/subluxation is an occasional sequel to uveitis, trauma or glaucoma or as a component of multiple ocular anomalies.

12.11 VITREOUS

The vitreous body is a translucent gel that occupies the majority of the ocular volume. The embryology of vitreous development comprises of three stages. Adult vitreous is formed from the third stage (tertiary vitreous). In the foal vestiges of the primary vitreous (hyaloid vessel system) can be identified; during post-natal life hyaloid regression occurs, and only Cloquet's canal and Mitterdorf's dot remain. Poor cohesion of the vitreous gel in adults can result in mobility of the vitreous components ('vitreal floaters') which are of no clinical significance.

Congenital vitreal anomalies

Persistent hyaloid vasculature and/or primary vitreous are uncommon findings in horses, although atrophying remnants of the hyaloid vessels are found frequently in foals. Diagnosis is by ophthalmic examination, and no treatment is available.

Acquired diseases of the vitreous

Inflammation

Inflammatory detritus may accumulate in the vitreous consequent to uveitis.

Clinical findings and diagnosis

- Generalized yellow discolouration ('haze') is common in acute stages.
- Cellular and/or proteinaceous debris.
- Progressive organization of inflammatory tissue may result in vitreoretinal detachments.

Treatment and prognosis

- Treatment is directed towards control of intraocular inflammation using systemically and locally administered corticosteroids.
- Prognosis is dependent on resolution of inflammation and development of sequela; vitreoretinal detachment causes permanent vision loss.

Intravitreal haemorrhage

Haemorrhage into the vitreous originates from retinal or uveal vessels as a result of inflammation, trauma or rarely neoplasia. Even if bleeding is controlled, clearance of vitreal blood is slow, often incomplete and an organizing haematoma can precipitate vitreoretinal detachment. Apart from addressing the underlying condition, there is no treatment, and the prognosis is guarded to poor.

Synchysis scintillans

This is an occasional finding resulting from small refractive crystals of cholesterol that float freely in liquefied vitreous and settle under the influence of gravity. The condition is of no clinical significance, and there is no treatment.

Asteroid hyalosis

This is a rare condition, found in old horses, appearing as refractile inclusions that remain suspended in the vitreous body. There is no treatment.

Neoplasia

Rarely, cellular debris in the vitreous is the result of neoplastic exfoliation into the posterior segment.

12.12 FUNDUS

The fundus comprises the neurosensory retina (NSR), choroid and the optic nerve head. There is considerable diversity of the appearance of normal fundus, and an appreciation of normal variants is important. In most horses when fundic abnormalities are detected, the role of the veterinarian is primarily to determine whether the disease is likely to impede vision or progress and ultimately whether the horse is suitable for a selected purpose. Normal vision is reliant on the integrity of all components of the retina; hence dysfunction of any component will influence sight. However, horses often adapt to vision loss, particularly if gradual, and even horses with extensive and/or bilateral fundic disease may not have signs referable to vision loss reported by the owner. Further, posterior segment disease does not result in pain, influencing detection. While accurate determination of vision is difficult and subjective, normal retinal vasculature is suggestive of a functional retina. Conversely, attenuation of vessels indicates retinal disease with likely visual deficits. Occasionally horses are presented for vision loss; however, most fundus disease is detected during examination for insurance or prior to purchase requirements. Interpretation of any abnormality must include any effect on present and future use and safety of the horse and rider, potential for lesion progression and effect on resale and insurance.

Normal variants of fundus appearance

Albinism and partial albinism

Partial albinism (subalbinism) is most common in animals with a light coat colour, and involves pigment dilution of varying degrees involving the non-tapetal fundus; it may be localized (e.g. peripapillary) or generalized. The pigment dilution permits visualization of the underlying choroidal vasculature. In addition, hypoplasia of the tapetum may be present, resulting in a reddening of the tapetal refection. True albinism occurs only in albino horses and result in a non-pigmented and atapetal fundus that highlights the choroidal vessels spectacularly against the scleral background.

Tapetal colour

The colour of the tapetum varies with coat colour; light-coloured and chestnut horses usually have a yellow tapetum, while dark-coloured horses tend to have a blue to aquamarine tapetum.

Optic disc shape and retinal blood vessels

Variants of optic disc shape are common and include rounding and stepping of the margin. Occasionally the fibrous meshwork of the lamina cribrosa is prominent, leading to a fascicular or granular appearance to the disc surface. 30–60 blood vessels radiate from the disc margin, apart from the ventral aspect. Variability in the diameter

of the vessels may occur, and in some horses a single or bifurcating vessel emerging from the centre of the disc can be observed.

Variations of retinal pigmentation

Hyperpigmentation, hypopigmentation of the fundus or both are commonly observed:

- The peripapillary region is involved commonly, and variations include:
  - a complete 'halo' of demarcated non-pigmentation.
  - foci of depigmentation adjacent to the ventral pole of the disc.
  - irregular hyperpigmentation involving the ventral margin, extending laterally and medially and occasionally circumpapillary.
- Focal and linear regions of pigmentation can occur at the tapetal/non-tapetal junction.
- Irregular focal and linear hyperpigmentation in the tapetal fundus.
- Well-demarcated single or multiple foci of pigment proliferation in the tapetal fundus (choroidal/chorioretinal naevi) of dark-coloured and grey horses.

Variations of myelination

The axons of the ganglion cells are usually non-myelinated; however, in some horses myelination occurs adjacent to the optic disc. Appearance may vary from subtle grey striae radiating from the disc to a pronounced 'sunburst' conformation.

Miscellaneous variants

- Remnants of the hyaloid artery at the centre of the optic disc.
- Choroidal veins entering the vortex drainage system are visible in subalbinism.
- Choroidal capillaries may be prominent in subalbinism, while in horses without pigment dilution they manifest as the 'stars of Winslow'.

Pathological variants of fundus appearance

Coloboma

Congenital discontinuity of ≥1 fundic layer is seen occasionally and is catagorized as typical (within the plane of the optic fissure) or atypical (no anatomical relationship with the optic fissure). Colobomas may be single or multiple, usually involve an individual structure (retina, choriod or sclera) and usually have no effect on vision. Rarely, colobomas are extensive and involve multiple structures usually in combination with other defects (e.g. anterior segment dysgenesis, microphthalmos), and vision is markedly compromised.

Retinal detachment

Separation of the inner NSR from the outer retinal pigmented epithelium (RPE) layer can be congenital or acquired. Congenital retinal detachment is a rare form of unilateral or bilateral blindness in foals. Acquired retinal detachment is caused by choroidal exudation/transudation, dissection of the vitreous between the NSR and RPE or traction from vitreoretinal membranes. Underlying conditions include blunt or penetrating trauma, uveitis and neoplasia.

Retinal haemorrhage

Linear intraretinal or discrete subretinal haemorrhages are occasional findings in neonatal foals. While an association with perinatal asphyxia syndrome (PAS) has been suggested, retinal haemorrhages may also be found in apparently normal foals. Subjectively, haemorrhages in foals with PAS are more extensive in size and number. Retinal haemorrhages in foals are incidental findings and resolve without treatment over several days. Haemorrhages are rarely observed in adult horses and causes include trauma, coagulopathies and systemic disease.

Congenital stationary night blindness

This is a non-progressive vision deficit, usually evident in dim light. The condition is likely to result from a disorder of neurotransmission in the NSR. The disorder affects predominantly Appaloosas and is thought to be hereditary. Occasionally other breeds (Paso Fino, Thoroughbred) are affected. The fundus has a normal appearance; electroretinography is required to confirm the diagnosis. There is no treatment, and affected horses should not be used for breeding.

Degenerative retinopathies

Retinal atrophy

Localized atrophy is common and may result from numerous causes including uveitis, retinal ischaemia (ligation of carotid arteries, embolism, marked hypovolaemia) and trauma. Atrophy is most evident in the tapetal fundus, where the loss of retinal thickness results in increased or altered tapetal reflectivity. Ectopic pigment proliferation or depigmentation of the RPE may occur. Vision deficits may not be apparent in cases of localized atrophy. Generalized retinal atrophy is rare, and affected eyes are blind.

Pigmentary retinopathies

Damage to the RPE affects the function of the NSR and results in visual deficits. Lesions are most evident in the junctional zone of non-tapetal and tapetal fundus and peripapillary. Manifestations include:

- Senile retinopathy is a common condition in old horses. Extensive loss of RPE with coincident irregular linear regions of hyperpigmentation in the non-tapetal fundus is characteristic. The aetiopathogenesis is unknown; however, age-related impairment of choroidal vasculature has been implicated. The condition is potentially progressive, and although vision deficits are likely, such impairment is reported rarely by owners.
- Horses with equine motor neurone disease often have a distinctive mosaic pattern of pigment loss and hyperpigmentation from oxidative injury. Lesions may be confined to the tapetal-non-tapetal junction or extend to involve the entire fundus. Tapetal hyper-reflectivity is observed in some affected eyes.
- Localized foci of RPE depigmentation that expose the choroid or tapetum are occasional findings. Lesions are non-progressive, the NSR is usually unaffected and vision deficits are not evident.
- Extensive pigmentary retinopathy consisting of pigment loss and concurrent areas of hyperpigmentation. Vision loss is expected due to NSR dysfunction. In some eyes, the pattern of hyperpigmentation is reticular or mosaic.

Retinal detachment

See above.

Ischaemic retinopathy

Ischaemia of the retina and optic disc results in a generalized and progressive retinopathy and neuropathy. The condition is irreversible and results in blindness. Initially ophthalmoscopic appearance is

normal; however, with time extensive loss of pigment and focal hyperpigmentation of the retina and atrophy of the optic disc occur. Causes include:

- Head trauma.
- Embolism.
- Marked hypovolaemia (especially haemorrhagic shock).
- Ligation of carotid arteries for treatment of guttural pouch mycosis.

Chorioretinopathies

Inflammatory conditions of the retina and choroid are common and important causes of fundus disease. However, detection of active inflammation is achieved rarely, and most often, chronic, inactive lesions are observed. Active lesions are often subtle and include poorly defined areas of exudation at the vitreoretinal interface.

Chronic, inactive chorioretiopathies

These are common incidental findings. Appearance varies and may include depigmentation, proliferation of pigmentation, tapetal hypo/hyper-reflectivity and abnormalities of the retinal or choroidal vasculature. Not all lesions are progressive, and any affect on vision is influenced by lesion extent, location and attenuation of the retinal and choroidal vasculature. Broadly, lesions can be allocated to one of three categories on the basis of appearance:

1. Focal chorioretinopathy ('bullet-hole' lesions): well defined, small areas of depigmentation (with/without a central area of hyperpigmentation) located in the non-tapetal fundus. Lesions may have an apparent random distribution or may be configured linearly medial, lateral or ventral to the optic disc. Focal lesions are very common, and small numbers are unlikely to result in vision loss; however, more extensive involvement of the fundus may result in vision deficits.
2. Peripapillary chorioretinopathy ('butterfly' lesions): an occasional incidental finding that is often unilateral and non-progressive. They usually appear as well-demarcated areas of depigmentation and peripheral hyperpigmentation adjacent to the medial and lateral margins of the optic disc. Occasionally, lesions may extend to include the ventral or entire peripapillary fundus, or isolated 'islands' of chorioretinopathy may occur. The clinical significance of peripapillary lesions without evidence of attenuated retinal vasculature is difficult to interpret, but when vasculature damage is present, vision deficits are expected.
3. Diffuse chorioretinopathy is rare; when present, marked deficiency in vision is likely.

12.13 DISORDERS OF THE OPTIC NERVE

Optic atrophy

Optic atrophy results from ischaemic injury to the nerve consequent to various insults including trauma, space-occupying lesions of the orbit, optic nerve, meninges or midbrain, posterior segment inflammation and glaucoma. The optic disc is pale, granular due to prominence of the lamina cribrosa and demonstrates marked attenuation of the retinal vessels. There is no treatment.

Optic neuritis

Inflammation of the optic disc is detected rarely; however, subclinical disease may occur. Optic neuritis has been associated with a variety of systemic diseases, including infectious and immunological (e.g. ERU) disorders. Mild disease may be subclinical and remain undetected; however marked inflammation usually results in vision loss. During active disease the optic disc appears hyperaemic, poorly defined, and exudation into the vitreous may occur. An investigation for any underlying cause should be performed, and treatment should be directed towards any specific aetiological agent identified and suppression of the inflammatory process (systemically administered).

Traumatic optic neuropathy

Traumatic injury to the optic nerve, especially by avulsion, tearing or compression, results in acute blindness. Most cases occur in horses that fall over backwards and sustain trauma to the occiput. The prognosis for vision in most cases is hopeless, despite aggressive anti-inflammatory therapy. Immediately after injury the optic disc may appear normal; however, papilloedema and hyperaemia of the disc and extrusion of myelin into the vitreous may be evident. With time the appearance is characteristic of optic atrophy.

Proliferative optic neuropathy

Proliferative, non-neoplastic lesions of the optic disc represent glial cell responses to various insults and can be focal or generalized. The reactive glial cells contain lipid-filled cytoplasmic vesicles.

- Focal lesions; lobulated mass at optic disc margin. Lesions are often an incidental finding in old horses, non-progressive, and vision appears unaffected.
- Generalized lesions; extensive lobulated glial cell responses that cover the entire optic disc. Most cases are consequent to marked haemorrhage or trauma. Blindness invariably results, and there is no treatment.

Optic nerve neoplasia

Neoplasia of the optic disc is rare. Astrocytoma is a small, non-progressive, discrete tumour that can be difficult to differentiate from focal proliferative optic neuropathy. Vision is not affected. Medulloepithelioma and neuroepithelioma are very rare causes of blindness. These tumours can result in exophthalmos and should be treated with exenteration of the orbit; however, prognosis is poor if invasion into the calvarium has occurred.

12.14 DIFFERENTIAL DIAGNOSES FOR COMMON PRESENTING COMPLAINTS

1. Sudden onset of ocular pain

1. Eyelid trauma.
2. Orbital trauma.
3. Penetrating or blunt injury to globe.
4. Foreign body (sub-palpebral/intra-ocular).
5. Entropion.
6. Keratitis (ulcerative/non-ulcerative).
7. Uveitis.
8. (Blepharo)conjunctivitis.

2. Gradual onset of ocular pain

1. Keratitis; especially non-ulcerative forms.
2. Glaucoma.
3. Blepharitis and meibomianitis.

4. Insidious forms of uveitis.
5. Orbital cellulitis/abscess.
6. KCS.
7. Endophthalmitis and panophthalmitis.

3. Ocular discharge

1. Orbital and globe injuries.
2. Orbital cellulitis/abscess.
3. Reduced orbital size; microphthalmos, phthisis bulbi.
4. Conjunctivitis/blepharitis.
5. Foreign body (sub-palpebral/intra-ocular).
6. Neoplasia (palpebral/conjunctival/corneal).
7. Congenital disorders of the nasolacrimal drainage system.
8. Acquired stenosis/blockage or inflammation of the nasolacrimal drainage system.
9. Keratitis.
10. Uveitis.
11. Endophthalmitis and panophthalmitis.

4. Ocular opacity

1. Congenital corneal disorders (microphthalmos, dermoid, sclerocornea, leukoma).
2. KCS.
3. Exposure keratopathy and evaporative keratopathy.
4. Corneal oedema (idiopathic, traumatic, associated with ulcerative keratitis or uveitis).
5. Non-inflammatory keratopathies (e.g. bullous keratopathy, linear keratopathy, pigmentation, retrocorneal membranes).
6. Corneal striae (idiopathic, secondary to glaucoma).
7. Keratitis (e.g. traumatic, ulcerative, viral, corneal stromal abscess, eosinophilic, immune-mediated, endotheliitis).
8. Corneal/perilimbal neoplasia (SCC most common).
9. Parasitic keratoconjunctivitis (e.g. onchoceriasis).
10. Corneal mineralization (often secondary to uveitis).
11. Hydrophthalmos secondary to chronic glaucoma.
12. Phthisis bulbi.
13. Abnormal aqueous constituents (hypopyon, hyphaema, aqueous flare [protein], keratic precipitates, neoplastic cells).
14. Lens luxation.
15. Cataract.
16. Glaucoma (corneal oedema, striae, neovascularization or pigmentation).
17. Congenital vitreal disorders (persistent primary vitreous, hyperplastic primary vitreous, vitreal membranes, vascular remnants).
18. Intravitreal inflammatory detritus.
19. Intravitreal haemorrhage.
20. Non-inflammatory vitreal opacities (synchysis scintillans, asteroid hyalosis).
21. Retinal detachment.

5. Sudden onset of unilateral blindness

1. Globe trauma (blunt or perforating).
2. Orbital trauma.
3. Skull trauma (e.g. basisphenoid/basioccipital fracture, optic nerve avulsion).
4. Cerebral trauma.
5. Inadvertent carotid injection.
6. Uveitis.
7. Intra-ocular haemorrhage (rare).
8. Retinal detachment (rare).
9. Optic neuritis (rare).

6. Sudden onset of bilateral blindness

1. Skull trauma.
2. Cerebral trauma.
3. Uveitis (rarely a cause of bilateral blindness except in foals with septicaemia).
4. Optic neuritis (rare).

7. Exophthalmos

1. Orbital trauma (displaced fracture, retrobulbar/extra-orbital haemorrhage or oedema).
2. Retrobulbar space-occupying lesion (cellulitis, abscess, neoplasia, hydatic cyst).
3. Extra-orbital space occupying lesion (ethmoidal haematoma, neoplasia).
4. Paranasal sinus disease (sinusitis, cyst, neoplasia, fracture).

8. Endophthalmos

1. Dehydration.
2. Loss of retrobulbar adipose tissue.

9. Asymmetry in globe size

1. Microphthalmos.
2. Unilateral phthisis bulbi.
3. Unilateral hydrophthalmos/glaucoma.

10. Eyelid/periocular swelling

1. Blepharitis (allergic, solar, bacterial, parasitic, meibomianitis).
2. Eyelid abscess.
3. Haematoma.
4. Chemosis.
5. Eosinophilic granuloma.
6. Prolapse of orbital fat pad.
7. Neoplasia (SCC and sarcoid are most common).
8. Amyloidosis.

FURTHER READING

Barnett KC, Crispin SM, Lavach JD, Matthews AG (2004) Equine ophthalmology: an atlas and text. Elsevier, St Louis

Cutler TJ (ed) (2004) Updates in ophthalmology. Veterinary Clinics of North America, Equine Practice 20:2

Gelatt KN, Brooks DE (eds) (2000) Veterinary ophthalmology. Equine Ophthalmology (special issue)

Gilger BC (ed) (2011) Equine ophthalmology, 2nd edn. Elsevier, St Louis

Matthews A, Gilger B, Hughes K (eds) (2010) Equine ophthalmology 111. Equine Veterinary Journal Supplement 37

Olliver FJ (2005) Medical and surgical management of melting corneal ulcers exhibiting hyperproteinase activity in the horse. Clinical Techniques in Equine Practice 4:50–71

Chapter 13

Dermatology

Linda J. Vogelnest

CONTENTS

13.1 Diagnostic approach to skin diseases 261
13.2 Skin diseases characterized by pruritus (itch) 263
Ectoparasitic skin diseases characterized by pruritus 264
Endoparasitic skin diseases characterized by pruritus 265
Insect irritation 266
Hypersensitivities 266
13.3 Skin diseases characterized by alopecia, scaling, and/or crusting 268
Infectious skin diseases characterized by alopecia, scaling, and/or crusting 268
Immune-mediated skin diseases characterized by alopecia, scaling, and/or crusting 270
Miscellaneous skin diseases characterized by alopecia, scaling, and/or crusting 273
13.4 Skin diseases characterized by ulceration and crusting 274
Photosensitization 274
Subepidermal bullous diseases 274
Cutaneous asthenia 274
Viral diseases 274
13.5 Nodular skin diseases 275
Ulcerative and/or exudative nodular skin diseases 275
Nodular to diffusely swollen skin diseases 278
Nodular to papular skin diseases without ulceration 279
13.6 Skin diseases characterized by depigmentation 281
Vitiligo 281
Leucoderma/leucotrichia 281
Reticulated leucotrichia 281
Spotted leucotrichia 281
13.7 Congenital and developmental skin diseases 281
Aplasia cutis 281
Albinism and other congenital pigmentation disorders 281
Congenital cysts 282
13.8 Miscellaneous skin diseases 282
Purpura haemorrhagica 282
Equine Cushing's disease 282
Pastern dermatitis (greasy heal) 282
Further reading 282

13.1 DIAGNOSTIC APPROACH TO SKIN DISEASES

A logical and thorough diagnostic approach is as important for skin disease as for disease in other body organs. It begins with the collection of a thorough *history*, and a careful and complete *physical examination*. Formulation of a *prioritized differential diagnosis list* follows, facilitating logical ordering of *diagnostic tests* and/or *trials*. Consideration of historical and physical data, and results from tests and trials, will allow confirmation of a clear diagnosis in many cases. Repeat testing may be required when the primary disease is dynamic (i.e. waxes and wanes). The primary disease process may occasionally be transient and insignificant, and secondary changes become responsible for the clinical signs. Even when limited by finances, consideration of the most likely differentials and performance of some simple diagnostic tests will at the very least allow more targeted treatment.

History

A client questionnaire aids collection of a complete history, especially for complex or chronic cases. Important background information should include age, sex, breed, husbandry (bedding, rugging), feeding (including supplements), local environment, activity, animal contacts, familial details, and general health. Specific disease information should include seasonality, duration, initial lesion appearance/location, degree of pruritus, and therapies and response.

© 2013 Elsevier Ltd
DOI: 10.1016/B978-0-7020-2801-4.00013-4

Physical examination

The physical examination should be thorough and include the full body, and careful and complete skin examination, assessing:

- General body condition.
- Mucous membrane colour and abnormalities (e.g. petechiation, ecchymoses or ulcers).
- General skin and hair coat.
- Specific skin lesions, noting distribution (generalized, regional or localized), size, and morphology. Accurate terminology to describe lesions, and characterization as primary or secondary, is important. *Primary lesions* are caused by the primary disease process, and are the most useful diagnostic clues and sampling sites:
  - Papules (<1 cm diameter): small, solid, palpable elevations; common in horses, most frequently due to insect bites, parasitic mites, follicular infections (bacterial pyoderma, dermatophytosis).
  - Plaques: groups of coalescing papules or wider flat-topped lesions (>1 cm diameter).
  - Nodules (>1 cm diameter): larger solid elevations; common in horses; may be inflammatory (bacterial, fungal, sterile) or neoplastic.
  - Wheals ('hives'): small, transient circumscribed elevations, filled with oedematous fluid, that pit with digital pressure; may be localized, regional or generalized; common in horses (referred to as urticaria).
  - Pustule: small, circumscribed elevations, filled with purulent material; very rare in horses; most commonly associated with follicular infections (bacterial pyoderma, dermatophytosis) or pemphigus foliaceus.
  - Vesicle/bullae (<1 cm/>1 cm diameter): well-demarcated elevated lesions filled with clear fluid; rare in horses, associated with viral infections, thermal or chemical injuries, and some rare diseases including bullous pemphigoid.
  - Macule/patch (<1 cm/>1 cm diameter): well-demarcated flat non-palpable lesions of colour change; uncommon in horses, caused by depigmentation (e.g. vitiligo), hyperpigmentation (e.g. post-inflammatory), erythema or haemorrhage (e.g. purpura, petechiae).

Secondary lesions are caused by factors complicating the primary disease (e.g. self-trauma, secondary microbes), and are often more noticeable and of more concern to the owner than primary lesions, although less specific and less useful. Some lesions may be primary or secondary (e.g. alopecia, scaling, crusting). Secondary lesions include:

- Alopecia: hairloss; may be localized, regional, extensive, patchy, diffuse, well-demarcated (often primary, due to follicular diseases e.g. infectious: dermatophilosis, dermatophytosis, superficial bacterial pyoderma; or non-infectious: alopecia areata, telogen effluvium), or poorly demarcated (often secondary, most commonly associated with self-trauma).
- Scale: loose fragments of stratum corneum; may be greasy, white, yellow, adherent, non-adherent; indicates increased epidermal cell turn-over, mostly a non-specific inflammatory response.
- Erythema: redness; may be localized, regional, or diffuse; often from increased vascular engorgement, a non-specific inflammatory response.
- Crust: dried exudate, serum, pus, blood, cells on surface of skin; form over erosions and ulcerations.
- Epidermal:
  - collarettes: circular erythemic lesions with a peripheral rim of scale; usually remnants from the roof of pustules, vesicles or bullae.
  - Erosion/ulcer: partial/full thickness loss of surface epidermis.
  - Excoriations: linear erosions or ulcerations; typically caused by self-trauma.
  - Hyperpigmentation: increased pigmentation due to increased melanin deposition; mostly non-specific chronic inflammatory response.
  - Lichenification: thickening and hardening of skin, with exaggeration of superficial markings; non-specific chronic inflammatory response.
  - Scar: inelastic, often atrophied areas of skin with normal dermal structures replaced to varying degrees by fibrous tissue.

Differential diagnosis list

A complete history and physical examination allows formulation of a prioritized differential diagnosis list to guide the most appropriate diagnostic tests or trials to recommend. Information about disease prevalence in your local area is also important; and with all other clues being equal, the most common diseases are always more likely.

Diagnostic tests

Skin scrapings

Skin scrapings allow examination for external parasites, primarily mites. Scrapings should be superficial for surface mites (majority of species of mites affecting horses), collecting ample scale and surface debris over wide areas (e.g. 5 cm^2) and/or multiple sites to maximize yield of potentially sparse mites. Paraffin oil / mineral oil is placed on the skin, a scalpel blade (sharp or blunted; No. 10 or 20) is angled to gently scrape the skin surface, and oil and surface scale are transferred onto a glass microscope slide. Superficial scrapings do not need to produce surface bleeding. Areas with papules may give a higher yield of mites. A cover slip is placed on top, and samples examined microscopically with 4× or 10× lens (40× or 100× magnification). Deep skin scrapings are required for deep dwelling mites, especially demodex (very rare in horses). The skin should be squeezed and scraped repeatedly to produce capillary oozing, to ensure mites are extruded from the hair follicles where they reside. Samples should be examined as for superficial scrapings. Skin scrapings can be used to search for dermatophytes; although trichograms or tape impressions, which contain more hair shafts, are generally higher yield tests for animal dermatophytes.

Skin cytology

Dry skin lesions are best sampled with acetate tape (e.g. Scotch® tape, or plain, non-patterned clear tape), pressed sticky-side down onto lesional skin (tape impressions). Initial gentle cleansing with water, saline or 0.05% chlorhexidine scrub is important to clear surface debris from dusty coats; or multiple samples should be collected and initial samples discarded. Tape impressions are useful for evaluating superficial bacterial pyoderma, dry forms of dermatophilosis, and dermatophytosis. Direct impression smears are effective for evaluating moist lesions. After initial cleaning of surface debris with saline, a glass slide is pressed firmly onto the affected area. Special techniques may be required when some organisms are suspected (e.g. maceration of crusts in saline for *Dermatophilus* spp.). Nodular lesions can be sampled with fine needle aspirates; using a 10 ml syringe and 25–21 g hypodermic needle (smaller gauge in more vascular nodules), with gentle suction and needle redirection within the lesion. The aspirated sample (often in the hub of the needle) is gently sprayed onto a glass slide. Slides from impression smears and aspirates should be

air-dried and stained (e.g. Diff-quik®; Gram, Giemsa, or Methylene blue sometimes indicated). Tape impressions can be Diff-quik stained, although the author prefers a few drops of the blue Diff-quik stain alone under the tape, fixed at one end on a glass slide, with the stain gently smeared out using tissue paper to create a flat even surface for examination. Oil can be placed directly onto the tape surface. Aspirates, impression smears, and tape impressions should be examined for the presence of inflammatory cells, bacteria, fungi or neoplastic cells. Tape impressions can also be examined for broken hair shafts containing fungal hyphae and/or spores. Scanning with the 4x or 10x lens is important initially to identify areas of inflammatory cells. Oil immersion (1000×) is required for bacterial examination, and 40x will reveal larger fungi and cell morphology.

Trichogram

Hairs are plucked from the periphery of alopecic lesions and laid carefully onto 2 or 3 drops of paraffin oil on a glass slide. The proportions of hairs in telogen (rest) and anagen (active growth) phases can be estimated, important information in some follicular growth diseases (e.g. telogen defluxion). Hairs can be examined for shaft abnormalities (e.g. alopecia areata), parasites or fungal elements. Samples do not require clearing with potassium hydroxide (KOH), as the vast majority of veterinary dermatophytes are ectothrix fungi (live on the outside of hair shafts) unlike the common human dermatophytes (endothrix; living within hair shafts).

Microbial culture

Microbial isolation and antimicrobial sensitivity testing is often important for accurate treatment of deep microbial skin infections, particularly when more unusual or resistant bacteria (e.g. *Nocardia*) or fungi may be involved. Skin biopsies collected with sterile technique are ideal for culture. A detailed history and differential diagnoses should be provided to the laboratory, as special techniques and culture media are required for some organisms. While waiting for histopathology results where the aetiology remains unknown, fresh sterile skin biopsies can be stored by laboratories, and the most appropriate culture techniques employed when results are available. Microbial culture is more problematic with superficial skin infections, as normal skin flora or transient contaminants may interfere with culture and/or be pathogenic, and is less commonly indicated. Interpretation of microbial culture results can be misleading, as negative cultures do not exclude infectious disease, and positive cultures may not always indicate organisms responsible for disease. Culture results must be combined with clinical and historical data, cytology findings and often histopathology for correct interpretation.

Skin biopsies

Skin biopsies for histopathology are often important for diagnosis of skin disease, but often misused. They should only be considered after formulation of a differential diagnoses list based on historical and clinical data, and after surface cytology tests are performed. Biopsies are not indicated simply faced with chronic disease of unknown aetiology, where secondary infections, self-trauma lesions, and drug effects may mask primary changes; nor with many superficial skin diseases, including hypersensitivities, where a non-specific perivascular mononuclear and/or eosinophilic dermatitis is common. Biopsies are most useful with deep dermal and nodular diseases, and unusual or extensive unexplained lesions. Secondary infections should ideally be treated before biopsy collection, and samples should be collected carefully; no surface disinfection, and delicate handling to avoid artifactual damage. Biopsies may be collected using local (2% lignocaine, placed subcutaneously beneath lesions) or regional anaesthesia, or occasionally general anaesthesia when fractious animals. Multiple biopsies should be collected; primary lesions whenever present (e.g. pustules, papules), and differing secondary lesions (e.g. crusts, alopecia, hyperpigmentation, scale). *A minimum of 4 samples, and ideally 6–8, should be collected.* Punch biopsies are useful for uniform lesions, papules, small pustules, and crusts. Elliptical excisions are preferable for transitional regions (e.g. border of normal and abnormal), larger pustules/bullae/vesicles, and deep lesions. Samples should be blotted dry, thin samples pressed gently onto a piece of cardboard to prevent curling, and immersed in 10% buffered formalin. The biopsy defects can be closed with non-absorbable suture material. *To attain maximum information from the histopathology report, it is vital to provide the pathologist with relevant history, a description of the lesion(s), and your differential diagnosis list.* A histopathologist with a special interest or qualifications in dermatohistopathology will often provide more useful information.

Specialized tests

Specialized tests are required for evaluating some diseases, in particular hypersensitivities. Intradermal testing is useful for managing atopic dermatitis; although it does not confirm this diagnosis as false positive and negative reactions can occur. Patch testing is required to firmly diagnose contact allergies. Treatment trials, including insect control, elimination diets, and occasionally symptomatic treatment trials are often essential for diagnosis of skin disease.

Skin diseases in the horse

In this chapter, skin diseases are grouped according to their common presentations. This classification is somewhat arbitrary; and some cases will present in less typical ways. For example, dermatophytosis is typically associated with alopecia and scaling, but some affected horses present with pruritus (itch) as the predominant feature. There will often be clues in the history, physical examination, and/or initial diagnostic tests to alert the clinician to the primary disease, even with an unusual presentation.

13.2 SKIN DISEASES CHARACTERIZED BY PRURITUS (ITCH)

- This group of diseases is very common, and can be frustrating and time-consuming to diagnose, as lengthy treatment trials may be required (e.g. hypersensitivities). However, long-term control is more likely successful after a diagnosis has been confirmed, because treatment can be targeted.
- The most common pruritic skin diseases are allergic or parasitic. The prognosis is:
  - excellent for cure in most parasitic diseases with thorough treatment.
  - variable for allergic diseases; some are relatively easily controlled with symptomatic treatment; others are more persistent and problematic.
- Classically non-pruritic skin diseases may rarely present with pruritus as a major feature (e.g. bacterial pyoderma, dermatophytosis, dermatophilosis).
- Primary lesions are often few or absent, and may be masked by secondary lesions.
- Secondary lesions commonly include alopecia, scaling, excoriations, and crusting from self-trauma, and lichenification and hyperpigmentation when the condition is chronic.
- Skin biopsies are frequently unrewarding; many pruritic skin conditions will result in very similar histopathology changes.

Ectoparasitic skin diseases characterized by pruritus

Pediculosis (lice)

Pediculosis in horses occurs worldwide, and is more common in cooler moist climates (e.g. Europe), during late winter and early spring. Animals in poor condition, kept together in large numbers, and/or not groomed regularly may have heavy burdens. Sucking (*Haematopinus asini*) and/or biting (*Damalinia equi*) lice may be involved. Adult lice lay operculate eggs attached to the hairs. Lice are host-specific and do not live for more than a few days off the horse; the life cycle takes 3–4 weeks. Transmission is by direct animal contact or via fomites (e.g. grooming equipment, harnesses, bedding etc.).

Clinical signs

- Pruritus: often a key feature may be absent despite heavy burdens; typically see mane and tail rubbing, flank biting.
- Lesion distribution: head, neck and thighs.
- Primary lesions: typically none.
- Secondary lesions: alopecia (patchy, irregular, poorly demarcated, dull dishevelled coats); superficial erosions and/or crusting may be caused by rubbing.
- Anaemia can occur in extremely heavy infestations of sucking lice.

Diagnosis

Adult lice (3–6 mm long) and/or eggs ('nits'; 1–2 mm long, attached to hairs) are typically visible; although may be sparse. Biting species, light brown in colour, tend to distribute dorsally (back, mane, tail, neck). Sucking lice, blue-black in colour when filled with blood, tend to distribute ventrally. All in-contact horses should be assumed infected, even if lice are not seen and skin appears normal.

Treatment

Adult lice are easily killed with topical synthetic pyrethroids (e.g. cypermethrin or permethrin), although eggs are more resistant, and treatment beyond the typical 3-week life cycle is important. Permethrin full-body sprays for 4–6 weeks are effective treatment. Sucking lice are also killed by ivermectin (200 µg/kg orally). It is vital to treat all in-contact horses. The environment will not need extensive treatment if *all* horses are treated beyond 4 weeks, however cleaning of grooming equipment is advised especially with heavy infestations.

Chorioptic mange

This common skin disease in horses worldwide predominantly occurs in horses with well-feathered fetlocks, e.g. Draught horses, and is more common in winter months, and with intensive housing. It is caused by the surface dwelling, non-burrowing mite *Chorioptes*, with mite species possibly all variants of *Chorioptes bovis*. This mite commonly infests cattle but transmission of the mite between species is not reported. Eggs are laid on the skin, and hatch in 1–5 days; and the life cycle is typically 2–3 weeks. Transmission is by direct or indirect contact. All in-contact horses should be assumed infected.Clinical signs

- Pruritus: ranges from intense, with foot-stamping and/or rubbing of lower limbs, to absent.
- Lesion distribution: fetlock and pastern areas.
- Primary lesions: papules (rare).
- Secondary lesions: scale and crusting frequent (mild, focal to extensive); alopecia usually minimal; marked lichenification and swelling can occur with chronicity.

Diagnosis

Typical history and clinical signs are suggestive; although confirmation requires superficial skin scrapings. Mites are relatively large (0.3–0.5 mm long), and superficial dwellers, living in surface scale and debris. Scrapings can be difficult to obtain on heavily haired legs; hair should be clipped or parted, and ample surface crusting and scale collected. Mites are usually plentiful, but may be sparse.

Treatment and control

Treatment with topical synthetic pyrethroids (e.g. permethrin), or topical fipronil (off-label) is effective. Treatment must be applied thoroughly to *all* lower limbs of *all* in-contact horses, and ideally continued once weekly for 4–6 weeks. Ivermectin or moxydectin are ineffective for treatment. Doramectin may be effective in some cases. Topical pour-ons (e.g. epiromectin) are newer options effective in ungulates. Chronic infestation is common in groups of Draught and Shire horses, flaring most typically in winter and settling again over summer. Sustained treatment of all individuals beyond the 3-week life-cycle, followed by quarantine measures and/or repeated prophylactic treatment prior to exposure to untreated horses should be curative.

Environmental mites

A number of environmental mites have life stages that can transiently parasitize animals. They are often problematic to detect, as mites are often transient. Clinical signs are often similar for all mites, and typically affect multiple animals although the degree of irritation may vary between individuals. Environmental mites are not host specific.

Clinical signs

- Pruritus: often intense.
- Lesion distribution: head and legs; also muzzle, neck and ventral abdomen with heavy infestations.
- Primary lesions: papules (numerous to sparse).
- Secondary lesions: patchy alopecia, erythema, excoriations, and crusting from self-trauma.

Trombiculidiasis

Trombicula spp, (harvest or chigger mites) are common in many parts of the world, including Europe and Australia. Clinical signs typically occur in late summer and autumn. The adult and nymph stages parasitize plants and invertebrate hosts; and larval stages cause disease. The complete life cycle takes from 50–70 days.

Specific clinical signs

Tiny orange-red mites, just visible to naked eye, may be present in the centre of papules.

Diagnosis

Demonstration of larval *Trombiculid* spp. (0.2–0.4 mm long) on skin scrapings is diagnostic, although they are often absent as they feed only briefly. A presumptive diagnosis is often made based on exposure to known *Trombiculid* areas at classical times of year, and typical physical findings. Infested environments are frequently well-known locally, although populations may vary considerably each season, depending on climatic conditions.

Treatment

Larval mite stages are easily killed (e.g. pyrethrins, permethrin, lime sulphur); however, repeated contamination from environmental sources often occurs. Repellent sprays (e.g. permethrin, daily to weekly) may be required to prevent infestation.

Forage mites

A variety of environmental mites from the Acaridae and Pediculoididae families live in hay and grain, and huge population outbreaks can occur when environmental conditions are suitable. These mites feed on animal and human skin, producing irritation and dermatitis.

Specific clinical signs

Lesion distribution: muzzle most commonly and ventral areas; may also affect dorsum of horses stabled beneath food storage areas (e.g. hay lofts).

Diagnosis

Demonstration of mites (0.3–0.6 mm long) from skin scrapings, combings, or tape impressions; or from forage samples (where they are usually plentiful), is diagnostic.

Treatment

Mites are readily killed; however, successful treatment requires removal of contaminated forage and treatment of environment (e.g. pyrethrin sprays) before new forage replaced.

Other rare mites

Poultry mites

Dermanyssus gallinae, the red or poultry mite, will occasionally feed on horses and people. Direct or indirect poultry or bird nest contact is required. Adult mites feed on blood, and are visible as red mites when engorged.

Clinical signs

- Non-specific lesions as for environmental mites.
- Lesion distribution: ventral areas including muzzle mostly typically; may also affect dorsum in stabled horses where overhead bird nests are present.

Diagnosis

Demonstration of mites (large: 0.6–1 mm long) is diagnostic; mites are often easier to find in infected environments than on horses.

Treatment

Eradication of mites from the environment, using approved environmental insecticides is required.

Sarcoptes

Sarcoptes scabiei mites of horses, once common worldwide, are now extremely rare, although previously common. They have been eradicated from many countries, including USA and UK, and remain absent from others, including Australia. They cause an extremely pruritic dermatosis, similar to sarcoptes infestation in other species. Sarcoptes is a reportable disease in the horse in most countries.

Clinical signs

- Pruritus: intense.
- Lesion distribution: head, neck, ears.
- Primary lesions: papules, scaling.
- Secondary lesions: poorly demarcated alopecia and excoriations from self-trauma.

Diagnosis

Demonstration of typical sarcoptid mites in superficial skin scrapings is diagnostic, however mites may be sparse. Miticidal treatment trials are indicated despite negative skin scrapings if history and clinical signs are consistent.

Treatment

Ivermectin is likely very effective (200 µg/kg orally once weekly for 4 weeks). All in-contact horses must be treated.

Psoroptes

Psoroptes spp. rarely produce skin disease in horses in any part of the world. They are surface dwelling mites, feeding on scale and tissue fluids on skin and within ear canals. Transmission is via direct or indirect contact.

Clinical signs

- Pruritus: absent to intense.
- Lesion distribution: ear canal disease (head shaking, ear rubbing or scratching, head shyness); and/or truncal dermatitis (mainly mane and tail).
- Primary lesions: papules.
- Secondary lesions: scaling, crusts, excoriations, poorly-demarcated alopecia.

Diagnosis

Demonstration of mites on skin scrapings or from ear canal samples is diagnostic; (scrapings may require sedation). Mites are large (0.4–0.8 mm long) and readily seen.

Treatment

Ivermectin is effective (200 µg/kg orally once weekly to fortnightly for 4 weeks); although cleaning of ear canals, at least at initial presentation, is also important. All in-contact horses will require treatment.

Endoparasitic skin diseases characterized by pruritus

The major diseases in this group are now relatively rare, although prior to the development of ivermectin were important diseases worldwide.

Onchocerciasis

Onchocerca spp. are common filarial nematodes of horses. *O. cervicalis* (worldwide), *O. reticulata* (Europe, Asia), and *O. gutturosa* (North America, Europe, Australia, Africa) have been associated with cutaneous disease. Adults live in the ligamentum nuchae, producing microfilaria that migrate in connective tissue: in skin to the upper dermis, particularly on the ventral midline. *Culicoides* spp. are vectors, and disease incidence may be higher in summer. Although infection is common, clinical disease which is likely due to hypersensitivity to dying larvae, is rare. Affected horses are usually >3–4 years old; with a history of inadequate treatment with anthelmintics.

Clinical signs

- Pruritus: mild to severe.
- Lesion distribution: ventral abdomen, chest, head, neck.
- Secondary lesions: alopecia (poorly demarcated), scaling, erosions, ulcerations, crusting, hypopigmentation.
- Ocular lesions may occur concurrently: keratitis, depigmentation, conjunctival papules.

Diagnosis

Diagnosis can be difficult; and treatment trials often indicated. Microfilaria (0.2 mm long) can be demonstrated in skin scrapings, direct impression smears, or in macerated tissue biopsies after incubation in saline (37°C [98.6°F] for 10–15 minutes). Skin biopsies for histopathology, revealing superficial dermal eosinophilic inflammation associated with microfilaria, are required for confirmation. Multiple skin biopsies may be required to demonstrate microfilaria, which tend to distribute in pockets.

Treatment

Oral ivermectin (200 µg/kg) and moxidectin (400 µg/kg) are very effective at killing microfilaria; typically only one treatment is required. An ocular examination is important prior to treatment, and concurrent administration of glucocorticoids is often recommended initially due to frequent exacerbation of cutaneous and ocular lesions associated with microfilarial death.

Oxyuriasis

The nematode *Oxyuris equi* (pin worm, thread worm) infests the caecum and large colon of horses. Cutaneous disease is associated with female nematodes crawling from the anus onto the perineum to lay eggs, producing perianal and tail pruritus. Disease is rare, and associated with a history of inadequate treatment with anthelmintics.

Clinical signs

- Pruritus: mild to severe.
- Lesion distribution: perianal skin, tail base.
- Secondary lesions: broken hairs, patchy alopecia.

Diagnosis

Tape impressions from the perianal area reveal characteristic triangular operculate eggs.

Treatment

Routine treatment with anthelmintics is effective, although recently a poor response to anthelmintics has been reported in Europe.

Other helminth dermatoses

Larvae of *Strongyloides westeri*, a common small intestinal nematode of horses, and *Pelodera strongyloides*, a free-living nematode found in damp soil and decaying organic matter, will occasionally invade percutaneously, typically under moist unhygienic conditions. Skin in contact regions is affected (lower legs, muzzle) with a pruritic dermatitis characterized by papules, alopecia, scaling crusts, and ulcers. Demonstration of larvae in skin scrapings (*Pelodera* typically numerous), or within hair follicles (*Pelodera*) or dermal granulomas on histopathology of skin biopsies is diagnostic. Treatment involves routine anthelmintic therapy for *Strongyloides*, and cleaning the environment.

Insect irritation

Fly irritation

A variety of flies can cause annoyance and discomfort to many horses when present in large numbers, and allergic responses in some individuals even in low numbers. Common species include

- *Tabanid*, *Chyrssops* and *Haematopota* spp. (horse flies which are up to 25 mm in size are most active on hot humid days; breed on vegetation near water sources).
- *Stomoxys calcitrans* (stable flies which are 5–8 mm in size feed during day; breed in wet straw, bedding, manure).
- *Haematobia irritans* (horn flies which are 3–4 mm in size feed on the ventrum in warm sunny weather, on the dorsum in cool wet weather; breed in manure).
- *Simulium* spp. (black flies; 1–5 mm in size feed dawn and dusk; breed in running water).
- *Culicoides* spp. (biting midges, gnats, sandflies; 1–4 mm in size feed dawn and dusk; breed in damp decaying vegetation or manure) which are an important and common cause of hypersensitivity in horses.
- *Musca* and *Hydrotaea* spp. (house flies) do not cause direct irritation as they lack biting mouth parts.

Clinical signs

- Pruritus: frequent; sometimes pain.
- Lesion distribution: less-haired areas (face, ears, neck, ventrum, legs); dorsum.
- Primary lesions: papules, wheals.

Fly irritation is usually obvious from observation. Control is by regular use of insect repellents (e.g. permethrin) and removal/reduction of breeding sites in the environment where possible. Knowledge of the fly species involved, their preferred breeding sites, and preferred times of feeding are helpful. Commercially available predator wasps may reduce some fly populations by as much as 80%.

Equine ventral midline dermatitis

This seasonal dermatitis is associated with *Haematobia irritans* (horn flies) and *Culicoides* spp. (gnats, sandflies). It is most common in horses >4 years of age, although the majority of horses on one premises will be affected. Pruritus varies from intense to minimal. Classical signs consist of well-demarcated areas of punctuate ulcers, haemorrhagic crusts, lichenification, and alopecia on the ventral midline. Control is achieved by regular use of insect repellents, as above.

Hypersensitivities

This group of diseases often persist life-long, and making a firm diagnosis is the first important step to guide the most appropriate treatment options. The majority of skin lesions are caused by self-trauma.

Insect hypersensitivity

Insect hypersensitivity (Queensland itch, sweet itch) is the most common allergic skin disease in the horse. It occurs worldwide and is best documented to *Culicoides* spp., although *Simulium* spp., *Stomoxys calcitrans,* and *Haematobia irritans* are also implicated. *Culicoides* spp. vary around the world, most commonly *C. pulicaris* (UK); *C. brevitarsus* (Australia); and *C. variipenis, C. insignis, C. spinosus* and *C. stellifer* (USA). There is a distinctly seasonal pattern in temperate climates (spring, summer and autumn), however it is often non-seasonal in sub-tropical and tropical climates. The incidence is higher in some breeds (e.g. quarter horses, German Shires, ponies, Arabians, Icelandic horses), although insect hypersensitivity is reported in many breeds. The typical age of onset is 3–4 years. Affected horses often have recurrent bouts of disease at similar times each year.

Clinical signs

- Pruritus: often intense.
- Lesion distribution: classically dorsal midline, ears, mane, tail; more rarely ventrally orientated (ventral thorax/abdomen, axillae, groin, intermandibular space).
- Primary lesions: papules.
- Secondary lesions: poorly-demarcated alopecia, scaling, lichenification; broken hairs on mane and tail.

Diagnosis

Insect hypersensitivity is often presumed based on typical seasonal patterns of pruritus and typical sites of lesions; however, atopic dermatitis, and possibly food hypersensitivities or adverse reactions, may cause identical clinical signs. Histopathology of skin biopsies is non-specific, with a superficial and deep perivascular to interstitial eosinophilic dermatitis that could be consistent with other hypersensitivities

or external parasites. Intradermal testing can provide supportive positive results although false positive results may occur. Testing with extracts from the relevant *Culicoides* species for each region seems important to help minimise false negative reactions. However, firm diagnosis of insect hypersensitivity requires complete response to a thorough insect control trial. The author's preferred option is permethrin aqueous spray or oil-based wipe-on applied twice daily (dawn and later afternoon) to all affected areas for 4 weeks. Rugs and hoods can be helpful to limit skin exposure; however, spraying is still required adjacent to, and on exposed areas. Complete response, and ideally relapse on re-exposure, confirms the diagnosis. The trial is very time-consuming for owners, but is essential to confirm the diagnosis and ensure that insect control measures are warranted.

Treatment

Measures to reduce insect populations and/or avoid insect exposure are essential. Treatment of un-rugged grazing horses is extremely difficult. Insect repellents (e.g. permethrin) should be timed prior to major insect feeding times; frequency of application will depend on other measures in place and current insect populations. Rugs and hoods; stabling at dusk and dawn, ideally with fans, fine-meshing, and/or sustained release insecticides; and environmental control which involves removal of stagnant water, decaying vegetation, and manure will all help. Anti-inflammatory doses of oral prednisolone (0.5–1 mg/kg sid) or injectable dexamethasone (0.05–0.1 mg/kg SC) can give short-term relief when pruritus is severe, although won't always be effective and side-effects must be considered, especially with long-term use. Insect immunotherapy is currently not widely available, and response is questionable.

Atopic dermatitis

Atopic dermatitis has recently been re-defined as a genetically predisposed inflammatory and pruritic allergic skin disease with characteristic clinical features. It is associated most commonly with development of IgE antibodies to a variety of environmental allergens (pollens, insects, mould spores, dust mites). In horses, the typical age of onset is 1–6 years, and disease may be seasonal or non-seasonal, often worsening with age. Thoroughbreds and Arabians may be at increased risk, although there is no clear evidence for a breed predisposition.

Clinical signs

- Pruritus: often intense.
- Lesion distribution: face, pinnae, ventral abdomen, ventral thorax, legs most commonly; also lateral neck, mane, tail base.
- Primary lesions: wheals (uritcaria, pruritic or non-pruritic).
- Secondary lesions: poorly-demarcated alopecia, scaling, lichenification, hyperpigmentation; broken hairs on mane and tail.

Diagnosis

Diagnosis requires a compatible history and clinical signs, and ruling out other possible differentials, especially insect hypersensitivity. Food allergy/adverse reactions and external parasites should also be considered. Testing for atopic dermatitis in horses is now performed in many parts of the world and involves the intradermal injection of a range of potential allergens relevant to the country/region of testing (Figure 13.1). Results are most typically scored subjectively, based on wheal size, turgor, and erythema (often hard to evaluate in dark horse skin). Positive intradermal results will support a diagnosis of atopic dermatitis and/or insect hypersensitivity, however firm diagnosis requires exclusion of other differentials. Recent studies have been helpful in determining appropriate concentration of allergen for testing in order to minimize false positive results which have previously caused confusion when intradermal testing. New serum allergen-specific IgE testing is being developed for horses, however, some of the currently available tests have poor sensitivity, specificity, and predictive value.

Figure 13.1 Equine skin testing. Intradermal testing in an atopic horse: multiple strong positive pollen reactions.

Treatment

Allergen-specific immunotherapy, based on positive intradermal test results, is ideal when clinical signs are persistent and/or severe, and poorly controlled using safe symptomatic treatments. Good response is claimed in at least 60% of horses, although controlled studies in the horse are yet to be performed. Allergen avoidance measures are ideal wherever possible, and may help in select cases. In cooler moist climates like the UK where mould sensitivity is more common, allergen avoidance measures can be very helpful; conversely, in warmer drier climates, such as Australia, pollen sensitivity is very common and allergen avoidance rarely possible. Information concerning local allergens is often available from local human and veterinary dermatologists.

Symptomatic treatment is variably effective for atopic dermatitis in the horse. Safer long-term options, that may be effective alone or in a steroid-sparing role, include antihistamines (e.g. chlorpheniramine 0.25–0.5 mg/kg bid; diphenhydramine 1–2 mg/kg bid-tid; hydroxyzine 1–2 mg/kg bid-tid [discontinued in some countries]); fatty acids (e.g. flax seed oil, linseed oil, fish oil 1–2 mL/kg sid); and topical therapies (cleansers and moisturizers, e.g. shampoos, rinses; antipruritics e.g. colloidal oatmeal, aloe vera, glucocorticoids). More potent forms of topical glucocorticoids can be effective for more localized disease. Off-label use of human or small animal preparations including triamcinolone 0.02% cream or ointment, and hydrocortisone aceponate spray have been used. Although more reliably effective, systemic glucocorticoids can have potentially serious short and long-term side-effects. Prednisolone and dexamethasone have been used, as for insect hypersensitivity, to reduce severe pruritus at anti-inflammatory doses, reduced to the lowest effective levels on an alternative-day basis or less. Potential side-effects should be discussed with owners.

Contact dermatitis

Contact dermatitis may be caused by an irritant (i.e., non-immunological) response, or by a type IV or delayed hypersensitivity response, whereby an immunological reaction occurs to a specific hapten, usually a small, chemically reactive, lipid-soluble molecule that binds to a protein to form an antigen. A variable sensitization period from 3–5 weeks up to 2 years or more is needed before a

hypersensitivity response develops. Few cases of true contact hypersensitivity are documented in horses, although 'contact dermatitis' is claimed to be common in the horse. Cotton, chrome, and jute are documented causes; pasture plants, bedding, numerous topical shampoos and insecticides, grooming aids, blankets and tack have all been implicated. A contact reaction requires direct contact between the offending agent and skin; hair protects against direct contact, while moisture (e.g. sweating) enhances it.

Clinical signs

- Pruritus: mild to intense; may be painful.
- Lesion distribution: head, extremities, ventral body surfaces and tail region (e.g. muzzle, lower legs due to pasture plants, bedding; face and trunk due to tack, or insect repellents and sprays.
- Primary lesions: papules, vesicles.
- Secondary lesions: erythema, scaling, alopecia to erosions and ulcerations with crusting when acute; lichenification and pigmentary changes when chronic.

Diagnosis

Diagnosis of contact hypersensitivity requires patch testing, where suspected allergens are placed in direct skin contact for 48 hours, and skin reactions evaluated. Problems with maintaining allergen contact at a fixed site under bandaging make this test difficult, and the test is currently not standardized for horses. Provocative exposure can be diagnostic, with resolution of clinical signs on withdrawal of allergens (up to 14 days), and relapse with re-exposure. Provocative testing is time consuming, with many potential irritants/allergens to choose from.

Treatment

Identification of the offending allergen or irritant, and avoidance where possible, will be curative. Topical and/or systemic glucocorticoids will help reduce severe reactions.

Food allergy/adverse reaction

Cutaneous food reactions are poorly documented and understood in the horse. These reactions, however, well-characterized in humans and some domestic species, and some cases have been confirmed in the horse. Reactions may be immunological, toxicities or intolerances. Typically the same diet has been fed for some time (at least months) before clinical signs develop. Clinical signs may be seasonal or non-seasonal, depending on the allergens involved and consistency of feeding. No age, breed or sex predispositions are reported. Food reactions in the horse have been suspected or confirmed to barley, bran, lucerne, oats, wheat, clover, concentrates, and supplements.

Clinical signs

- Pruritus: mild to intense.
- Lesion distribution: face, neck, trunk, rump; exclusive tail and perianal lesions also reported.
- Primary lesions: wheals (urticaria, pruritic or non-pruritic).
- Secondary lesions: poorly-demarcated alopecia, scaling, lichenification, hyperpigmentation; broken hairs on mane and tail.

Diagnosis

Diagnosis requires an elimination diet followed by provocative re-exposure. Ideally a novel diet is fed, however this is often difficult in the horse, and a common approach is to feed a bulk food not previously fed (e.g. lucerne, oat, or timothy grass hay), and withhold all concentrates, additives and supplements. If a grain is also necessary, a pure grain (e.g. oats, corn) rather than a mix should be fed. The necessary duration of the diet is currently unknown, but 4 weeks is often suggested. Resolution of clinical signs during the diet, relapse with provocative re-exposure, and resolution again on avoidance will confirm the diagnosis.

Treatment

Avoidance of offending foods is curative.

13.3 SKIN DISEASES CHARACTERIZED BY ALOPECIA, SCALING AND/OR CRUSTING

- This group of skin diseases is also very common in the horse; more often associated with localized lesions, although may be generalized.
- The major differential diagnoses are infectious dermatoses (bacterial, fungal, and parasitic); with sterile immune-mediated or miscellaneous dermatoses less frequent. Fairly simple skin surface diagnostics will often identify an infectious cause.
- Primary lesions are often few or absent; well-demarcated alopecia, scaling and crusting may be primary lesions, however also commonly secondary.
- Secondary lesions are usually more prominent, including alopecia, scaling, excoriations, and crusting from self-trauma.
- Pruritus will most typically be absent or minimal; an important clue is whether lesions developed before or after any pruritus.
- Skin biopsies: frequently diagnostic but not often required for diagnosis; essential for diagnosis in some diseases in this group.

Infectious skin diseases characterized by alopecia, scaling, and/or crusting

Dermatophilosis

Caused by the bacteria *Dermatophilus congolensis*, dermatophilosis ('rain scald', 'mud rash' or 'mud fever') is common in horses, and associated with moisture, skin damage, and individual susceptibility. Some horses will have repeated bouts each year while other horses in the same conditions remain disease-free. Classically there is prolonged exposure to rain, although sometimes associated with sweating under rugs. Infection can occur directly or indirectly; zoospores remain viable in the environment for many months (in shed scale or crusts, and at least transiently in soil).

Clinical signs

- Lesion distribution: dorsal trunk; occasionally muzzle, or distal limbs.
- Primary lesions: papules (transient).
- Secondary lesions: crusts (multiple circular, up to 5 cm diameter, often at the base of matted hair, with a concave under-surface with central hair projections ('toothbrush' appearance) and underlying purulent exudate; dry firm 'nobbly' crusts typical on the muzzle (Figure 13.2); more chronic 'dry' form associated with non-adherent fine white scaling and patchy alopecia.
- Lesions may be painful.
- Concurrent oedema, pain and lameness may occur when lesions are on the distal limb.

Diagnosis

Classical crusting and an appropriate history are strongly supportive of a diagnosis. However confirmation, especially with less classical or localized cases, relies on cytology and/or skin biopsy, and occasionally bacterial culture. Direct smears of purulent exudate, tape impressions from dry scaling skin, or saline-soaked minced crusts (Figure 13.3)

Figure 13.2 Characteristic dry 'nobbly' crusts associated with *Dermatophilus congolensis* infection in a 1-month-old foal.

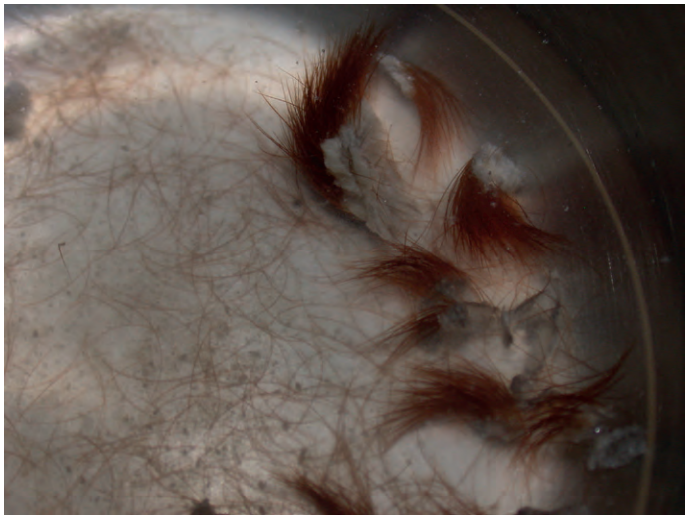


Figure 13.3 Dermatophilosis: soaking crusts. Cytology for dermatophilosis is more reliable if crusts are soaked for ~15 minutes in warm saline, then the undersides of crusts are smeared onto a glass slide for staining (e.g. Diff Quik® or Gram stain).

may reveal gram-positive branching filaments consisting of parallel rows of zoospores forming a 'railroad track' appearance (Figure 13.4). Histopathology will typically reveal palisading crusts containing the filamentous bacteria. Culture can be difficult, and ideally requires microaerophilic conditions.

Treatment

Effective treatment requires avoidance of prolonged exposure to moisture whenever possible, and daily topical application of antibacterial sprays or solutions. The author prefers a 2–3% chlorhexidine

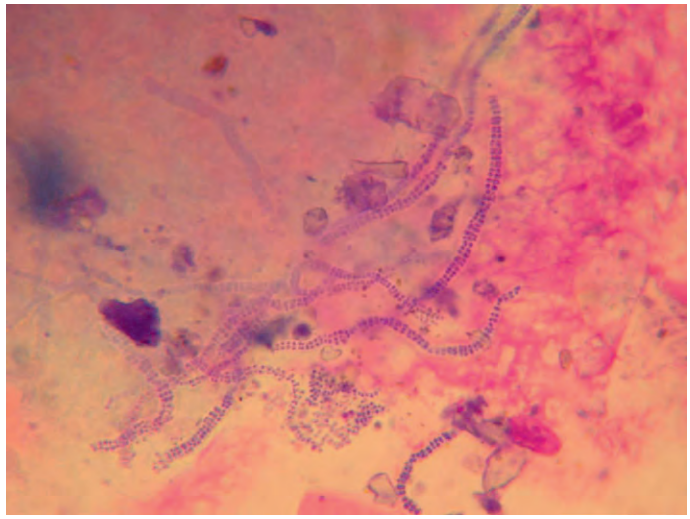


Figure 13.4 Dermatophilosis cytology. Typical 'railroad track' appearance of *Dermatophilus congolensis* zoospores that may be evident on cytology or tape impression samples (oil immersion field: 1000× magnification).

solution, applied as a spray or rinse, with additional 3% chlorhexidine or benzoyl peroxide shampooing as needed to assist with crust removal. Crusts should be disposed of carefully to avoid further environmental contamination. Parenteral antibiotics are occasionally required in severe cases (e.g. procaine penicillin, 20 000 IU/kg IM, bid or trimethoprim sulphur, 30 mg/kg PO, bid for 10–14 days). Regular topical antibacterial sprays are often protective at higher risk times in susceptible individuals, although some protection from constant rain is required.

Dermatophytosis

Also common worldwide in horses, dermatophytosis ('ringworm') is most frequently caused by *Trichophyton equinum*, but also by other dermatophyte species including *T. mentagrophytes, Microsporum equinum, M. canis, and M. gypseum*. It is common in riding and racing horses, particularly from stables, with young animals who is more susceptible. Cases occur more commonly in autumn and winter in cooler climates, and during warm, moist weather in warmer climates. Transmission may be direct or indirect via grooming equipment, tack, clippers or contaminated boxes. Some species are more virulent, although some skin damage is typically required for infection to establish. The incubation period may be up to 4 weeks. Dermatophytosis is a zoonosis; transmission is rare with the common *T. equinum equinum* infections, and more common with *T. equinum autotropicum* and *Microsporum canis* (Australia and New Zealand), or *T. verrucosum* infections.

Clinical signs

- Lesion distribution: girth, saddle, neck or facial regions (tack-associated).
- Primary lesions: alopecia, tending to circular and well-demarcated; papules; early lesions may resemble urticaria.
- Secondary lesions: scaling, erythema, crusting; severe oedema, erythema, exudation rarely.
- Pruritus: mostly absent.

Diagnosis

A positive fungal culture with consistent historical and clinical findings is diagnostic. Samples of hair, skin surface debris and keratin should be collected from the margins of active lesions and ideally sent

to a commercial laboratory for fungal identification. Speciation aids with prognosis, helps to identify the most likely source of infection, and guides environmental decontamination. Histopathology will also confirm dermatophytosis, although culture remains important to determine the fungal species involved. Trichograms or tape impressions (which easily pick up broken hairs and may be higher yield) may reveal fungal spores and/or hyphae, although they may be absent. KOH preparation is not required as most animal dermatophytes cause ectothrix infections (outside of hair shafts), unlike common endothrix infections (inside hair shafts) in people. Wood's lamp examination is useful in only a small group of patients, being positive only with *M. equinum* and 50% of *M. canis*.

Treatment

Infection is usually self-limiting, with spontaneous remission occurring in 1 to 3 months in healthy animals; however, the risk of contagion and zoonosis often prompt treatment to hasten resolution. Topical treatments are often effective; the author's preference is topical 0.2% enilconazole rinse (Imaverol®; every 5–7 days for a minimum of 4 weeks or until beyond resolution of lesions; will occasionally irritate), although 2% lime sulphur preparations (unavailable as veterinary product in Australia) are also effective. Systemic griseofulvin has not been clearly evaluated in the horse, and is not currently recommended. Anecdotally, fluconazole (generic, less-expensive forms) has been used and may be effective. Environmental decontamination is vital in eliminating infections from stables or outbreak situations as fungal spores can remain viable for months to years. Household bleach (sodium hypochlorite 5%) is one of the most effective agents for solid surfaces, but must be used with care. Bedding, rugs, brushes and combs, and tack should be disposed of (especially with virulent infections) or thoroughly cleaned, dried, and disinfected. In some countries prophylactic vaccines are available, which may be helpful in high exposure situations, although no clear data to support efficacy are currently available.

Bacterial pyoderma – superficial

Frequently caused by coagulase-positive *Staphylococcal* spp., bacterial pyoderma is a common secondary skin disease in horses, most typically associated with relatively minor underlying problems, including mild skin surface trauma (e.g. ill-fitting tack and saddles, heavy riding), excessive rugging, surface irritants, and inadequate grooming. It is also recognized secondary to hypersensitivities, including atopic dermatitis. Disease may be more frequent in spring and summer associated with coat shedding, higher temperatures and humidity, increased training schedules, and increased insect populations. *S. aureus, S. intermedius* and *S. hyicus* have been isolated. Apparent outbreaks can occur in racing stables; often with problems in coat management (e.g. irritant washes, frequent rugging, rugging immediately after exercising, and routine hygiene), and/or associated with onset of training and possibly associated stresses.

Clinical signs

- Lesion distribution: saddle and tack areas; pasterns and heels (e.g. 'greasy heal').
- Primary lesions: follicular papules, rarely pustules.
- Secondary lesions: alopecia (initially raised hairs over palpable papules, giving a ruffled patchy appearance to the coat; progressing to demarcated, roughly circular patches of alopecia (from 2–3 mm to occasionally 5–8 mm or greater) (Figure 13.5); scaling, epidermal collarettes, crusting; may progress to nodules +/– draining tracts when furunculosis occurs.
- Pruritus: often inapparent.

Diagnosis

Diagnosis requires consistent history, physical examination, and surface skin cytology. Tape impresssions are most useful for dry alopecic skin; impression smears for moist skin; fine needle aspirates for nodules; all revealing neutrophils with intracellular bacteria (typically cocci). Confirmation can be difficult in early cases with follicular papules and minimal alopecia. Bacterial culture and sensitivity from tissue biopsies can be helpful, particularly with nodular lesions. Culture of superficial skin lesions, however, may result in growth of normal flora or transient organisms rather than a true primary pathogen. Histopathology will be supportive; however, bacteria will not always be present to clearly confirm.

Treatment

Topical antibacterial products are often effective treatment for superficial infections. The author's preference is 2–3% chlorhexidine solution (not scrub) sprayed twice daily on affected areas (and left on for residual action) until resolving, then gradually reduced. Twice weekly application may prevent further recurrence, assuming the primary problem is relatively minor (e.g. repetitive trauma). Chlorhexidine or benzoyl peroxide shampoos once to twice-weekly help with removal of crusts and exudation if present, and are useful to clean coats and remove surface debris, although antibacterial action is only transient. Iodine solution or sprays can be used alternatively but they have less residual action, discolour white hair, and can cause irritation. Horses with more severe lesions, including those with furunculosis, may require administration of systemic antibiotics. A minimum of 3-weeks' treatment is required, for which oral trimethoprim sulphur is the authors' preference (30 mg/kg bid), although oral doxycycline (10 mg/kg bid; more possibility of GIT side-effects), or oral enrofloxacin (7.5 mg/kg sid; poultry formulation; off-label) may be used. IM procaine penicillin twice daily is often effective initially; however long-term IM injections are poorly tolerated. It is important to search for underlying diseases or important predisposing factors for horses with recurrent or severe pyoderma.

Demodicosis

Demodicosis in the horse is very rare, and caused by increased numbers of the non-contagious follicular mites, *Demodex equi* or *D. caballi*. Disease is associated with immunosuppression, typically caused by chronic glucocorticoid treatment, allowing an increase in mite numbers. Areas of patchy alopecia, with some well-demarcated borders, and scaling occur most typically on the face and neck. Occasional primary papules or pustules may be present. Diagnosis requires deep skin scrapings, and mites are usually plentiful. Demodicosis in the horse is rarely symptomatic, although treatment should include resolution of immunosuppression if possible. Spontaneous resolution has also been reported. Ivermectin has been used successfully. Amitraz rinse, used to treat dogs for demodectic mange, can be lethal in horses.

Immune-mediated skin diseases characterized by alopecia, scaling, and/or crusting

Pemphigus foliaceus

Pemphigus foliaceus is uncommon in the horse, although one of the more common immune-mediated skin diseases. Loss of intercellular adhesion occurs in the superficial epidermis, producing superficial pustules and crusting. A wide age range may be affected, although two groups are often reported; young foals from 2–12 months, and adult horses. The prognosis is generally better for affected foals. Clinical signs may wax and wane.

Figure 13.5 Secondary bacterial folliculitis. Superficial bacterial pyoderma in a pony with underlying insect bite hypersensitivity. Note the typical circular multifocal alopecic lesions associated with bacterial pyoderma.

Clinical signs

- Lesion distribution: face or limbs initially, then often generalized (Figure 13.6); may remain localized to face or coronary bands.
- Primary lesions: transient vesicles, bullae, pustules.
- Secondary lesions: extensive crusting, scaling and well-demarcated circular areas of alopecia (Figure 13.7).
- Pruritus: may be present.
- Systemic signs: lethargy, pyrexia, weight loss in 50%; oedema of ventral abdomen, limbs in >50%.

Diagnosis

Cytology of active lesions (e.g. impression smears from beneath crusts, aspirates from intact pustules, tape impressions from drier lesions) often reveals acantholytic cells and neutrophils, that may contain bacteria. Biopsies for histopathology are required for disease confirmation; even though samples collected from primary lesions are more likely diagnostic, biopsies of crusted lesions should also be included.

Treatment

Treatment requires immunosuppression, and owner commitment; successful treatment is often intensive initially, and prolonged to life-long. Oral prednisolone is the mainstay of treatment (2–4 mg/kg sid initially for 2–3 weeks until clinical remission, then tapered slowly [e.g. 25% of the 48 hour dose every 2–3 weeks], reducing to alternate-day therapy as soon as possible). Occasionally dexamethasone (0.2–0.4 mg/kg sid) or triamcinolone are used initially, and may help to achieve remission in more severe or poorly responsive cases. Gold salts have been used successfully in a steroid-sparing role, and occasionally as sole therapy in foals. It is important to discuss potential side-effects with owners, including a possible increased risk of laminitis. A concurrent management plan to reduce risks of laminitis is recommended, using low energy diets, weight control, and regular gentle exercise.

Alopecia areata

This uncommon immune-mediated disease in the horse is caused by destruction or damage to hair bulbs producing well-circumscribed areas of non-inflammatory hair loss.

Clinical signs

- Lesion distribution: face, neck and trunk.
- Primary lesions: small to large circular patches of well-demarcated alopecia; also leukotrichia (loss of hair pigmentation) (Figure 13.8).
- Secondary lesions: absent – no associated pruritus and normal skin appearance.

Diagnosis

Trichograms from the periphery of active lesions are highly suggestive when typical broken 'exclamation point' hair shafts are present, or sites of weakness as for trichorrhexis nodosa (hair shaft defect) may be seen (Figures 13.9, 13.10). Skin biopsies for histopathology may reveal lymphocyte infiltration of hair bulbs; however, classical histological changes may be absent by the time of presentation.

Treatment

This is a cosmetic disease only, and no treatments have been clearly shown to be effective. Lesions may spontaneously resolve

Figure 13.6 Pemphigus foliaceus. Generalized pemphigus foliaceus in an adult horse. Note the extensive circular to coalescing areas of well-demarcated alopecia.

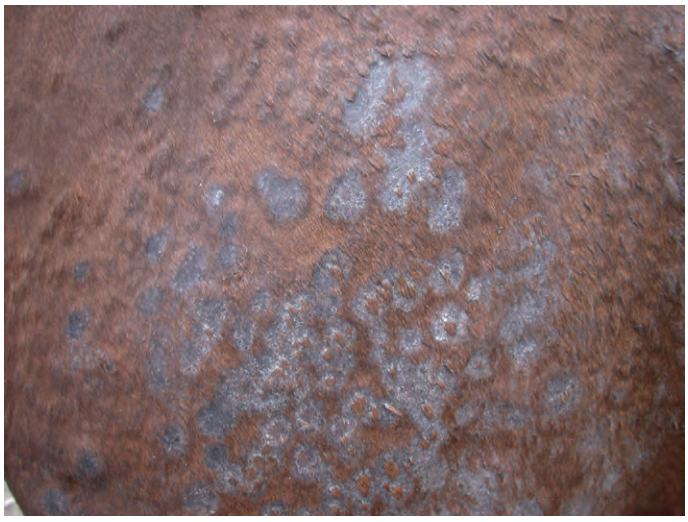


Figure 13.7 Pemphigus foliaceus. Closer view of annular to circular areas of alopecia, scaling and light crusting from the horse shown in Figure 13.6.

Figure 13.8 Alopecia areata: clinical. Well-demarcated area of alopecia in a horse with alopecia areata.

from 1–2 months up to 2 years. Minoxidil, a vasodilator that can stimulate hair growth in humans (Rogaine®), has been used with anecdotal success in some horses (2% solution applied twice daily), and appears well-tolerated, however no safety studies have been reported.

Lupus erythematosus (LE)

Systemic LE is very rare in the horse; characterized by variable multisystemic signs, including alopecia, depigmentation and scaling of skin (face, neck and trunk), lymphoedema, panniculitis, polyarthritis, thrombocytopenia, proteinuria, fever, depression and weight loss.

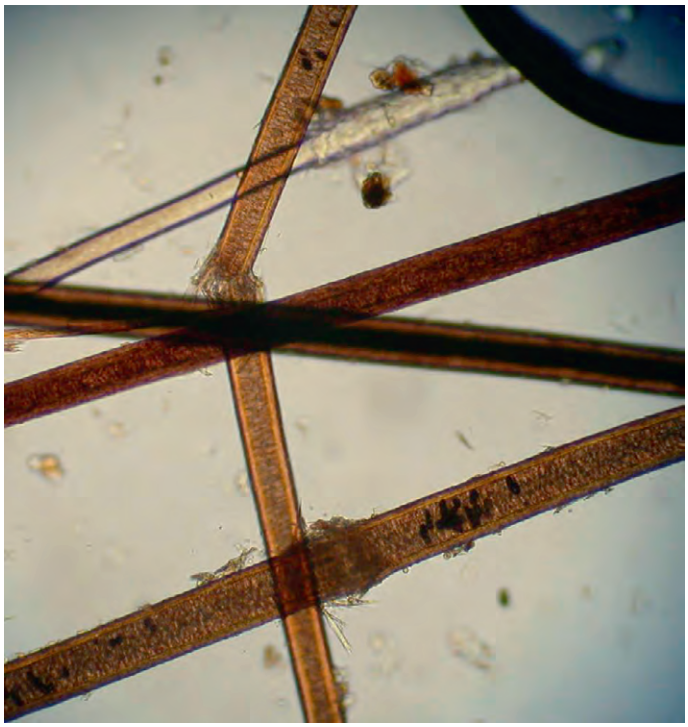


Figure 13.9 Alopecia areata trichogram. Trichogram from the horse shown in Figure 13.8 showing sites of weakness in hairs associated with alopecia areata (40× magnification).

Figure 13.10 Alopecia areata. Closer view of Figure 13.9 (100× magnification).

Discoid LE is also rare; characterized by erythema, scaling and alopecia on the face, especially the lips, nostrils and periocular region. Diagnosis requires skin biopsies.

Miscellaneous skin diseases characterized by alopecia, scaling, and/or crusting

Seborrhoea

Seborrhoea is a poorly understood term that indicates a keratinization defect, with excessive production (most commonly) or reduced desquamation of the stratum corneum, producing clinical 'scale' or 'dandruff'. The vast majority of cases are secondary, due to an increased cell-turnover associated with stimulation from a wide variety of underlying primary diseases; environmental conditions (e.g. topical products, stabled with heating), allergic or parasitic diseases, and nutritional deficiencies are the most common causes. Occasional cases of primary seborrhoea occur, where the epidermal defect is inherent. Primary seborrhoea is present from a young age (6 months or younger).

Clinical signs

- Lesion distribution: secondary seborrhoea: regional or generalized; face, trunk, and/or legs most often; Primary seborrhoea: often mane and tail; may be generalized.
- Primary and secondary lesions: scale; dry (seborrhoea sicca) or greasy (seborrhoea oleosa).

Diagnosis

Scaly skin is by definition called seborrhoea. Skin biopsies are required to confirm primary seborrhoea. Resolution of scaling with treatment/correction of underlying disease or factors confirms secondary seborrhoea.

Treatment

Specific treatment is rarely indicated for secondary seborrhoea, as management of the underlying disease/problem should result in resolution. For primary disease, or with the occasional more persistent secondary case, antiseborrheic shampoos are indicated; starting with mildest emollient shampoos (e.g. salicylic acid) and only progressing to degreasing shampoos (e.g. sulphur, tar) with very greasy skin and poor response to initial products. Management of secondary bacterial infections may also be important with primary cases.

Equine sarcoidosis

This rare disease is characterized by generalized seborrhoea, severe wasting, and sarcoidal granulomas in multiple body organs. Scaling, crusting and alopecia begin on the face or limbs and progress to a generalized or multifocal exfoliative dermatitis, with the mane and tail often unaffected. Most horses become systemically ill, with inappetance, weight-loss, and pyrexia. Diagnosis requires skin biopsies which reveal nodular to diffuse sarcoidal granulomas. The prognosis is generally poor, with progressive deterioration; however the disease in some horses spontaneously resolves. Immunosuppressive doses of glucocorticoids, especially early in the disease, can produce partial or complete response.

Exfoliative eosinophilic dermatitis

This rare disease (also known as multisystemic eosinophilic, epitheliotropic disease or MEED) is characterized by severe eosinophilic infiltrate in multiple body organs, typically associated with a generalized exfoliative dermatosis, ulcerative stomatitis, diarrhoea and weight-loss. The aetiology is unknown. Lesions often begin with transient vesicles and bullae progressing quickly to ulcers on the muzzle, coronets and mucocutaneous junctions; progressing to more prominent

generalized scaling, crusting, and alopecia. Pruritus is variable. Diagnosis requires skin biopsies. The prognosis is frequently grave, with most horses progressively deteriorating over several months. Temporary response can be achieved with immunosuppressive doses of glucocorticoids.

Epitheliotropic lymphoma

This clinically non-specific form of neoplasia is very rare in the horse; most typically presenting with multifocal to generalized areas of alopecia, scaling and crusting; and sometimes nodules. Diagnosis requires biopsies. Prognosis is poor. Little information is currently available on chemotherapeutic options, however response to treatment for this tumour in other animal species is often poor.

Exudative dermatitis

Occasional cases of a generalized exudative dermatitis are anecdotally reported in horses, with serous exudation from extensively affected areas resulting in hair-matting, patchy hair-loss and crusting. An underlying aetiology is rarely confirmed, although some cases are seasonally recurrent and hypersensitivity suspected. Glucocorticoid therapy, as for hypersensitivities, can provide temporary response.

13.4 SKIN DISEASES CHARACTERIZED BY ULCERATION AND CRUSTING

- This group of diseases is relatively rare in the horse. Environmental causes, immune-mediated diseases, and viral infections should be considered.
- Primary lesions: vesicles or bullae (transient).
- Secondary lesions: ulceration, crusting; alopecia and scarring if more chronic.
- Pruritus: absent or minimal.
- Skin biopsies are often required for diagnosis, although biopsies of ulcerated areas are rarely useful. Sample vesicles or bullae, or transitional samples including adjacent intact epidermis.

Photosensitization

This disease occurs when a photodynamic agent accumulates within the skin, together with exposure to a sufficient amount of ultraviolet light, and absorption of ultraviolet radiation; most likely to occur in areas with lack of pigment and hair coat. The photodynamic agent may be produced endogenously (e.g. phylloerythrin; increases with hepatic disease [infectious, degenerative, neoplastic, toxic e.g. rape, heliotrope, blue-green algae]), referred to as secondary photosensitization; or may be primarily acquired via ingestion (e.g. plants such as alfalfa, St John's Wort, some clovers), drugs (e.g. tetracyclines, chlorothiazides, sulphonamides), or direct skin contact (e.g. some plants).

Clinical signs

- Lesion distribution: primarily non-pigmented lightly haired areas; rarely extends into adjacent haired and/or pigmented areas; typically distal extremities and muzzle for contact forms.
- Primary lesions: vesicles.
- Secondary lesions: swelling, erythema, ulceration, crusting; chronic scarring and alopecia.

Diagnosis

Diagnosis is often based on clinical signs (distribution of lesions, evidence of liver dysfunction with secondary disease) and an accurate history (grazing habits, seasonality of signs, recent drug administration). Although histopathology is not specific for photosensitization, skin biopsies will help rule out other differentials.

Treatment

In cases of liver disease the prognosis is guarded. Otherwise, identification and removal of the photodynamic agent provides long-term control; short-term management relies on preventing exposure to sunlight, and good wound hygiene.

Subepidermal bullous diseases

A range of subepidermal bullous diseases, difficult to differentiate using readily available diagnostics, are reported rarely in horses, with bullous pemphigoid most frequently reported. Epidermolysis bullosa acquisita, and mucous membrane pemphigoid may also occur. Disruption of adhesion structures between the epidermis and dermis occurs, resulting in transient vesicles and bullae, progressing to ulceration and crusting, which most frequently affect mucocutaneous junctions (lips, vulva, anus, eyelids), axillae, groin and the oral cavity. Diagnosis requires skin or mucosal biopsies for histopathology, revealing subepidermal clefting and vesicle formation; and immunofluorescence or immunohistochemical staining to demonstrate the antigen target characterizing each disease. In all reported cases of bullous pemphigoid in the horse the disease has been severe and progressive. Aggressive immunosuppressive therapy can be considered in early cases.

Cutaneous asthenia

This group of rare inherited, congenital diseases is characterized by loose, hyperextensible and fragile skin, includes Ehlers–Danlos syndrome, dermatosparaxis, and hyperelastosis cutis. It has been reported in Quarter Horses (primarily cutting horses) and occasionally other breeds in the USA, UK and other countries. One form, hereditary equine regional dermal asthenia (HERDA) is a form of hyperelastosis cutis seen in Quarter Horses or their crosses, with a confirmed autosomal recessive mode of inheritance. In addition to regions of hyperextensible skin, there are often areas of skin tearing and ulceration which heal with scarring, and seromas or haematomas. The dorsum is more commonly affected, with saddle-trauma occasionally associated with lesions. The mean age of onset of signs in a study of 50 horses in USA was 2.4 years-of-age, with horses often having apparently normal skin prior to this. Diagnosis is suspected when there are classical historical and clinical findings, and is supported by histopathologic abnormalities in collagen fibre appearance and arrangement in the deep dermis (tightly grouped thin and shortened fibres arranged in clusters). Special stains are required. Electron microscopy has shown no abnormalities in the periodicity of collagen bundles. DNA testing is now available using hair or blood samples to detect affected, normal, and carrier horses. Minimizing skin trauma is currently the only possible management plan. Early results from screening for carrier animals has detected a carrier rate of 14–15% in Quarter Horses in USA.

Viral diseases

See also Chapter 19.

Horse pox

This rare mildly contagious viral disease in horses, reported in Europe, Australia and North America, is caused by a poxvirus. Disease is characterized by typical pox lesions, beginning with vesicles that rapidly ulcerate, and progressively crust. Lesions occur most common on the muzzle and face. Spontaneous resolution is typically within 4 weeks.

Herpes coital exanthema

This contagious venereal disease of horses, caused by equine herpes virus 3, occurs sporadically in most parts of the world. Transmission may occur via coitus, insects, fomites, or inhalation. Initial lesions include small papules, vesicles to bullae, or pustules on the penis, prepuce, vulva or perineum, which may be pruritic. Spontaneous healing occurs over 3–5 weeks.

Vesicular stomatitis

Restricted to regions in North and South America, this disease is associated with systemic illness, and causes vesicles and ulcers in the oral cavity, and occasionally on the prepuce, udder, and coronets.

Equine viral arteritis

Contagious via coitus and inhalation, this viral disease occurs in many parts of the world, and is characterized by oedema of the distal limbs, and occasional papular to urticarial lesions on the back. Spontaneous resolution occurs.

Equine papular dermatitis

This poorly characterized disease, caused by a poxvirus, is reported in North America, Australia and Africa. Annular to papular lesions are most common on the trunk and scrotum. Affected horses are systemically well. Lesions resolve spontaneously.

13.5 NODULAR SKIN DISEASES

Nodular skin diseases are common in the horse. The major differential diseases are infectious, sterile inflammatory, and neoplastic; accurate confirmation of a diagnosis is important for prognosis and successful treatment. Nodules may be well or poorly demarcated, solitary or multiple, soft or firm, and have intact overlying skin or tend to ulcerate and produce exudation.

- Primary lesions are nodules and papules.
- Secondary lesions include ulceration and discharging sinuses.
- Pruritus is typically absent or minimal.
- Skin biopsies are frequently important for diagnosis.

Ulcerative and/or exudative nodular skin diseases

Nodules that tend to ulcerate and exudate are common in horses.

Infectious ulcerative and/or exudative nodular skin diseases

Cutaneous habronemiasis

Cutaneous habronemiasis ('summer sores') is caused by the aberrant intradermal migration of the larvae of the helminths, *Habronema musca, Habronema majus* and *Draschia megastoma*. Adult worms live in the horse's stomach. Eggs and larvae pass into faeces, are ingested by fly stages, and subsequently deposited on moist or open areas on horses while flies are feeding. Disease occurs worldwide, and is most common in warm wet climates and summer months, and is uncommon in the UK. A hypersensitivity reaction to the larvae is proposed.

Clinical signs

- Lesion distribution: legs, ventrum, prepuce, medial canthus of eye, on wounds; a conjunctival form of disease is also common.
- Primary lesions: single or multiple nodules, often reddish brown in colour.
- Secondary lesions: nodules often ulcerate, and produce a haemorrhagic or serosanguinous discharge that may contain small yellow granules (necrotic debris, called 'kunkers'); exuberant granulation tissue can occur.
- Pruritus: mild to severe.

Diagnosis

Consistent historical and clinical signs, and confirmatory histopathology with larvae in the dermis surrounded by inflammation is diagnostic; however, larvae are often absent, and a nodular to diffuse eosinophil and mast cell rich dermatitis, with discrete areas of coagulative necrosis is more typical. Histopathology will clearly exclude many other differentials (e.g. sarcoids, bacterial or fungal granulomas, or squamous cell carcinoma).

Treatment

Ivermectin or moxidectin are variably effective at killing *Habronema* larvae, and are usually recommended (200 µg/kg orally, two doses given 3 weeks apart). Treatment may produce severe exacerbation of lesions; concurrent anti-inflammatory treatment is often recommended (e.g. prednisolone 1 mg/kg sid; or intralesional or topical glucocorticoids). Excess granulation tissue may be removed by conventional surgery or cryosurgery, although the location of lesions often makes this difficult. To prevent reinfection, immediate wound care and fly control are important (bandaging, strict manure management, fly repellents), together with regular worming programs. Larvae will die and lesions frequently spontaneously resolve in winter.

Other nodular parasitic infections

These generally occur in restricted locations around the world.

1. Hypodermiasis. This economically important disease in cattle is caused by the warble flies (*Hypoderma bovis* and *H. lineatum*) that are found in many countries in the northern hemisphere; and occasionally infect horses. Migrating larvae form nodules in the dermis, about 2 cm in diameter, often with a central breathing pore, most commonly over the withers. Diagnosis is made on the appearance of the lesion. The lesions can be poulticed or surgically lanced to express the warble fly.
2. Parafilariasis. The adult filarid *Parafilaria multipapillosa*, found in Eastern Europe, lives in subcutaneous nodules in infected horses. Nodules generally develop discharging tracts and a haemorrhagic exudate that contains eggs and larvae, often visible on cytology. Lesions occur over the neck, shoulders and trunk. The disease is more prevalent in spring and summer; and cases tend to recur annually for 3–4 years before resolving.
3. Dracunculiasis. Adult *Dracunculus* spp. worms, found in Africa, Asia and North America, live in subcutaneous nodules in a number of animal species, including the horse. Solitary nodules occur on the limbs or trunk, and often ulcerate, and release masses of larvae when in contact with water. Nodules may be surgically excised or the worms, up to 100 cm long, gradually removed by winding carefully onto a stick over several days.

Bacterial pseudomycetoma

Previously erroneously called botryomycosis, this uncommon nodular skin disease in the horse is caused by bacteria, typically *Staphylococcus* spp.

Clinical signs

- Lesion distribution: lower limbs or muzzle, at sites of previous skin trauma; occasionally spermatic cord or scrotum following castration.

- Primary lesions: typically solitary firm nodules.
- Secondary lesions; nodules often ulcerate and develop draining tracts; white to yellow granules (grains) may be present.

Diagnosis

Cytology typically reveals pyogranulomatous inflammation with numerous multinucleated giant cells, with or without bacterial cocci. Histopathology is diagnostic, with a nodular to diffuse suppurative to pyogranulomatous dermatitis to panniculitis, with central tissue granules and numerous bacterial cocci, often associated with Splendore-Hoeppli phenomenon. Bacterial culture from sterilely collected tissue biopsies will identify the causal organism.

Treatment

Systemic antibiotics (e.g. oral trimethoprim sulphur) often produce complete resolution if continued for 4–6 weeks or longer. Surgical excision is curative for solitary skin or spermatic cord. Lesions are often asymptomatic, and may also gradually resolve without treatment.

Deep cutaneous nodular mycoses

A variety of environmental fungi occasionally cause deep cutaneous infections in the horse; often acquired by traumatic implantation. Most infections are chronic but remain localized. Lesions are typically single to multiple nodules, more frequent on the muzzle or extremities.

Eumycotic mycetoma

These fungi form granules or grains in tissues, and are often soil-borne species (e.g. *Pseudallescheria boydii* (white-grain), *Curvularia geniculata* (black or dark grain)). Early, nodules are firm and asymptomatic, progressing to ulcerate or develop draining tracts with serous, purulent or haemorrhagic discharge and tissue grains. (Note. Some bacteria can also produce clinically similar discharging nodules with tissue grains or granules e.g. *Staphylococcus, Actinomyces, Nocardia*.)

Phaeohyphomycosis

These fungi are present as pigmented (dematiaceous) hyphae in tissues (e.g. *Bipolaris speciferum*, *Alternaria alternata*). Multiple lesions are more common, and nodules are typically firm, well-circumscribed, and asymptomatic.

Sporotrichosis

The dimorphic fungus *Sporothrix schenkii* forms pleomorphic yeasts in tissues. Organisms are found worldwide in soil and vegetation, although infection is very rare. Disease often starts with a local wound, often on medial aspects of limbs, and progresses to multiple firm nodules that progressively develop along regional lymphatic chains. Nodules may become large and ulcerate. Organisms are typically sparse in the horse, and the risk of zoonosis from tissue infection is low compared to infection in other animal species.

Alternaria dermatitis

Alternaria spp., produce non-pigmented broad hyphae in tissues; very rarely cause disease; characterized by multiple 1–2 cm diameter nodules, which may be alopecic or crusted.

Diagnosis

Fungal elements can be found during cytological examination of samples (aspirates or impression smears) from lesions. They are generally prevalent within tissue grains. *Sporothrix schenckii* are typically sparse and very difficult to find. Biopsy findings are characteristic for all deep fungal infections, with a nodular to diffuse pyogranulomatous to granulomatous dermatitis and panniculitis. Multinucleated giant cells are often present. Fungal culture is required for speciation; a zoonotic potential exists when culturing *Sporothrix schenckii*. Fungal in-vitro sensitivity testing should be requested to guide use of topical or systemic antifungal therapies.

Treatment

For single or few lesions, wide surgical excision is the treatment of choice; this is generally associated with a good prognosis. Adjunctive antifungal therapy can be considered, especially where complete excision is not possible, and should be based on sensitivity testing. Ketoconazole, itraconazole, amphotericin B, and iodides are more frequently reported antifungal choices. Sodium iodides have previously been reported as the treatment of choice for Sporotrichosis, administering a 20% solution of sodium iodide slowly intravenously daily for 2–5 days, followed by oral administration of organic or inorganic iodide; both routes at a dose of 20–40 mg/kg/day. Side-effects of treatment with iodides are uncommon; and the more expensive newer antifungals may be safer and efficacious, although only limited information on their use in the horse is available.

Pythiosis and zygomycosis

See also Chapter 19.

Pythiosis ('phycomycosis', 'swamp cancer') is caused by the aquatic fungal-like organisms *Pythium* spp. (e.g. *Pythium insidiosum*) that live on aquatic plant or organic substance. The infective motile zoospore is chemotactic towards damaged plant or animal tissues; infection requires prolonged water contact (ponds, lakes, swamps, flooded areas), and only occurs in wet tropical/subtropical areas, including the Gulf Coast of the USA, South America, Asia and northern Australia. Zygomycetes include a variety of ubiquitous fungal saprophytes found in soil and decaying vegetation, also most typically in tropical and subtropical areas, that may cause ulcerative nodular lesions in horses similar to Pythiosis. *Basidiobolus haptosporus* has been associated with large, ulcerative granulomas on the head, neck, chest and trunk, and *Conidiobolus coronatus* with ulcerated nodules on the external nares and in the nasal passages causing intermittent serosanguineous nasal discharge.

Clinical signs

- Lesions distribution: distal limbs, ventral abdomen, pectoral area.
- Primary lesions: nodules (rapidly progressive and invasive).
- Secondary lesions: ulceration, discharging sinuses containing white necrotic tissue ('leeches' or 'kunkers') with Pythiosis primarily, although occasionally with Basidiomycosis.
- Pruritus: often intense.

Diagnosis

A quick diagnosis is vital with pythiosis due to the rapidly progressive nature of this disease. *Pythium* spp. may be found in cytology aspirates or direct smears, especially from necrotic tissue ('kunkers') as broad septate, branching or non-branching hyphae; organisms may be very sparse, and cannot be readily distinguished on appearance from *Basidiobolus* or *Connidiobolus* spp. Histopathology for both diseases is typically a nodular to diffuse pyogranulomatous dermatitis and panniculitis, with a prominent eosinophilic component for pythiosis. Culture is rapid on blood agar or Sabouraud's dextrose agar, from tissue samples or 'kunkers', and allows clear identification of the causal organism. Immunohistochemical staining (immunoperoxidase technique) can be performed on formalin-fixed tissues, and is specific and rapidly diagnostic for pythiosis. Differentiation between pythiosis and zygomycosis may not be clinically important, as treatment guidelines are similar for both. Serological diagnosis is also possible.

Treatment

Aggressive early treatment is vital, with surgical excision to remove as much infected tissue as possible of major importance, often repeated over a number of days if continued necrosis occurs. Additional immunotherapy or systemic antifungals including amphotericin B are possibly effective. If treated early (i.e. within 2 weeks) response to vaccination is excellent (Pan American Veterinary Labs). Chronic lesions (>1–2 months) carry a guarded prognosis. Untreated disease is reported as invariably fatal.

Systemic mycoses

See also Chapter 19.

Some fungi cause deep mycoses, characteristically involving internal organs, and possibly with skin involvement. These fungi exist as saprophytes in soil and vegetation, typically in specific localized areas with unique environmental conditions.

1. Histoplasmosis (epizootic lymphangitis). The dimorphic fungus *Histoplasma farciminosi* is endemic in Africa, Asia and Eastern Europe, and infection occurs almost exclusively in horses, mules and donkeys. Fungal spores remain viable and infective in the environment for extended periods in moist temperate conditions. Skin trauma is important to infection. Insects may be important vectors. Skin nodules predominate; typically on the head and neck. Nodules rupture producing a greenish haemorrhagic exudate, then develop bright red granulation tissue; lesions occasionally follow lymphatic chains. Lesions gradually become more extensive over a number of weeks if not treated.
2. Cryptococcosis. *Cryptococcus* spp. are ubiquitous in the environment, although infection is very rare in the horse. *Cryptococcus neoformans* var *neoformans*, associated with pigeon roosts and droppings, is the most common species causing infection in USA and Europe. *Cryptococcus neoformans* var *gattii*, associated with certain species of eucalypt tree, is more prevalent in Australia, Asia, Africa, South America and Southern California. The most common presentations in the horse include rhinitis, nasal granulomas, and meningitis. Nodular dermal and subcutaneous masses occur rarely.
3. Blastomycosis. The dimorphic fungus *Blastomyces dermatitidis* is found in North America primarily, and also Africa and Central America in localized areas that have a unique combination of moisture, sandy acidic soil, wildlife and soil disruption. Infection is sporadic within these areas, and is extremely rare in horses. Multiple nodules with draining tracts are reported.
4. Coccidioidomycosis. The dimorphic fungus *Coccidioides immitis* is restricted to southwestern USA, Mexico, and Central and South America. Most humans and dogs in these areas are infected, although only few develop disease. Infection is rare in horses. Systemic signs in horses include chronic weight loss, coughing, musculoskeletal pain, and pyrexia; and skin lesions when present include abscesses.

Diagnosis

Cytology from skin nodules typically reveals pyogranulomatous to granulomatous inflammation, and most fungi are numerous (*Histoplasma*: small ovoid yeasts; *Cryptococcus* spp.: pleomorphic round to elliptical yeasts; and *Blastomyces*: round to oval, broad-based budding yeasts); fungi are seldom seen in lesions caused by coccidioidomycosis.

Histopathology typically reveals a nodular to diffuse pyogranulomatous to granulomatous dermatitis and panniculitis containing numerous organisms except for *Coccidioides immitis,* which are usually present but sparse. With cryptococcosis there may be a relatively acellular vacuolar dermal pattern.

Treatment

Prolonged administration of systemic antifungals including ketoconazole, itraconazole and amphotericin B can resolve deep mycoses in humans and small animals; however, in the horse such treatments would be extremely expensive and efficacy is currently unknown.

Glanders ('farcy')

See also Chapter 19.

This rare, contagious, highly fatal disease, caused by the obligate parasitic bacterium *Burkholderia mallei*, occurs in Asia, Africa and the Middle East, and is a reportable disease in many countries. Disease is characterized by nodule formation in various body organs including the skin, where 1–2 cm diameter nodules form, typically at sites of mild trauma. Nodules frequently ulcerate and produce a yellowish or honey-coloured exudate. Cytology typically reveals pyogranulomatous inflammation with numerous multinucleated giant cells, and plentiful Gram-negative bacterial rods. Treatment is generally not attempted and animals are euthanised. Glanders is a zoonosis; infection in people is frequently fatal.

Sterile ulcerative and/or exudative nodular skin diseases

Exuberant granulation tissue ('proud flesh') (See Chapter 23)

Exuberant granulation tissue which occurs commonly in wounds of the distal limb in horses prevents those wounds from healing. The inherent slow epithelialization of lower leg skin, frequent movement, and in some cases wound contamination, irritation, or infection with bacteria, fungi, or nematodes (e.g. *Habronema*) may all be involved in the pathogenesis. Clinical lesions are ulcerated nodules that progressively granulate and fail to epithelialise. Diagnosis is often suggested by the typical clinical signs at a wound site, although biopsy is necessary to exclude other potential or concurrent differentials, including habronemiasis, squamous cell carcinoma, and fibroblastic sarcoid. Treatment involves surgical excision of excessive granulation tissue, good wound hygiene, treatment of any secondary bacterial infections, avoidance of any topical irritants (including simple products like povidone iodine) and bandaging in some cases to limit further granulation tissue formation. Simple cleaning with saline or dilute chlorhexidine (0.05–0.1%) solution, and application of silver sulfadiazine (Flamazine®) topical cream twice daily is one of the author's preferred treatment plans after surgical removal of excessive tissue, with or without light bandaging. Every attempt should be made to prevent any inhibition of or disruption to epithelialisation by using good bandaging technique and using non-adherent dressings.

Neoplastic ulcerative and/or exudative nodular skin diseases

Sarcoids

Of fibroblastic cell origin, the sarcoid is the most common equine skin neoplasm, and is unique to horses. Bovine papillomaviruses appear involved in the pathogenesis, and insect transmission may be important. Young to middle-aged horses are most commonly affected, and there may be a genetic predisposition. Lesions are often persistent and / or recurrent although they do not metastasize.

Clinical signs

- Lesion distribution: head (especially periocular), legs, ventrum (axillae, girth, ventral abdomen, especially genital areas), often occur at sites of previous dermal injury; lesions may be single or multiple.

- Primary lesions: vary with the type of sarcoid:
  - verrucous (wart-like): scaly 'wart'-like papules and plaques.
  - fibroblastic: ulcerated sessile or pedunculated nodules.
  - occult (flat): alopecia, scaling, hypopigmentation or hyperpigmentation (most common on the face, neck and inguinal areas).
  - nodular: nodules.
  - malevolent: extension of nodular, fibroblastic or often mixed forms via lymphatics.
  - mixed: combinations of above.
- Secondary lesions: ulceration, crusting, alopecia.

Diagnosis

Although many lesions are presumed to be sarcoids based on classical appearance, biopsy for histopathology is required for confirmation as other tumours or inflammatory diseases can look identical. Histopathology reveals typical hyperplasia of the epidermis and spindle-cell fibroblastic invasion of the dermis. It is suspected that biopsies may exacerbate previously inactive lesions, especially occult or small verrucous forms, so confirmation of disease is often not performed. PCR testing using shave samples is a recent new alternative to full thickness biopsies that may be useful in such cases.

Treatment

Sarcoids remain notoriously difficult to treat despite a range of treatment options. Wide surgical excision is the treatment of choice, however the location of many lesions often makes this impossible; and recurrence rates remain high. Small verrucous and occult forms will sometimes remain quiescent for many years, and observation without treatment may be recommended. Traditional therapies used as alternatives to surgery, or after tumour debulking include cryosurgery, BCG or autogenous vaccines, and intralesional radiation; each reported with variable success rates. Cryosurgery is often the most readily available of these options. An accurately timed 'quick-freeze, slow-thaw' cycle should be performed at least twice. Liquid nitrogen is often used, and may be applied by probe or spray. Sloughing of the lesion occurs during the following 3–6 weeks. BCG (Bacillus Calmette–Guérin) is a sterile bovine mycobacterial preparation, injected intralesionally to stimulate a local cell-mediated immune response. It is injected repeatedly, 2–3 weeks apart until resolution occurs. There may be some risk of anaphylaxis, and premedication with a steroidal (e.g. dexamethasone) or non-steroidal anti-inflammatory drug (e.g. flunixin) is suggested. Swelling, hyperaemia and sloughing may occur after the second treatment. Intralesional chemotherapeutics (e.g. cisplatin or carboplatin) are often difficult to inject in large volumes into dense lesions, with associated hazards for involved personnel. Newer topical therapies include Imiquimod (Aldara®), an immune-enhancer in a gel form, which has recently been reported very effective treatment for some sarcoids, especially superficial (i.e. dermal) forms. 5-fluorouracil has been used topically and intralesionally; however, little information is available. A heavy-metal preparation from Liverpool, England (Liverpool Cream), is anecdotally reported as often effective but no safety or efficacy studies are available.

Prognosis

The general prognosis for treatment of sarcoids is poor, as recurrence is common regardless of treatments. More superficial lesions, and lesions treated aggressively from the outset carry a better prognosis. Treatments should be repeated continually or changed to alternative options until there is a complete and sustained response. Cessation of treatment after partial response commonly results in recurrence.

Squamous cell carcinoma

This form of ultraviolet light-associated neoplasia is very common. It occurs predominantly at unpigmented sites, and typically in older, light coloured horses. Carcinogenic effects of equine smegma may also be involved. Many lesions are locally aggressive but they rarely metastasize.

Clinical signs

- Lesion distribution: head, third eyelid, mucocutaneous junctions, genitalia.
- Primary lesions: ulcers, wart-like or ulcerated nodules; usually solitary.

Diagnosis

Typical appearance at non-pigmented sites is highly suggestive, however biopsy for histopathological confirmation is essential.

Treatment

Wide surgical excision is the most common form of treatment although adjunctive cryosurgery, 5-fluorouracil or radioactive implants are often used. Adjunctive systemic treatment with non-steroidal anti-inflammatory drugs with activity against cyclo-oxygenase-2 (COX-2) may be helpful in some cases. Prognosis is poor without complete removal. The margins of excised tissue should be examined for tumour cells to determine if removal was complete.

Nodular to diffusely swollen skin diseases

These diseases are characterized by regional swelling and poorly demarcated nodules. A number of important infectious causes should be considered.

Abscesses and cellulitis

A variety of ubiquitous bacteria normally found in the environment or as skin flora can cause deep cutaneous infections, typically associated with skin trauma, resulting in cellulitis, nodular swelling, and/or abscesses. Common anaerobic bacteria (e.g. *Bacteroides* spp., *Fusobacterium* spp., *Clostridium* spp.) invade traumatic puncture wounds. Rapid progression of swelling, necrosis and putrid odour are common, and horses are frequently unwell. *Clostridium* spp. infection classically follows intramuscular injection, and may produce extensive skin ulceration and sometimes crepitus from gas production. Associated toxaemia is often fatal without treatment. Less commonly, *Streptococcus* spp. (e.g. *S. equi equi*; associated with equine respiratory disease ('strangles')), and *Actinomyces* spp. (normal anaerobic flora in the oral cavity and upper respiratory tract) cause abscesses in the retropharyngeal or submanidular areas. *Staphylococcus* spp. (normal skin flora); *Actinobacillus* spp. (normal oral and upper respiratory tract flora); and *Nocardia* spp. in immunocompromised patients (widespread normal environmental saprophytes) all rarely cause solitary to multiple cutaneous abscesses with draining tracts, and regions of cellulitis. *Corynebacterium pseudotuberculosis* can cause deep subcutaneous abscesses ('false strangles', dryland distemper or pigeon fever), typically in the pectoral and ventral abdominal areas. This disease is largely restricted to western USA. The abscesses discharge creamy or caseous purulent exudate, with associated oedema, fever and lameness. *Rhodococcus equi* (associated with pneumonia and lymphadenitis in foals) will occasionally cause single subcutaneous abscesses or regions of cellulitis, especially on the chest, axillary regions, or hind legs.

Diagnosis

Clinical appearance is often suggestive of infection, although bacterial culture is required to confirm the bacterial species involved. Ideal

samples are tissue biopsies collected with sterile technique; fine needle aspiration from areas of cellulitis or intact nodular swellings may also provide diagnostic results. It is important to remember normal flora and surface contaminants are often easily cultured, especially from draining tracts and surface exudates when completely incidental to the disease. Results of bacterial culture should always be supported by cytology findings, ideally using fine needle aspirates from intact nodules or cellulitic regions. Cytology also provides a more immediate indication of the likely species involved based on gram staining and appearance of organisms. Nocardia organisms are distinctively partially acid-fast.

Ulcerative lymphangitis

This is a rare disease caused by bacterial infection of the cutaneous lymphatics; it is most commonly associated with wounds, poor hygiene, and *Corynebacterium pseudotuberculosis.* Other bacteria occasionally implicated include *Streptococcus* and *Staphylococcus* spp., *Actinomyces pyogenes,* and *Rhodococcus equi*.

Clinical signs

Initial oedematous swelling of one or more limbs, often the hind limbs, is followed by painful nodules that ulcerate and develop draining tracts with purulent discharge. The regional lymphatics are corded, and there is frequently lameness and debilitation. Fibrosis and disfigurement often occur.

Diagnosis

Diagnosis relies on cytology and histopathology findings, with a diffuse to nodular suppurative to pyogranulomatous dermatitis, often with numerous bacterial rods or occasionally cocci, and bacterial culture from tissue biopsies.

Treatment

Aggressive treatment must be commenced early in the disease course to avoid excessive fibrosis. High doses of procaine penicillin for prolonged periods may be effective together with exercise, hydrotherapy, and surgical drainage. Bacterial culture and sensitivity results should guide antibiotic therapy.

Anthrax

Bacillus anthracis causes this acute infectious disease which typically results in sudden death. Infection can occasionally cause cutaneous lesions, mostly painful, hot, oedematous regions that develop necrosis and a characteristic black eschar. Diagnosis is confirmed by histopathology and bacterial culture.

Nodular to papular skin diseases without ulceration

This group of diseases has papules and smaller nodules that tend to remain covered by intact skin.

Infectious nodular to papular skin diseases without ulceration

Bacterial pyoderma – deep

Typically caused by *Staphylococcus* spp., deep bacterial pyoderma (folliculitis and furunculosis) is a common cause of nodular skin disease in the horse. Other bacteria occasionally implicated include *Streptococcus* spp. and *Corynebacterium pseudotuberculosis.* Multiple papules to small nodules that occasionally ulcerate and crust are most common at mild trauma sites, especially the saddle and tack areas. Diagnosis requires histopathology (suppurative to pyogranulomatous dermatitis with folliculitis and furunculosis) and bacterial culture. Treatment and management guidelines are similar to superficial bacterial infection (see alopecic, scaling and crusting skin diseases), although systemic antimicrobials are needed for at least 4–6 weeks for established infections, with oral trimethoprim sulphur (30 mg/kg bid) most frequently recommended.

Papillomatosis

Caused by equine papilloma viruses of at least two subtypes, papillomatosis ('warts') is most common in young horses, typically < 3 years of age; or older immunocompromised horses.

Clinical signs

Lesion distribution:

- Verrucous form: muzzle and lips.
- Aural plaques: inner pinnal surfaces.

Primary lesions:

- Verrucous form: papules to small nodules, typically multiple, with scaly verrucous appearance; up to 2 cm diameter.
- Aural plaque form (less common): multiple small grey to white plaques.

Diagnosis

The clinical appearance is usually highly suggestive, although biopsy for histopathology is required for definitive diagnosis.

Treatment

Papillomatosis is usually self-limiting with complete resolution in 2–4 months, and treatment rarely indicated; although aural plaques may persist. Surgical excision or cryotherapy may be used for problematic lesions. Imiquimod (Aldara®), an immune stimulant available in a topical gel, is used for viral papillomas in humans, and can be considered for treatment of persistent lesions.

Molluscum contagiosum

Caused by a molluscipox virus, infection in the horse occurs rarely worldwide, although appears non-contagious between horses. Possible transmission from infection in people is suggested. Affected horses typically have hundreds of papules, predominantly on the chest, shoulders, neck and limbs, although sometimes restricted to localized areas (e.g. prepuce, muzzle). Papules may be haired initially, although become alopecic, and are typically covered with a powdery white scale. Some develop a characteristic central pore with a caseous plug. Papules are not pruritic or painful. Diagnosis is suggested by clinical appearance, and supported by typical histopathology from biopsies, although viral confirmation requires electronmicroscopy. Viral isolation has not been possible so far. Lesions may spontaneously regress, although often persist for months to years. No successful treatment has been reported.

Sterile nodular to papular skin diseases without ulceration

Urticaria

Urticaria is a common entity in the horse, characterized by sudden onset of multiple oedematous papules to nodules, and associated with a wide variety of underlying causes. Oedema results from mast cell degranulation via immunological or non-immunological mechanisms induced by drugs (systemic or topical e.g. antibiotics, vaccines), hypersensitivities (atopy, food reactions, insect hypersensitivity), physical factors (cold, heat, pressure), psychological factors (stress, excitement), and infections (bacterial, fungal, viral). Approximately 50% of cases remain idiopathic.

Figure 13.11 Urticaria: clinical. Multifocal circular raised lesions, typical of urticaria, that pit with digital pressure.

Clinical signs

- Lesion distribution: head, neck, shoulders and thorax; regional or generalized.
- Primary lesions: multiple pitting wheals, circular to irregular; small circular (2 mm) to giant (20–30 cm) single or coalescing (Figure 13.11); lesions transient (few hours), although continual new lesions may produce persistent clinical signs.
- Secondary lesions: occasionally surface serum oozing if oedema severe.
- Pruritus: usually absent.

Diagnosis

Characteristic clinical lesions that pit on digital pressure confirm urticaria. A range of further diagnostic tests and trials are indicated to search for the underlying cause, including drug withdrawal, intradermal testing for atopy, and food trials, particularly with persistent or recurrent cases. Histopathology is generally unrewarding.

Treatment

Some untreated cases resolve spontaneously. Orally administered antihistamines can be helpful for acute or chronic cases (e.g. chlorpheniramine 0.25–0.5 mg/kg bid; diphenhydramine 1–2 mg/kg bid-tid; hydroxyzine 1–2 mg/kg bid-tid [discontinued in some countries]). Glucocorticoids are often helpful (e.g. dexamethasone 0.1 mg/kg sid IM; oral prednisolone 1 mg/kg sid); however, side-effects should be considered, especially for long-term treatment.

Erythema multiforme

This poorly understood disease is uncommon in the horse. It is typically acute, self-limiting, and caused by individual keratinocyte cell death associated with multiple triggers, including drugs, infections, and neoplasia. Lesions, often bilaterally symmetrical on the trunk and legs with no associated pain or pruritus, include macules, papules, urticarial plaques, and classical 'target lesions' with a peripheral raised erythemic rim and central resolving area. Biopsy for histopathology is diagnostic. Spontaneous resolution is typical.

Equine eosinophilic granuloma

Also called nodular necrobiosis, this common inflammatory nodular skin disease in the horse is of unknown aetiology, although insect hypersensitivity and repetitive trauma (from saddles and tack) may be involved. Clinical lesions are multiple firm nodules of variable size, most common on the withers and back with no associated pruritus or pain.

Diagnosis requires skin biopsies for histopathology, with a nodular eosinophilic dermatitis and granuloma formation. Treatment involves removing repetitive trauma when applicable (e.g. ill-fitting saddles), and immunosuppressive treatments. The author has had success with temporary cessation of riding, and potent topical glucocorticoids ointments (e.g. mometasone 0.15% or triamcinolone 0.02% bid for 2–3 weeks, then gradually reduced), although debulking may be necessary for larger lesions where mineralization is likely. Intralesional triamcinolone is also reported successful. Systemic prednisolone can be helpful for multiple lesions, although recurrence may occur after treatment is ceased.

Amyloidosis

Amyloidosis is a term used to describe a series of diseases characterized by deposits of an extracellular proteinaceous material (amyloid) in tissues. Both systemic and localized forms have been described. In

secondary reactive amyloidosis, fragments of serum amyloid AA (an acute phase protein) are deposited in tissues. Thus, any chronic inflammatory condition can predispose to this form of amyloidosis. Amyloid L (AL) proteins are monoclonal immunoglobulin light chains (or fragments of these) and are associated with primary idiopathic amyloidosis and the amyloidosis occasionally observed in animals with multiple myelomas.

Systemic amyloidosis

Usually of the AA type and occurs secondary to chronic immune stimulation. In horses, has been associated with chronic pleuropneumonia, peritonitis, chronic parasitism and hyperimmunization. Amyloid is deposited primarily in the liver and spleen, ultimately impairing the function of those organs. Diagnosis is confirmed by biopsy or at necropsy.

Nasal amyloidosis

May be AA or AL type. Present with amyloid deposits in the subcutaneous tissues of the head, neck and trunk or in the nasopharyngeal submucosa and ventral turbinates. Nasal amyloidosis has been associated with epistaxis. Diagnosis confirmed by biopsy with special stains.

Neoplastic nodular to papular skin diseases without ulceration

Melanoma

Melanomas are common neoplasms of the older horse, arising from melanocytes or melanoblasts, and may be benign or malignant. Melanomas occur almost exclusively in horses turning dappled grey and then white as they age; horses may be any colour initially.

Clinical signs

- Lesion distribution: perineum, ventral tail surface, periocular region, distal limbs.
- Primary lesions: pigmented nodules, single or multiple, dermal or subcutaneous, with three patterns of behaviour: slow growth for years without metastasis, slow growth for years then suddenly rapid growth, and rapid growth and malignancy from onset.
- Secondary lesions: nodules may ulcerate.

Diagnosis

Clinical appearance is often highly suggestive, and cytology from fine needle aspirates further indicative. Biopsy for histopathology is required for absolute confirmation of diagnosis.

Treatment

With older animals and apparently benign lesions, treatment is rarely undertaken, as their slow growth is rarely problematic. Prognosis is very poor for more aggressive lesions, and for lesions on non-grey horses. Surgical debulking can be palliative, along with cryotherapy, intralesional cisplatin, or orally administered cimetidine to slow down the rate of growth in rapidly growing melanomas.

Lymphosarcoma

This uncommon neoplasm of middle-aged to older horses typically presents with multiple subcutaneous nodules on the trunk. Horses may have internal involvement and be systemically ill; although some may survive years with cutaneous lesions alone. Diagnosis requires histopathological examination of skin biopsies.

13.6 SKIN DISEASES CHARACTERIZED BY DEPIGMENTATION

Vitiligo

Vitiligo, caused by an acquired presumed immune-mediated destruction of melanocytes, occurs most commonly in Arabian horses, especially in greys ('Arabian fading syndrome', 'pinky syndrome') but can occur in any breed. It may be hereditary. Macular areas of skin and/or hair depigmentation occur most commonly on the muzzle, lips and periocular areas, which may wax and wane, and occasionally spontaneously resolve, however mostly persist. Histopathology from skin biopsies often reveals a lack of melanocytes in affected regions, and occasionally a mild lymphocytic interface dermatitis.

Leucoderma/leucotrichia

Depigmentation of the skin (leukoderma) and/or hair (leukotrichia) commonly occurs in any breed of horse following localized trauma or inflammation. This change may be temporary or permanent.

Reticulated leucotrichia

This uncommon pigmentation disorder reported in Quarter Horses, Thoroughbreds and Standardbreds most commonly, is proposed to have a genetic link. Most horses become affected as yearlings, with sudden onset of linear crusts in a crosshatch or net-like pattern over the back, followed by permanent re-growth of white hair.

Spotted leucotrichia

This idiopathic disorder, reported most commonly in Arabian horses although also in many breeds, is characterized by leukotrichia over the rump and sides, typically without depigmentation of underlying skin. Typically there is no history of preceding trauma or inflammatory diseases at the affected spots. Leukotrichia is permanent.

13.7 CONGENITAL AND DEVELOPMENTAL SKIN DISEASES

Aplasia cutis

This disorder is associated with a regional absence of skin due to an autosomal recessive genetic defect. Strictly defined areas are affected, especially on the limbs. Euthanasia is generally recommended, as lesions are often extensive. Foals with mild lesions can survive.

Albinism and other congenital pigmentation disorders

Albinism is a congenital absence of normal pigmentation due to an autosomal recessive enzymatic defect preventing normal production of melanin from melanocytes. Affected horses lack pigment in hair and skin and may have pink irides. Waardenburg-Klein syndrome occurs in American paint horses, with lack of pigment in skin and hair, blue irides, and possible deafness. Lethal white foal disease occurs in Overo paint horses, and along with lack of pigment in hair and skin there are serious developmental defects of the digestive tract (e.g. colonic atresia), incompatible with life. Lethal lavender foal syndrome occurs rarely in Arabian horses; foals are born with a bluish or

purplish hair coat and a range of neurological defects incompatible with life.

Congenital cysts

A variety of congenital cysts occur occasionally, including epidermoid inclusion cysts (atheroma) of the false nostril, dermoid cysts of the dorsal midline (contain hair and caseous material), and temporal teratoma adjacent to the petrous part of the temporal bone.

13.8 MISCELLANEOUS SKIN DISEASES

Purpura haemorrhagica (see Chapter 10)

This syndrome represents the most common form of cutaneous vasculitis in the horse. Incidence is low and sporadic. Aetiology is largely unknown, although it may occur within 2 to 4 weeks of bacterial infections (*Streptococcus equi equi, S. zooepidemicus*). A range of other factors can induce vasculitis in other species, including drugs, other infectious agents, and neoplasia, although ~50% of cases remain idiopathic.

Clinical signs

- Lesion distribution: distal limbs (typically all four) and ventral abdomen predominantly; sometimes also face, muzzle.
- Primary lesions: wheals (urticaria), initially or concurrently.
- Secondary lesions: extensive oedematous swelling, especially of limbs, typically well-demarcated; surface serous exudation; later skin necrosis and ulceration may occur if severe; petechiae and ecchymoses may occur on mucous membranes.
- Concurrent signs of systemic illness: reluctance to move, anorexia, pyrexia, tachycardia, tachypnoea.

Diagnosis

The collection of typical clinical findings defines this syndrome, which is usually readily recognised. Deep skin biopsies for histopathology are required to confirm vasculitis; although multiple and/or repeat samples may be needed for confirmation. Diagnostics to search for underlying factors/diseases are indicated in persistent cases.

Treatment

Disease may be mild, transient and self-limiting, or severe and/or persistent. Aggressive early treatment is indicated for severe disease to limit tissue damage and scarring. Glucocorticoids are the mainstay of treatment; prednisolone (2–4 mg/kg sid orally until clinical remission then tapered); dexamethasone (0.2–0.4 mg/kg sid may be more effective in recalcitrant cases); and occasionally triamcinolone (not recommended by the author). A concurrent management plan to reduce the risk of laminitis is important, especially when triamcinolone is used for treatment. This includes low energy diets, weight control, and regular mild exercise. Supportive care, including hydrotherapy, walking, and good skin hygiene (hosing, antibacterial shampooing/solutions); and minimizing factors likely to exacerbate vasculitis (excessive stress, heat or cold exposure; stasis in blood vessels [physical inactivity]) are important.

Equine Cushing's disease (see Chapter 9)

This disease is almost exclusively restricted to older horses, and typically presents with non-cutaneous signs including a sway back associated with muscle wasting, poor dentition, and/or laminitis. Hypertrichosis is common, with long thick coarse coats (persistent 'winter coat').

Pastern dermatitis (greasy heal)

This extremely common entity in the horse is caused by multiple underlying diseases, despite similar clinical signs, and should be investigated as for dermatitis elsewhere on the body. The *most common* diseases causing pastern dermatitis include:

- Superficial bacterial pyoderma (very common secondary and perpetuating factor).
- Dermatophilosis.
- Chorioptes (especially in heavy-feathered lower legs).
- Physical factors (e.g. insect bites, wetting/ skin maceration, mild repetitive trauma, irritants).
- Contact irritants (e.g. plants, topical treatments).
- Vasculitis (possibly photoactivated).

Clinical signs

- Lesion distribution: pastern area, palmar or dorsal surfaces or circumferential, mostly multiple limbs; white skin often more severely affected regardless of underlying disease.
- Primary lesions: rare papules, pustules.
- Secondary lesions: alopecia, erythema, scaling with mild cases; progressing to ulceration, crusting, swelling with chronicity.

Diagnosis

The most appropriate diagnostic tests are often surface cytology (impression smears for moist lesions; tape impressions for dry), superficial skin scrapings, and sometimes skin biopsies. Treatment of secondary bacterial infections is important in many cases before considering biopsies.

Treatment

Treatment of secondary bacterial infections is often important (see alopecic, scaling, and/or crusting skin diseases), and together with minimizing irritants (many topical preparations can irritate) will often allow good control or resolution of signs when primary factors are relatively insignificant (e.g. repetitive skin trauma or frequent wetting). If multiple horses are affected or if lesions recur, chorioptic mange mites are often the cause. Treatment for this condition has been previously discussed in this chapter.

FURTHER READING

Knottenbelt DC (2009) Pascoe's principles and practice of equine dermatology. Saunders, London

Pascoe RR, Knottenbelt DC (eds) (1999) Manual of equine dermatology. Saunders, London

Scott DW, Miller WH (eds) (2003) Equine dermatology. Elsevier, St Louis, MO

Chapter 14

Reproduction

Carlos Pinto, Grant S. Frazer

CONTENTS

14.1 The non-pregnant mare 283
Stages of the oestrous cycle 283
Examination of the mare for breeding soundness 285
Diseases of the mare's reproductive tract 287
14.2 The pregnant mare 292
Complications during pregnancy 294
Elective termination of pregnancy 296
14.3 Parturition 296
Endocrinology of parturition 296
Induction of parturition 296
14.4 Dystocia 297
Obstetric procedures 297
14.5 The postpartum period 297
Postpartum complications 297
14.6 The stallion 298
Examination of the stallion for breeding soundness (BSE) 298
Abnormalities of the genital tract 300
Abnormal reproductive behaviour in stallions 303
Ejaculatory dysfunction 303
Pharmacological aids 303
14.7 Reproductive management 303
Artificial control of photoperiod 304
Vernal transition 304
Breeding season 304
Aberrant patterns of cyclicity 304
Foaling mares 304
Lactational anoestrus 304
Pregnancy losses 304
Manipulation of the oestrous cycle 304
Teasing programmes 305
Breeding management 305
Artificial insemination (AI) 305
Advanced assisted reproduction 306
Further reading 307
Appendix 1 307
Appendix 2 308

14.1 THE NON-PREGNANT MARE

The oestrous cycle is normally 21–22 days. Most mares are seasonally polyoestrus (cycle April–September) and long-day breeders. Reproductive activity is strongly influenced by the photoperiod. Ovarian activity usually begins with increasing daylight and ceases under conditions of decreasing light. In the winter, artificially extending the photoperiod in the evening suppresses melatonin release by the pineal gland, thus stimulating ovarian activity. Low melatonin concentrations in the mare permit gonadotrophin (LH, FSH) stimulation of ovarian activity.

Stages of the oestrous cycle

Oestrus

- Usually lasts 5–7 days; oestrus behaviour typically ceases 24–48 hours after ovulation.
- Mare is sexually receptive – stands to be mounted, seeks stallion's attention, squats, raises the tail, rhythmically everts the clitoris ('winking') and urinates, does not flatten ears or kick (Figure 14.1).
- The cervix becomes increasingly pink, moist, relaxed, open and oedematous. When it is fully relaxed, the caudal portion of the cervix lies on the floor of the vagina.
- The uterus is oedematous and flaccid. On ultrasonographic examination, the oedematous endometrial folds make the cross-section of the uterine horn resemble a sliced tomato or a wagon wheel (Figure 14.2). Endometrial oedema declines as the follicle matures and approaches ovulation and it is typically minimal at ovulation.

© 2013 Elsevier Ltd
DOI: 10.1016/B978-0-7020-2801-4.00014-6

Figure 14.1 Mare in oestrus being teased over the fence by a stallion – tail raised, everting clitoris and squirting urine.

Figure 14.2 Split-screen ultrasound images from an oestrual mare obtained by transrectal ultrasonography using a 5-MHz, linear transducer. Right panel: cross-section of a uterine horn with significant endometrial oedema; left panel: preovularory follicle filled with characteristic anechoic fluid.

- Characterized by high concentrations of oestrogen and luteinizing hormone (LH) and basal concentrations of progesterone. Follicles often soften 24 hours prior to ovulation, but some become turgid again prior to ovulation. The mare is frequently sensitive to the palpation of the ovary at this time due to the relatively large diameter of the preovulatory follicle and its proximity to the broad ligament. The preovulatory follicle increases from approximately 30 mm 6 days prior to ovulation to an average of 45 mm by the day of ovulation. At the height of the breeding season, however, it is not unusual for a mare to be in oestrus for only 3–4 days and ovulate a follicle of less than 35 mm in diameter. On ultrasonographic examination the pre-ovulatory follicle becomes less circular and tends to 'point' towards the ovarian fossa – the ultimate site of rupture for follicles in the mare.

Figure 14.3 Ultrasound images from a mare in diestrus obtained by transrectal ultrasonography using a 5-MHz, linear transducer. The image depicts a characteristic hyperechoic corpus luteum 3 days post ovulation.

- Following ovulation, the follicular cavity fills with blood and is termed a corpus haemorrhagicum (CH). Double ovulations occur in approximately 16% of cycles with the highest incidence in Thoroughbreds, Warmbloods and draught horses. The average interval between ovulations is 1 day with a range of 0–5 days.

Dioestrus

- Usually lasts 14–15 days.
- Mare actively rejects the stallion – clamps tail down, swishes tail, flattens ears, strikes, squeals, bites.
- A corpus luteum (CL) is present on the ovaries. The CL may be fluid-filled (50%) or non-fluid-filled (Figure 14.3). The CL is not normally palpable in the mare.
- The cervix is pale, firm, dry and closed. The caudal portion of the cervix is positioned high in the vaginal lumen, lifted by the dorsal cervical ligament.
- Uterine tone increases, and the uterine horns become tubular.
- Characterized by high circulating concentrations of progesterone that rise sharply following ovulation. Prostaglandin F-2α, released from the uterus on day 14 or 15, causes luteolysis and return to oestrus.
- Mares can have considerable follicular growth during dioestrus. Ovulation occasionally occurs during dioestrus despite the presence of high concentrations of progesterone and absence of oestrous behaviour.

Vernal and autumn transition stages

- Occurs before and after seasonal anoestrus.
- In early spring, oestrous behaviour may be observed intermittently or continuously for several weeks; however these transitional cycles are anovulatory. The first ovulation of the season is generally followed by normal, cyclic activity.
- Clusters of large follicles (>30 mm) are often found on the ovaries, but there is no ovulation.
- The cervix does not close tightly until the mare ovulates.

Seasonal anoestrus

- Often the mare is passive to the stallion or may even show some mild signs of oestrus.
- The ovaries are inactive.
- The cervix is moderately closed or thin and open.
- The uterus is flaccid.
- Progesterone and oestrogen concentrations are low.

Examination of the mare for breeding soundness

History

1. General:
   Age.
   Health.
   Breed.
   Management.
2. Reproductive:
   Regularity and length of oestrous cycles.
   Behaviour.
   Breeding dates – artificial insemination (AI) or natural cover, stallion fertility.
   Foaling date, normality, occurrence of retained foetal membranes.
   Indications of previous uterine infection and treatment.

General physical

Check for the presence of any painful or chronic conditions, especially those related to the musculo-skeletal system.

Examination of external genitalia

1. Vulva: the vulvar lips should meet evenly. For maximal function the dorsal commissure of the vulva should be no more than 4 cm above the pelvic floor; at least 2/3 of the vulva should be positioned below the pelvic floor There should be a cranial-to-caudal slope of no more than 10° from the vertical (Figure 14.4).
2. Discharge on the vulvar lips, tail or hindquarters.
3. Clitoris: can harbour contagious equine metritis (CEM) organism (*Taylorella equigenitalis*) or other venereally transmissible organisms. Use a standard hospital-type swab to sample the clitoral fossa. A narrow-tipped (paediatric-type) swab is then used for the central and, if present, lateral clitoral sinuses (Figure 14.5). The swabs are then placed in Amies charcoal transport medium and kept at 4 °C until their arrival at the laboratory. If more than one mare is being swabbed a disposable glove should be worn on the hand used to evert the clitoris.
4. Mammary glands: assess capability to lactate normally.

Examination of internal genital tract

Palpation per rectum and transrectal ultrasonography:

1. Cervix: correlate shape and consistency with cycle stage.

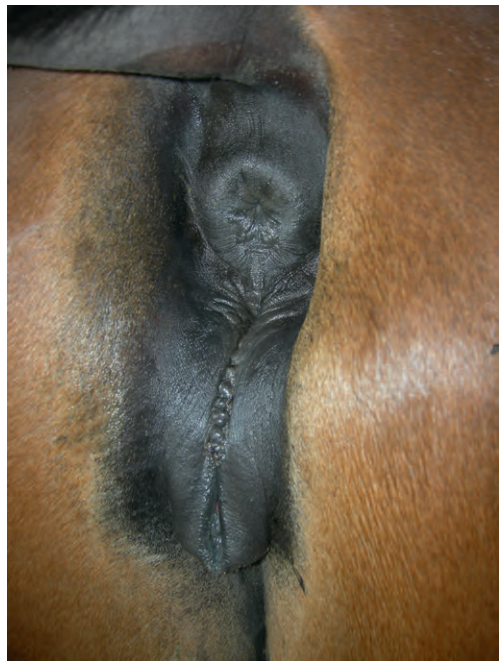


Figure 14.4 Poor perineal conformation. Anus is recessed and the vulvar lips are sloped.

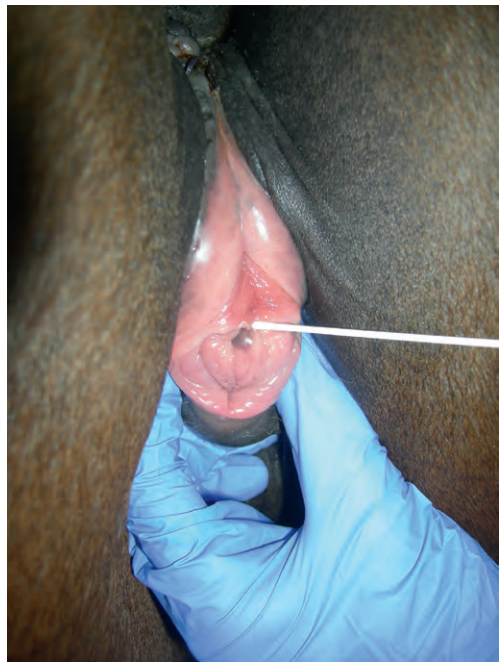


Figure 14.5 Swabbing the central clitoral sinus with a narrow-tipped swab.

2. Uterus and endometrial folds. Check for:
   Presence of free fluid in the lumen.
   Presence of endometrial cysts.
   Presence of endometrial oedema.
   Sacculations: commonly at body-horn junction.
   Pregnancy; embryonic vesicles measuring 5 mm in diameter (~10–11 days of pregnancy) are detectable by most available ultrasound equipment.

Figure 14.6 Double-guarded swab. Once the swab is in the uterus the inner tube is advanced through the tip of the outer tube. The cotton swab is then advanced and held in apposition to the endometrium for a few seconds. The swab and then the inner tube are retracted inside the outer tube before withdrawing the swab.

3. Ovaries.
   Size, shape.
   Follicles, CH and CL (not readily palpable).
   Presence of ovarian fossa.

Endometrial swabbings for microbiological culture

Bandage the mare's tail or place in plastic sleeve. Wash perineum thoroughly with mild soap or povidone-iodine solution, rinse and dry. Use sterile sleeve or clean sleeve and sterile surgeon's glove. Lubricate with a small amount of sterile water-soluble lubricant. Use double-guarded swab (Figure 14.6). Try to avoid sampling the uterus during dioestrus because of high susceptibility to infection at this stage of the cycle. If examination of the mare is necessary during dioestrus, administer a luteolytic dose of $PGF_{2\alpha}$ at the end of the procedure.

Endometrial cytology

Obtain smear from aspirated uterine fluid or from swab. Cytology brushes are also available and provide better sampling of endometrial cells for the cytology smear. Stain with 'Diff-Quik'. The presence of neutrophils suggests active endometritis. Fungal elements, if present, are readily observed in the endometrial cytology smear (Figure 14.7)

Vaginal examination

1. Speculum: the vestibulovaginal seal serves as a barrier to ascending bacterial infection. This seal is best tested during oestrus using a mildly lubricated speculum. If the seal is effective, resistance will be met. The windsucker test can also be performed to evaluate the integrity of the vestibulovaginal seal. When the labia are gently parted, mares with a weak, incompetent vestibulovaginal seal produce a characteristic sound, indicating air inrushing into the vagina (windsucker). If the mare has a competent vestibulovaginal seal, air does not enter the vagina.
   Appearance of cervix and vagina.
   Urovagina.
   Source of exudates.
   Vaginal varicosities in region of vestibulovaginal seal (Figure 14.8).
2. Digital:
   Cervical tear or adhesions.
   Vaginal tears, adhesions or small rectovestibular fistulas.

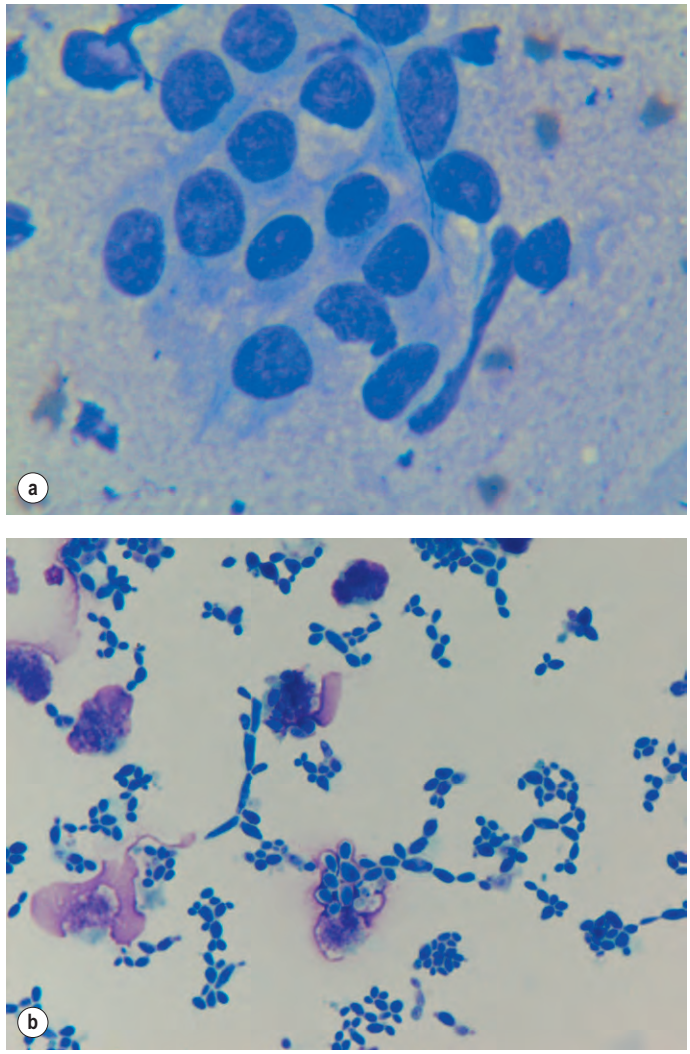


Figure 14.7 Endometrial cytology. Smears were stained with Romanowski (Diff-Quik) stain. (a) Endometrial epithelial cells from mares in oestrus. (b) Endometrial epithelial cells interspersed with fungal elements (yeast) at various stages of development ('budding').

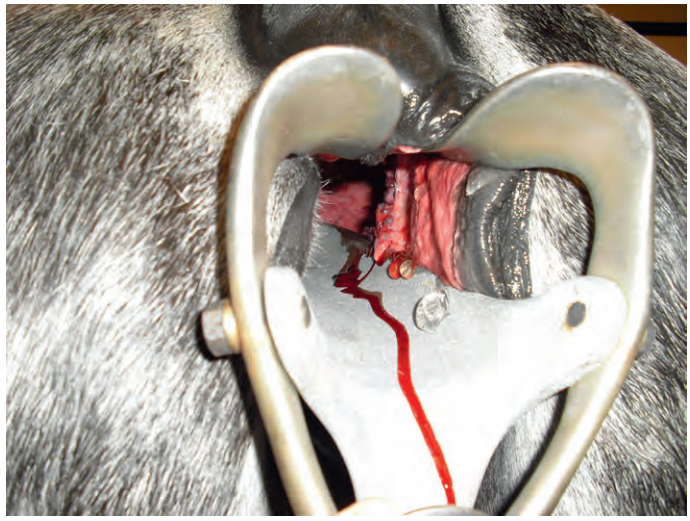
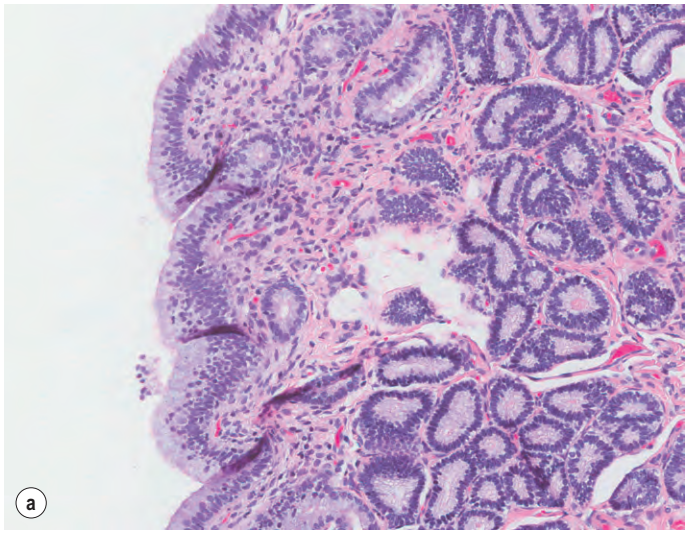


Figure 14.8 Varicose veins in the vestibular area of a mare.



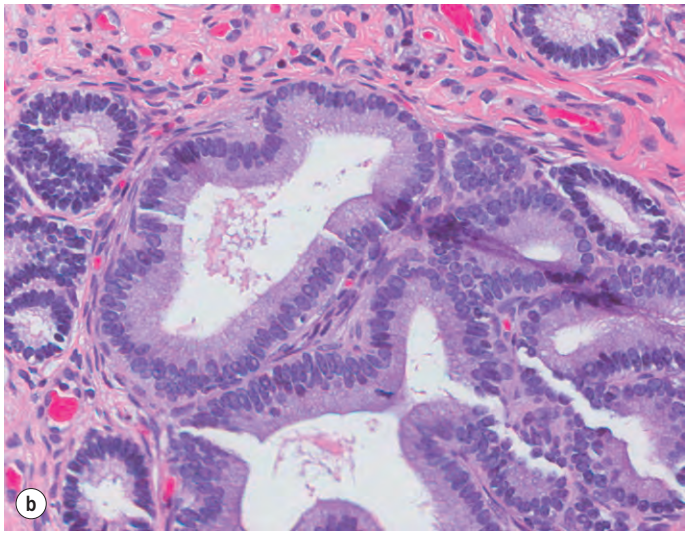


Figure 14.9 (a) Endometrial biopsy showing a heavy infiltration of inflammatory cells into the endometrial stroma. (b) Endometrial biopsy showing severe cystic glandular distension with periglandular fibrosis.

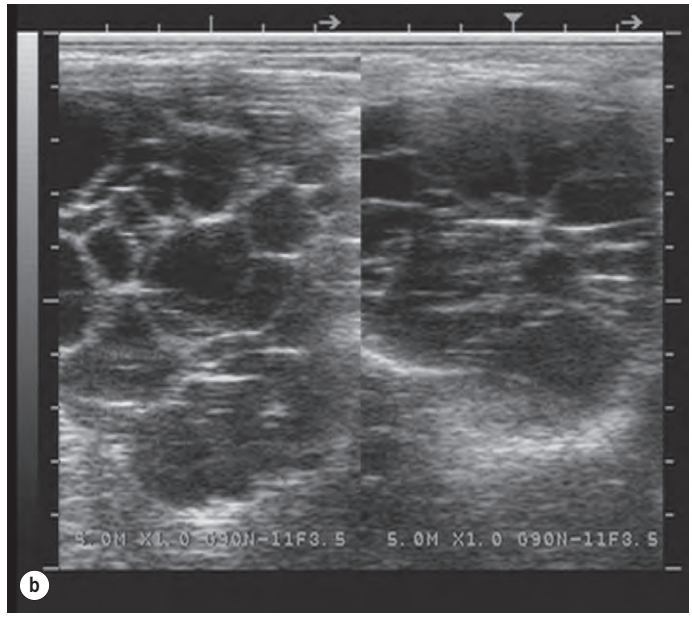


Figure 14.10 (a) Cross-section of granulosa-cell tumour showing numerous irregular cystic spaces. (b) Ultrasound appearance of bilateral granulosa cell tumours – polycystic appearance.

Endometrial biopsy

Use Bouin's fixative. Endometrial biopsy is used as a prognostic indicator to evaluate ability of a mare to carry a foal to term. Abnormalities seen include:

- Endometritis (Figure 14.9a).
- Periglandular fibrosis (Figure 14.9b).
- Cystic glandular distension (Figure 14.9b).

The following diagnostic aids are not carried out routinely during a breeding soundness examination.

Endoscopy

- Endometrial cysts.
- Transluminal adhesions.

Hormone analysis

- Progesterone – test of ovarian function.
- Inhibin and testosterone – increased concentration of each may indicate the presence of a granulosa-cell tumour.

Diseases of the mare's reproductive tract

A. Enlarged ovaries

Ovarian neoplasm

Ovarian tumours are not uncommon in mares. They may cause abdominal pain and discomfort to the mare if they reach a sufficient size.

Granulosa-cell tumour: most common ovarian tumour. It originates from the sex cord stromal tissue, is usually multilocular, benign, unilateral and often secretes hormones (Figure 10a).

Clinical signs

One ovary is enlarged and has no ovulation fossa. The contralateral ovary is usually small and firm and has no follicular activity because of:

- Suppression of gonadotropins by negative feedback from ovarian steroids and persistent elevated concentrations of inhibin produced by the tumour.

Behaviour varies depending on the predominant hormone secreted:

- Male-type behaviour – high testosterone.
- Nymphomania or anoestrus – not closely associated with circulating hormones.

Diagnosis

Palpation and ultrasonography of an enlarged ovary with a small inactive contralateral ovary. Ultrasonographic findings may range from a uniformly dense structure to a structure containing one or many large cysts (Figure 10b).

- Obliteration of the ovarian fossa.
- Behavioural changes.
- Elevated concentrations of serum inhibin (diagnostic value).
- Histopathology of removed ovary.

Treatment

Treatment involves removal of the affected ovary (see Chapter 8). It may be 3–18 months before the other ovary becomes functional.

Teratoma: benign, unilateral originating from germ cells. May contain bone, cartilage, teeth and hair. Does not interfere with cyclicity and pregnancy.

Serous cystadenoma: benign, primary epithelial tumour arising from surface epithelium of the ovulation fossa. Appears to have minimal effect on the function of the contralateral ovary. Affected ovary contains palpable large cysts.

Dysgerminoma: rare, malignant tumour of germ-cell origin. Rapidly metastasizes. Affected mares may or may not have chronic weight loss and abdominal discomfort. Poor prognosis. Perform thoracic radiographs and abdominocentesis for assessment of metastases.

Other tumours: melanoma, haemangioma, lymphosarcoma.

Haematoma

A haematoma results from the follicular cavity overfilling with blood after ovulation or from an anovulatory follicle that becomes haemorrhagic. It can become quite large (>10 cm), but usually regresses within 2–3 cycles, occasionally leaving a calcified nodule. The mare continues to cycle normally. Occasionally a haematoma may expand to destroy part or all of the ovary (Figure 14.11). On rare occasions, ovarian haematomas fail to organize, get to unusually large sizes and become filled with fibrinosuppurative material (Figure 14.12), leading invariably to adhesions and inflammation of surrounding tissues including the gastrointestinal tract. Unresolved ovarian haematomas that reach sizes greater than 15 cm in diameter should be removed surgically to avoid life-threatening complications.

Anovulatory follicles

These may be termed persistent or autumn follicles. They can be 10–15 cm in diameter and are commonly found in autumn associated with declining concentrations of LH. They are nonpathological structures containing blood, which can be gelatinous in consistency. The ultrasonographic appearance of the uterus is that of a uterus in diestrus rather than estrus.

B. Small ovaries

1. Seasonal anoestrus.
2. Hypoplasia – Turner's syndrome (63X0 genotype) or 64XY sex reversal.

Clinical signs

- Anoestrus.

Diagnosis

- Small firm ovaries.
- Lack of follicular activity.

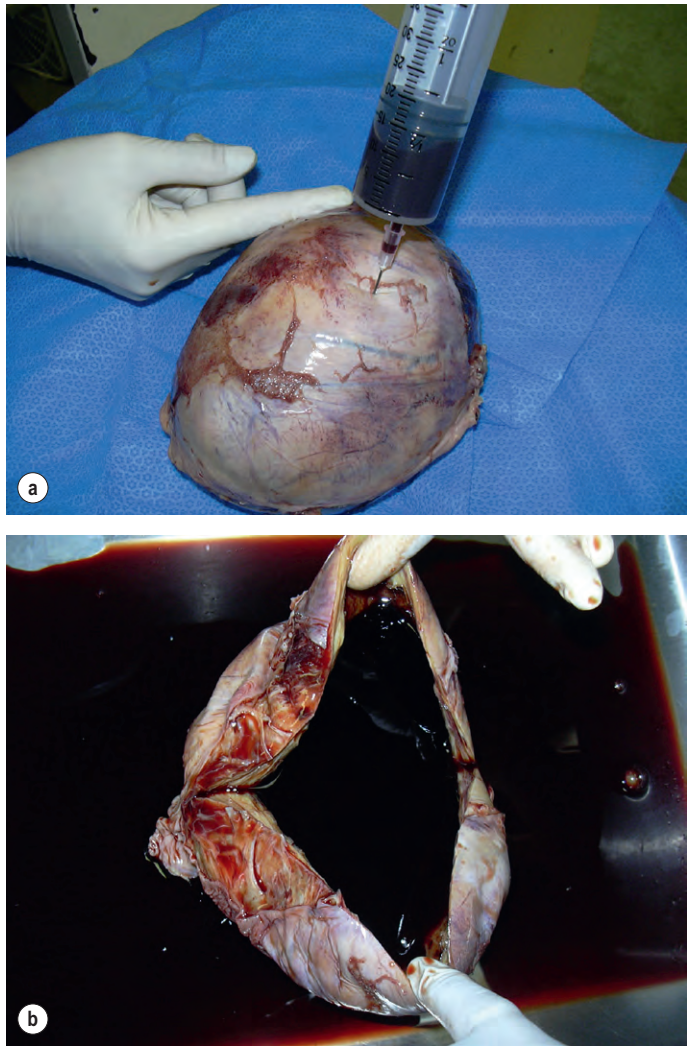


Figure 14.11 (a, b) Ovarian haematoma. All normal ovarian tissue has been destroyed.

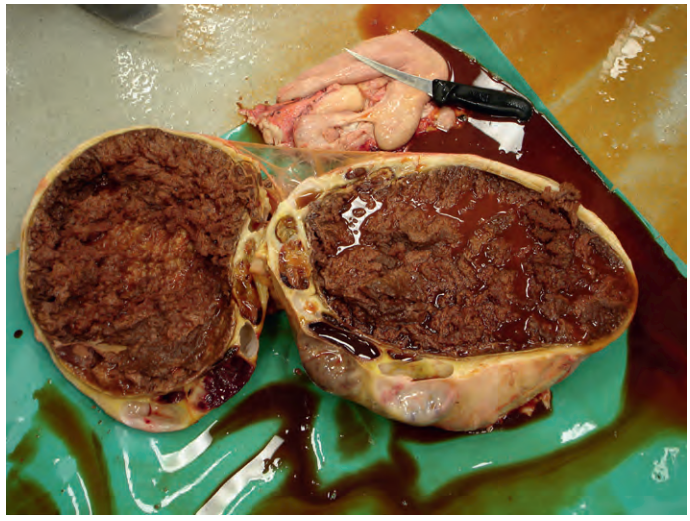


Figure 14.12 Ovarian haematoma that has turned into an abscess and is filled with fibrinosuppurative material.

- Uterus and cervix lack tone – infantile.
- Karyotype analysis.

3. Ovarian atrophy. This may be caused by severe malnutrition, parasitism, chronic disease, anabolic steroids.

C. Uterine tubes

Abnormalities are rare. Hydrosalpinx has been reported to occur secondary to segmental aplasia and to external pressure from adhesions obstructing the lumen.

Cysts in the region of the ovarian fossa are relatively common. There are three types based on their origin.

1. Mesonephric origin – cysts located adjacent to the ovary. Usually called paraovarian cysts.
2. Paramesonephric origin – cysts in the region of the ovarian fossa, but are not of ovarian origin.
3. Epithelial inclusion cysts – often called fossa cysts. Originate in the ovarian fossa. Surface epithelium is disrupted at ovulation and becomes pinched off and embedded in the ovarian cortex (Figure 14.13). The cysts can become numerous in old mares and can eventually obstruct ovulation. In severe cases, most of the ovary can be destroyed by pressure necrosis.

D. Uterus

Endometritis

Persistent mating-induced endometritis is probably the commonest cause of subfertility in mares.

Aetiology

Normal, resistant mares are able to clear bacteria from the uterus within 72 hours. Susceptible mares appear to have defects in uterine contractility and/or uterine immune defence mechanisms and remain infected after bacteria are introduced into the uterus. The three physical barriers to microorganisms gaining entrance to the uterus are the vulva, the vestibulovaginal seal and the cervix. If any of these structures are incompetent the mare is predisposed to developing persistent endometritis. Another aggravating factor in the pathogenesis of endometritis is delayed uterine clearance, which refers to the mare's failure to mechanically clear the uterus of fluid and contaminants associated with mating or artificial insemination.

Infection is introduced by:

- Pneumovagina.
- Parturition.
- Coitus.
- Veterinary gynaecological procedures.

Causative organisms are:

- Beta-haemolytic streptococci (most commonly isolated).
- *Escherichia coli*.
- *Klebsiella pneumoniae*.
- *Pseudomonas aeruginosa*.
- Yeasts.
- Fungi.

Clinical signs

- May be none.
- Discharge from uterus.
- Fluid in uterus.
- Failure to become pregnant.

Diagnosis

- Fluid in uterus (ultrasonography).
- Presence of neutrophils in endometrial cytology smear (>10%).
- Cloudy uterine lavage.
- Heavy growth of microorganisms (not diagnostic unless other signs are present).
- Neutrophils migrating through tissue in endometrial biopsy sample (Figure 14.9a).
- Hyperaemia of vagina or cervix.

Treatment

- Correct any predisposing conformational defects.
- Remove fluid from the uterus by lavage with sterile, isotonic saline solution. Can use oxytocin (10–20 IU IV) to assist in fluid removal.
- Infuse uterus with antibiotic (choice depends on microorganism cultured – use IV preparation). Dilute antibiotic into 30–60 mL for intrauterine infusion.
- Treat the mare daily during oestrus (short cycle with PGF2α if necessary).
- Use hygienic techniques during examination and breeding.

If the mare is still infected at the oestrus at which she is to be bred, the following treatment regimen can help to control infection.

- If the uterus contains a large amount of fluid on the day before breeding, flush with consecutive litres of buffered, isotonic saline solution (non-irritant) until the recovered fluid is clear. Lavage of the uterus with mucolytic agents (acetylcysteine) may be helpful to remove bacteria that form biofilms in the endometrium. Infuse broad-spectrum antibiotic or choose antibiotic based on antimicrobial sensitivity testing.
- Use artificial insemination if possible. Otherwise, infuse 100 mL of semen extender containing an appropriate antibiotic into the uterus immediately prior to covering.
- Flush the uterus with consecutive litres of buffered, isotonic saline solution not earlier than 4 hours after breeding until the recovered fluid is clear. Infuse antibiotic.
- Repeat flushing and infusion of antibiotic until one day after ovulation. Oxytocin can be administered to ensure 100% fluid recovery during these flushes.

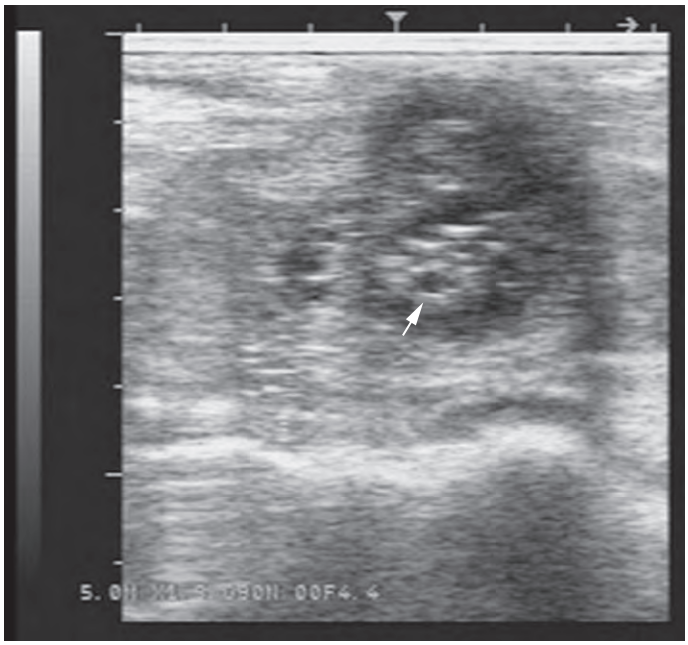


Figure 14.13 Ovarian epithelial inclusion cysts in a mare with a history of 4 years of infertility. Epithelial inclusions cyst may obliterate the ovarian fossa.

Acute toxic metritis

Aetiology

The entire uterine wall is inflamed. Metritis occurs after parturition and is often related to trauma. It can be secondary to retained placenta. Toxins can pass through the uterine wall to the general circulation.

Clinical signs

- May be systemically ill.
- Uterine discharge may be present.
- May develop secondary laminitis.

Treatment

- Systemically administered antibiotics.
- NSAIDs.
- Large volume uterine lavage (several litres of sterile, isotonic saline solution).
- Oxytocin to assist fluid recovery.

Endometrosis (formerly called chronic degenerative endometritis)

Chronic degenerative changes occur within the endometrium, increasing with age. There are three main pathological findings:

1. Periglandular fibrosis – fibrosis around the glands. Associated with reduced ability to carry a foal to term (Figure 14.9b).
2. Cystic glandular distension – dilated glands. Commonly contain inspissated secretion. Fibrosis may interfere with glandular secretion.
3. Endometrial cysts – may interfere with pregnancy if extensive. Caused by coalescence of lymphatic lacunae (enlarged lymphatics). If large, can surgically excise via endoscope using biopsy instrument, electrocautery or laser. Some workers have advocated the use of repeated flushes using hot (45–50°C), hypertonic saline solution to enhance uterine contraction.

Ventral uterine sacculations are commonly palpated at the junction of the uterine horns and body in old, infertile mares. Focal myometrial atony results in lymphatic lacunae in these mares.

Intrauterine adhesions

Aetiology

- Dystocia.
- Intrauterine infusion of caustic solutions.
- Endometritis.

Diagnosis

Diagnosis is by hysteroscopy. There may be single or multiple bands, partial or complete sheets or tunnels between adjacent endometrial folds. They result in infertility if extensive, cause pyometra or impede movement of embryo, which is necessary to signal maternal recognition.

Treatment

Break down manually or with endoscope and biopsy forceps. Laser or electrocautery is possible. They may recur.

Pyometra

Unlike the cow, pyometra in the mare is not always accompanied by a retained corpus luteum and anoestrus. Affected mares may cycle regularly or may have short cycles due to premature release of $PGF_{2\alpha}$. The uterus may accumulate 0.5 to 60 L of exudate. Mares with pyometra frequently have a fibrosed or occluded cervix. Usually there are no systemic signs.

Treatment

Treatment is often unsuccessful.

- Break down any adhesions.
- Lavage uterus and infuse appropriate antibiotics daily for 4–7 days or longer if necessary.

Mucometra

Mucometra is rare. It can be due to a non-patent cervix.

Treatment

Repair cervix.

Uterine abscess

This is rare. It can be secondary to dystocia, AI, severe metritis or uterine therapy. In the acute phase, there may be systemic signs – fever, neutrophilia, peritonitis. If small, can attempt surgical drainage into the uterus; otherwise, use abdominal approach. Poor prognosis for future breeding.

Uterine neoplasia

Leiomyomas or sarcomas are rare.

Clinical signs

Usually there are none.

Diagnosis

Diagnosis is by palpation and ultrasonography per rectum. Classification of tumour is by histopathological exam after surgical removal.

Treatment

If small and pedunculated they can be removed using a snare. Otherwise perform hysterectomy if benign. Partial hysterectomy may preserve fertility.

Contagious equine metritis (CEM)

Aetiology

This disorder is caused by *Taylorella equigenitalis*, a microaerophilic, Gram-negative coccobacillus. Transmission is venereal. The organism persists in clitoral sinuses (under anaerobic conditions) of carriers. Stallions are inapparent carriers. In some countries CEM is a reportable disease.

Clinical signs

Signs are severe endometritis with profuse watery, mucopurulent discharge. Mares have short oestrous cycles. Chronic infection can develop when clinical signs are not obvious. A symptomless carrier state can also exist.

Diagnosis

Isolation of organism on chocolate agar in atmosphere of 5–10% CO_2.

Treatment

Many mares recover spontaneously. If treatment is needed, use intrauterine infusions of penicillin and topical treatment of clitoris with dilute chlorhexidine.

E. Cervix

Cervicitis

Aetiology

Cervicitis is usually associated with vaginitis and/or endometritis. Apart from infection, it can be associated with irritants, such as air or urine in the vagina, and uterine medications.

Diagnosis

Diagnosis is by direct visualization of the caudal aspect of the cervix using speculum examination.

Treatment

Identify and treat the endometritis or vaginitis.

Cervical laceration

Aetiology

- Trauma at parturition.
- If severe, can prevent closure of the cervix resulting in uterine contamination and endometritis.
- If detected at foaling, should wait 30 days before attempting repair.

Diagnosis

Diagnosis is by digital examination of the cervix while it is under the influence of progesterone (dioestrus).

Treatment

Treatment involves surgical correction by a 2- or 3-layer closure after evaluating the endometrium by endometrial biopsy. Mares are given a sedative/analgesic combination of romifidine or xylazine plus butorphanol or epidural anaesthesia plus sedation. Retraction of the cervix may be facilitated by infiltration of local anaesthetic solution dorsal to the cervix. An incision is made along the scar of the healed edges of the tear and extended 1 cm cranial to the end of the tear, to ensure separation of mucous membrane from the fibromuscular layer. The wound can be sutured in three layers (internal mucous membrane, fibromuscular, and external mucous membrane) using a continuous horizontal mattress for the outer and inner layers and a continuous suture for the fibromuscular layer, or two layers (internal mucous membrane + inner fibromuscular layer, outer fibromuscular layer + external mucous membrane) in a continuous suture pattern using absorbable suture. The lumen of the cervix should be checked for patency throughout the procedure. Give a course of antibiotics plus NSAIDs if necessary. Wait 30 days before breeding. Advise the owner that the cervix may tear again at the next parturition.

Cervical adhesions

Aetiology

Result of trauma at parturition or uterine therapy with irritating agent. Can also be a sequel to repair cervical laceration.

Diagnosis

Diagnosis is by digital and speculum examination.

- Can be transluminal or attach the cervix to the vaginal wall.
- Interfere with cervical function.

Treatment

Treatment can be very difficult. The adhesions can be manually broken down and steroid cream applied, but they tend to reform rapidly.

F. Vagina

Pneumovagina (windsucking)

This is an important and relatively common condition.

Aetiology

- Poor vulvar and perineal conformation, especially the presence of an incompetent vestibulovaginal seal (Figure 14.4) (2nd physical barrier to potential contaminants to the uterus).
- Poor body condition predisposes.
- Vulvar vertical slope >20°.
- <2/3 of the vulvar lips below the level of the tuber ischii.

Diagnosis

- Aspiration of air when vulvar lips are parted.
- Commonly associated with endometritis.
- Severe cases may have pneumo-uterus and urovagina.

Treatment

Treatment is Caslick's vulvoplasty. Treat endometritis first. Infiltrate vulvar margins with local anaesthetic solution from just below level of the ischium to the dorsal commissure. Remove a 1-cm strip of mucosa from the mucocutaneous margin (N.B. do not remove skin). Use continuous or interrupted sutures to appose denuded tissue surfaces. Remove sutures after 10–12 days. Do not close vulva excessively – predisposes to urovagina. A 'breeder's stitch' of umbilical tape can be placed at the ventral edge of the repair to protect the suture line during coitus until the mare is confirmed pregnant. Alternatively, surgical staples may be also used to provide temporary vulvar closure until the mare is examined for pregnancy, especially for maiden mares. Open vulvoplasty prior to foaling. The vulvoplasty should be closed as soon as possible after foaling unless there is significant oedema or uterine lavage is necessary.

Some mares with a sunken anus and forward sloping of the vagina have both pneumovagina and urovagina. Horizontal transection of the perineal body has been used successfully in these cases.

Urovagina

Urine accumulates in the vagina (urine pooling, vesicovaginal reflux) causing inflammation. The condition is associated with poor conception and early embryonic death.

Aetiology

- Poor perineal conformation.
- Poor body condition.
- Relaxed ligaments – may occur only at oestrus or postpartum.

Pneumovagina and urovagina often occur concurrently.

Diagnosis

- Speculum examination reveals urine in vagina.
- Associated with endometritis.

Fluid in the vagina can be positively identified as urine by observing calcium carbonate crystals by microscopic examination, or determining the creatinine and urea concentrations.

Treatment

Treatment depends on the cause. Poor body condition should be corrected. Urogvagina may correct itself if it occurs after foaling. In mild cases, mares can conceive if urine is physically removed from the vagina before breeding. Intrauterine infusion of semen extender protects sperm from the deleterious effects of urine. If there is injury to the vestibulovaginal seal or if the vagina has a severe cranioventral slope, surgery is required. Urethral extension appears to give the most reliable results. The mare is sedated, and epidural anaesthesia is induced. After appropriate cleansing of the region, the vulvovaginal fold (urethral fold or transverse fold) is retracted with forceps. The dorsal mucosa and submucosa of the vulvovaginal fold are incised horizontally along the dorsal edge 2–4 cm cranial to its caudal border. The incisions are continued onto the middle of the vestibular wall and extended caudally to the labia. The cut edges are freed from the deep submucosa by dissection to create mucosal/submucosal flaps. The

opposing edges of the flaps are joined in a Y-shape by an inverting suture line to create a tunnel from the urethral orifice to the caudal vestibule. A sterile catheter can be placed in the urethra prior to performing the procedure to prevent damage to the urethral orifice and to provide a template. Antibiotics and NSAIDs should be given post-surgery, and the reproductive tract should not be examined for at least 2 weeks.

Persistent hymen

This is more common in mares than in other domestic species.

Aetiology

The hymen may be imperforate or may be present because the caudal section of the paramesonephric duct failed to fuse with the urogenital sinus.

Clinical signs

Failure of intromission.

Diagnosis

Manual and speculum examination.

Treatment

Treatment is by surgical excision after sedation of the mare. It is usually adequate to nick the membrane with a guarded blade and then extend the incision with the operator's fingers. Secretions may accumulate cranial to the hymen. These secretions may infrequently become infected and result in infertility.

G. Vulva

Neoplasms

- Melanoma – common in old grey mares. Usually not treated.
- Squamous cell carcinoma.
- Leiomyoma.

Perineal lacerations

See Chapter 8.

Enlarged clitoris

Aetiology

Intersex. Most common form is the male pseudohermaphrodite, which has testes and a 64XX karyotype. Often they are also SRY negative (Figure 14.14).

Coital vesicular exanthema

Aetiology

Venereal transmission of equine herpesvirus 3.

Clinical signs

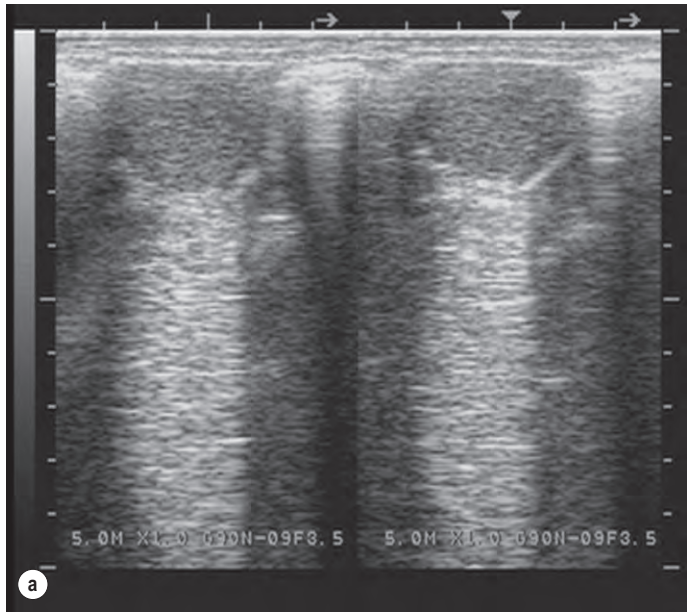
Papules, vesicles, scabby erosions on the vulva (penis and prepuce of stallion).

Diagnosis

- Examination.
- Intranuclear inclusion bodies on cytology.

Treatment

- Sexual rest for 60 days.
- Treat lesions with antiseptic cream to prevent secondary bacterial infection.



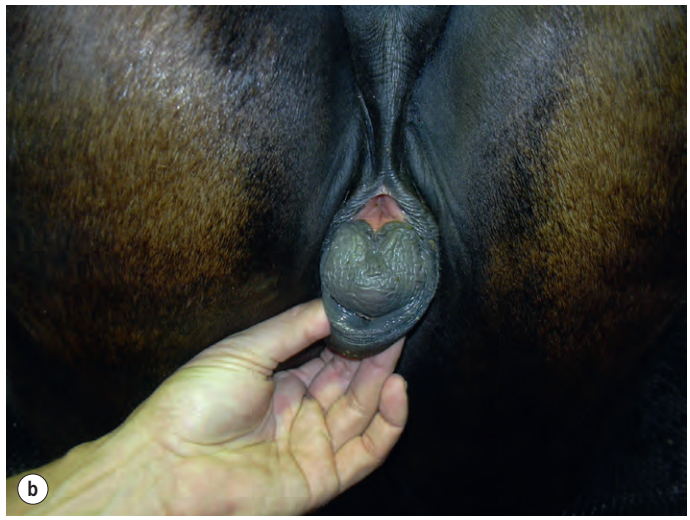


Figure 14.14 XX SRY-negative intersex mare. (a) Split-screen ultrasound images from abdominal gonads obtained by transrectal ultrasonography using a 5-MHz linear transducer. (b) Enlarged clitoris (penis-like).

14.2 THE PREGNANT MARE

Ovulation

- Occurs through the ovarian fossa.
- Signs of behavioural oestrus cease 1–2 days following ovulation.
- Oocyte is in metaphase II at ovulation (mature, ready to be fertilized).

Fertilization

- Occurs in the ampulla.
- The fertilized ovum enters the uterus as an early blastocyst approximately 5.5 to 6 days after ovulation.
- Unfertilized ova are retained in the uterine tubes but occasionally pass into the uterus.

Embryonic period

- The embryo is mobile in the uterus until day 16 after ovulation. This mobility of the embryo may be necessary for maternal recognition of pregnancy because the mobile embryo inhibits PGF2α release.
- The zona pellucida is shed at day 7, and the embryo is surrounded by a glycoprotein capsule. 'Hatching' from the zona pellucida, as seen in the cow, does not occur in the mare because the formed capsule retains the embryo's spherical shape. The capsule persists until the fourth week of pregnancy.

Placenta

1. Diffuse, non-deciduate.
2. Epitheliochorial.
3. Microcotyledonary. Attachment by microvilli does not occur until approximately 38 days. The macrovilli, which appear at day 45, are the forerunners of the microcotyledons. Maximum placental attachment does not occur until completion of the interdigitation of the placental microcotyledons at day 150.

The uterine glands secrete 'uterine milk' throughout pregnancy.

Direct apposition of the trophoblastic membrane with the endometrium is necessary before microcotyledons develop, and, therefore, they are absent at the following areas:

- Overlying the endometrial cups.
- At the cervical os (called the cervical star).
- Opposite the opening of each oviduct.
- Overlying the attachment of the yolk sac.
- Along invaginated folds of allantochorion.

Hippomanes

These are large, flat bodies composed of concentric rings of amorphous material which occur in the allantoic fluid. They originate from deposition of material from the allantoic fluid onto a nucleus of epithelial cell debris. They have no apparent clinical significance.

Endometrial cups

These are discrete, raised areas arranged in a horse-shoe or circular fashion at the caudal portion of the gravid uterine horn. The cups arise from invasion of the uterine epithelium by trophoblastic cells of the chorionic girdle. They are present between approximately day 40 and 120 of pregnancy. After sloughing, the remnants may be enclosed by invaginations of allantochorion termed allantochorionic pouches.

The endometrial cups secrete equine chorionic gonadotrophins (eCG) formerly called pregnant mare serum gonadotrophin (PMSG). eCG has LH-like activity in the horse and may support formation of secondary corpora lutea. Endometrial cups are also thought to induce maternal immunotolerance towards the antigenically foreign fetus.

Changes in the reproductive tract during pregnancy

Ovaries

- Numerous follicles >10 mm are present on the ovaries between days 10 and 100.
- The primary CL persists until about day 120–150.
- Secondary CL form after day 40 from ovulated follicles and accessory CL from luteinized, unruptured follicles. These supplementary CL also persist until around day 120–150.
- After day 100 of pregnancy, the ovaries are not necessary as a source of progesterone. Progesterone secretion by the ovaries return to basal levels by 180 days of gestation.

Cervix and uterus

- After ovulation, the cervix closes, and uterine tone increases.
- By 14–17 days, there is a marked increase in tubularity of the uterus.
- By 17–21 days, the cervix tightens and lengthens giving a characteristic pencil-like shape. The endometrial folds are no longer palpable.
- At 23–28 days, the embryonic vesicle may be palpated at the base of the pregnant horn.
- At 40 days, one half of the gravid horn is filled by the conceptus.
- At 60 days, the gravid horn is filled by the conceptus.
- At 80 days, both horns are filled by the conceptus.
- After 85 days, a decrease in tension of the allantois allows direct palpation of the fetus.

Pregnancy diagnosis

- Palpation per rectum: Accurate after about 28 days and optimal around day 42.
- Transrectal ultrasonography: Day 10 to term.
  Day 9–12: Blastocoele converts to yolk sac, which is detectable as a round structure.
  Day 18: Changes from round to triangular appearance – due to hypertrophy of dorsal uterine wall.
  Day 21: Embryo present ventrally.
  Day 23–25: Heart beat detectable. Allantois visible.
  Day 30: Allantois occupies ventral half of vesicle.
  Day 40: Allantois occupies most of vesicle; yolk sac has regressed.
  Day 50: The umbilical cord has lengthened, and the foetus has descended to the floor of the allantoic sac.
- Transabdominal ultrasonography: Day 80 to term.
- Oestrone sulphate: Produced by precursors from foetal gonads and, therefore, confirms viability of foetus. Measured in high concentrations in serum from day 90.
- eCG: Day 40–120. Problem with false positives. Indicates presence of endometrial cups, not foetus.

Endocrinology of pregnancy

1. Progesterone:
   Secreted by corpora lutea.
   Reaches first peak around day 20, then declines until day 40–50.
   Between day 40 and 90, secondary CL secrete progesterone resulting in increased concentration.
   Progesterone concentration decreases to low concentrations by day 150 and to basal concentrations by day 180.
   The placenta secretes progestagens (progesterone and progesterone metabolites) from day 70 to term by de novo synthesis. Takes over from CL as source of progestagens and maintenance of pregnancy.
2. Oestrogen:
   Increases between day 35 and 40 (? result of stimulation by eCG). Second rapid increase after day 80. Peaks at 7th–8th month gestation and then decreases gradually to term.
   Until day 70 maternal ovaries are the major source.
   After day 70, foetoplacental oestrogen production predominates.
   Precursor for oestrogen derives from foetal gonads; therefore both viable foetus + placenta required.
3. eCG:
   Secreted by endometrial cups.
   First detected day 35–40. Peaks at day 70 and disappears by day 150.
   Does not have the FSH-like activity seen in other species.

4. Relaxin:
   Secreted by the placenta.
   First detected around day 70 and peaks at 5–6 months.
   Increases again near parturition.

Complications during pregnancy

A. Infectious causes of abortion

Bacterial

Bacterial infections are responsible for approximately 30% of aborted foetuses seen at postmortem. Many species of bacteria have been incriminated. Most commonly associated are *Streptococcus* spp., *Escherichia coli*, *Pseudomonas* spp., *Klebsiella* spp. and *Staphylococcus* spp. *Leptospira* spp. are now also being recognized as causing abortion in mares, and *Ehrlichia* spp. are suspected as well. Higher bacteria, that is bacteria resembling fungi, can cause chronic proliferative placentitis and abortion.

Frequently secondary non-pathogenic bacteria are isolated, but if only one species is found in multiple organs, the stomach contents and placenta, it is likely that the organism is a primary or secondary cause of abortion. The bacteria may be considered a primary cause if inflammatory changes are seen in the foetus.

Most infections are caused by ascending infection through the cervix. This tends to occur early in pregnancy.

Fungal

Fungal infection usually occurs in second half of gestation. Abortion results from an ascending infection spreading from the cervical area. The chorioallantois is thickened and leathery, and a thick mucoid exudate is often present. The foetal organs are usually not decomposed. The liver is often large, pale and mottled. *Aspergillus fumigatus* is the most frequently isolated fungus. Fungi are ubiquitous; therefore signs of an inflammatory response to the fungus should be demonstrated before the organism is considered significant. Even in these cases, fungi may only be opportunistic and secondary to another predisposing cause. The foetus is usually small and emaciated, but near-term foals generally have a good chance of surviving.

Viral – equine herpesvirus I (EHV-1)

See Chapter 19. EHV-1 is a respiratory virus which is widespread among the horse population but responsible for fewer than 15% of abortions seen at post-mortem. The adoption of vaccination strategies has helped to reduce the incidence of viral abortion. Mares usually abort suddenly during the last four months of gestation. It usually only affects one or two in a group but occasionally causes multiple abortions.

Foetuses are fresh with petechiation of mucosae. Excessive straw-coloured fluid is present in thoracic and abdominal cavities.

Diagnosis

- Histology – intranuclear inclusion bodies in liver and adrenals. Focal necrosis in liver, adrenals, lungs, thymus and spleen.
- Virus isolation.
- Fluorescent antibody test and immuno-histology.
- Serology (may not detect an increase in antibody titre).

Prevention

- Isolate pregnant mares after the third month of gestation.
- Vaccination every 2 or 3 months during pregnancy, but protection is not complete.

Viral – equine viral arteritis (EVA)

See Chapter 19. EVA is transmitted by the respiratory route and venereally by carrier stallions. Incubation period is 3–14 days. The virus localizes in the accessory sex glands of stallions and about 34% of stallions recovered from EVA shed virus in their semen for long periods. Shedder stallions are always seropositive but not all seropositive stallions are shedders. Recovered mares do not shed the virus longer than 3 weeks. Seropositive mares with a stable or declining antibody titre are thought not to infect stallions or in-contact mares. Clinical signs vary from mild fever and conjunctivitis to severe illness comprising fever, depression, filling of the distal limbs, ventral oedema, urticarial rashes and oedematous plaques, conjunctivitis with periorbital oedema and nasal discharge. Up to 80% of non-immune mares may abort especially in late gestation. Foetuses are decomposed.

Diagnosis

- Virus detection in nasopharyngeal swabs, heparinized blood, semen, serum and possibly urine.
- Serology – rising titre in blood samples collected 2 weeks apart.

Prevention

- Test mating. Identify and isolate infected horses.
- Vaccination. Vaccinated horses become seropositive, and this can affect eligibility for international movement. Horses should be serologically tested prior to vaccination to demonstrate that the horse has not previously been infected.

Because EVA virus can be secreted by mares for up to 3 weeks post-infection, newly arrived horses should be quarantined for at least 3 weeks. Identification of shedder stallions is very important in control of the disease.

Viral – equine infectious anaemia (EIA)

See Chapter 19. EIA is a systemic disease caused by a retrovirus. Signs vary from peracute to chronic, with infection in some horses being inapparent. Infected horses remain persistent carriers. The disease is usually transmitted by blood-feeding insects. On first exposure to the virus acute signs may develop including fever, anorexia and petechiation on mucosae. This phase is transient and is followed by recurrent episodes of fever, anaemia, jaundice and weight-loss. It only rarely causes abortions.

Diagnosis

- Virus isolation.
- Coggin's test.

Prevention

No vaccine is available; therefore, identify and control movement of infected horses.

B. Non-infectious causes of abortion

Twinning

Twinning is common in all breeds of horses, especially Thoroughbreds. It accounts for approximately 20% of all aborted foetuses examined at post-mortem. There is a high rate of natural reduction before day 40. After day 45, 65% of twins are aborted. Abortions are due to placental insufficiency. They most commonly occur between mid-pregnancy and term. The foetuses often appear to have died at different times. When only one twin is found, diagnosis can still be made by demonstration of a large avillous area of chorion – the contact area between the chorion of the two twins.

Umbilical cord abnormalities

In Thoroughbred mares cord length should be less than 84 cm

1. Too long:
   Can twist and compromise vascular supply to foetus. Must demonstrate evidence of ischaemic necrosis before diagnosing abortion due to a twisted cord.
   Can become wrapped round a foetal appendage.
2. Too short:
   May break prematurely at birth leading to perinatal asphyxia.
   N.B. Many normal foals are born with abnormal umbilical cords.

Placental insufficiency

- Aborted foetuses have retarded growth due to insufficient nutrients from the placenta.
- Associated with endometrial fibrosis or atrophy in old mares.
- Can also be due to infectious placentitis causing atrophy of the chorionic villi.

Uterine body pregnancy

- Uncommon.
- Chorioallantois fails to expand, and the foetus develops in the uterine body.
- Foetal growth is retarded, and abortion usually occurs after the 7th month of pregnancy.

Luteal insufficiency

In some mares with repeated unexplained abortions in the first third of gestation, supplementation with altrenogest until day 100 has resulted in the mare carrying a foal to term. Luteal insufficiency is probably an extremely rare occurrence, and there are very few documented cases.

C. Uterine torsion

- Occurs between the 5th and 11th month.
- Approximately 50% occur at foaling.
- Mare presents with colic – usually only moderate pain.
- Does not usually involve cervix (unlike cows).
- Torsions can be clockwise or counterclockwise.

Diagnosis

- Signs of colic unresponsive to treatment. If intestine is entrapped by the torsion, intestinal dysfunction (nasogastric reflux, intestinal distension etc.) may be present.
- Palpation of broad ligaments per rectum serve to diagnose the condition and indicate direction of torsion.

Treatment

- Check for evidence of foetal death, uterine necrosis or rupture.
- If cervix is open – manipulate foetus and untwist uterus via cervix.
- If cervix is closed – roll anaesthetized mare (variable results).
- Surgical correction – high flank incision in standing mare (on side towards direction of torsion). If torsion occurs at term, surgical correction and caesarean section can be performed through a midline celiotomy.

D. Ventral ruptures: rupture of prepubic tendon and the abdominal muscles

Late gestation

- Most common draught breeds.
- In mild cases may just present as oedematous swelling.
- Abdominal pain.

Treatment

- Supportive care until term – abdominal sling; analgesics.
- Induce delivery if intractable pain. Surgical repair only possible in a minority of cases.
- Do not rebreed.

E. Hydrops

- Usually hydrops allantois.
- Excessive accumulation of allantoic fluid at 8–11 months.
- Rapid onset of abdominal distension.
- Foetus cannot be palpated.
- Risk of ventral rupture (prepubic tendon; body wall).

Treatment

- IV fluid therapy while slowly draining the uterus.
- Treat shock.
- Induce parturition in the mare and assist foetal extraction. Many foals are born alive but are malformed or soon die. Surviving mares can have normal fertility.

F. Vulvar bleeding

Occasionally small veins of the vestibule or vagina become distended and may bleed (Figure 14. 8). Vulvar bleeding is only rarely associated with placental detachment and impending abortion.

Diagnosis

Diagnosis is by speculum examination.

Treatment

Pack the vestibule, ligate vessels or cauterize vessels with electrocautery, but treatment is usually not required – varicosities often shrink when pregnancy ends.

G. Prolonged gestation

Approximately 1% of pregnancies exceed 370 days. The majority of these mares have a spontaneous parturition and produce a foal of normal size (occasionally small).

Causes

- Embryonic diapause between 30 and 60 days has been suggested as a possible cause (although not unequivocally documented).
- Foetal gonadal and/or placental dysfunction.
- Foetal mummification.

Treatment

Do not induce parturition if the mare and foetus appear healthy (as assessed by transabdominal ultrasound).

Note: Induction of parturition is only safe if concentrations of milk electrolytes (calcium, sodium and potassium) indicate foetal readiness for parturition.

H. Premature udder development

- Commonly seen before abortion of twins.
- Secondary to placentitis.
- Perform a complete physical examination of dam.
- Check foetal heartbeat with ultrasound.
- Depending on cause, therapy may include progesterone, NSAIDs, antibiotics.

Elective termination of pregnancy

Day 5–35

Give PGF2α IM.

Day 35–120

If endometrial cups are present:

- $PGF_{2\alpha}$ IM once or twice/day until abortion (usually within 3–5 days).
- Uterine lavage 3 × 1 litre sterile, isotonic saline solution. If the foetus is not expelled within 24 hours, repeat the lavage. The mare will not come into oestrus until the endometrial cups have regressed.

Day 120–near term

Termination is not recommended because of the risk of cervical and uterine damage, retained placenta and dystocia.

Pharmacological induction of abortion after 120 days: infuse cervix with PGE (dinoprostone, alprostadil or misoprostol); abortion in 24–72 hours. May associate this treatment with daily oxytocin injections or uterine lavage with warm, sterile, isotonic saline solutions to facilitate the expulsion of the foetus and its membranes.

Uterine lavage: Dilate cervix. Rupture membranes and remove foetus. Large volume lavage helps to dislodge foetal membranes, especially if before or around 120–140 days of gestation.

14.3 PARTURITION

Gestation length: average 342 days (range 321–365 days).

Signs of impending parturition

1. Enlarged abdomen.
2. Mammary development.
   3 to 6 weeks prepartum. Major changes within 2 weeks of term.
   Initially straw-coloured discharge.
   Udder distends with colostrum 2–3 days prepartum.
   Teat waxing in last 48 hours.
   Increase in calcium and magnesium content of milk.
   At term, calcium >10 mmol/L; concentrations of potassium becomes greater than that of sodium in the milk ('cross-over').
3. Relaxation of sacrosciatic ligaments and vulva.
4. Relaxation of cervix (may occur 0 to 30 days prepartum)

Mares vary tremendously in the signs that they show, ranging from none to all.

N.B. Remember to open vulva at least 2 weeks before expected foaling date if Caslick sutures are present.

The foetus is highly mobile within the uterus during the first half of gestation. During the last trimester the foetus is in the dorso-pubic position with the head and forelimbs flexed.

Stages of parturition

Stage I:

- Lasts approximately 1 hour (mares have a high degree of maternal control over length of stage I).
- Uterine contractions start.
- Cervix relaxes and dilates.
- Restlessness.
- Signs of colic.
- Patchy sweating.

The end of 1st stage labour is marked by rupture of the allantochorion at an avascular area, the cervical star, and release of straw-coloured watery allantoic fluid. The foal rotates to a dorsosacral position just before birth. The amnion should protrude through the vulvar lips within 5 minutes of rupture of the allantochorion.

Stage II:

- Lasts less than 30 min.
- Mare assumes lateral recumbency.
- Abdominal contractions.
- Foal is delivered covered by amnion (more correctly termed the allantoamnion).
- The equine placenta separates from the endometrium rapidly, and foetuses not delivered within a short time of onset of 2nd stage labour are deprived of oxygen.
- Normal presentation is anterior, dorsosacral, extended.

N.B. If the red allantochorion appears at the vulvar lips, rupture the membrane and extract the foal immediately. Differentiate from prolapsed bladder!

Stage III:

- Expulsion of placental membranes usually occurs within 1 hour. The allantochorion is turned inside out.
- N.B. Always examine the foetal membranes for completeness.

The foal should be standing within an hour and should have sucked within 4 hours. As retained meconium is common, it may be advisable to give all foals an enema (saline solution, soapy water or lubricant) soon after birth.

Endocrinology of parturition

Oestrogens decrease during the last month of pregnancy. Progestogens increase during the last month of pregnancy. However, progestogen concentrations decrease during the 24-hour period before parturition; hence the ratio of oestradiol: progestogen increases prior to parturition.

$PGF_{2\alpha}$ and oxytocin increase dramatically at parturition. The sudden enhanced secretion of oxytocin may trigger second stage labour.

Induction of parturition

Because of the great variability in gestation length in the mare, it is impossible to arbitrarily set a gestation length at which induction can be performed. The mare must be at least 321 days pregnant (preferably >330 days), have an enlarged udder, teats distended with colostrum, and relaxed sacrosciatic ligaments. Ideally the mare's cervix should be softening, but this is not essential. Concentrations of electrolytes in the milk have been used to assess foetal maturity. Ca^{2+} concentrations ≥40 mg/dL, Na^{+} concentrations ≤30 mEq/L and K^{+} concentrations ≥35 mEq/L generally indicate foetal readiness for birth. Commercial mare-side kits are available for measuring Ca^{2+} concentrations in mammary secretions. Do not induce if the mare shows signs of impending abortion or if foal viability is questionable. Examine the mare per rectum to check foal presentation as well as viability of the foetus. Place tail wrap on mare and thoroughly wash perineum. Then perform vaginal examination to evaluate cervical dilation.

- Oxytocin: Most widely used drug. Only induce if readiness for birth is confirmed by milk electrolytes. Can be administered IM or IV. Dosage varies with degree of cervical relaxation. If relaxed to 2 cm can give a 10–20 IU bolus IV. Delivery of foetus within 30–45 min; rarely up to 60 min. If cervix closed, give 2.5–10 IU every 20 min IV until labour commences, typically within 60 min. If cervix relaxes after 4–5 boluses but labour has not started, give 20 IU IV. May also give as IV infusion 60 IU oxytocin in 1 L

isotonic saline solution over 1 hour. Larger doses of oxytocin have been associated with premature placental separation and foetal hypoxia.
- $PGF_{2\alpha}$ and dexamethasone are not recommended to induce parturition in the mare.

14.4 DYSTOCIA

The incidence of dystocia in horses is low (approximately 4%). However, it represents a life-threatening situation to the foal, and the future reproductive performance of the mare is often reduced. Dystocia exists when 1st or 2nd stage labour is prolonged or not progressive. Examination of the birth canal and foetus is necessary to determine the cause of dystocia.

Examination of the mare:
- Adequate restraint. Epidural anaesthesia may be administered to prevent straining.
- Hygiene – bandage tail, scrub perineal region with povidone-iodine soap.
- Lubrication. Assess birth canal for size, evidence of previous trauma, and degree of relaxation. Check presentation of foetus and determine viability.

Obstetric procedures

Mutation
- Repulsion – pushing the foetus back into the uterus where space is available to correct position or posture. It is always necessary to repel before attempting other mutations.
- Rotation – turning the foetus on its long axis to bring it into a dorsosacral position.
- Version – turning the foetus on its transverse axis to convert a transverse presentation into a longitudinal presentation.

To adjust extremities, it is necessary to repel the proximal portion, bringing the middle portion of the extremity laterally and the distal portion medially. If mutation cannot be accomplished successfully in less than 15 minutes, an alternative form of delivery should be used.

Forced extraction

This may be attempted after foetal malposture has been corrected. Traction snares or chains are placed around the pasterns. Traction devices should be used only to position the head, not to exert traction on the head (the bones of the skull are not as well developed as in the calf).
- Limit force to 2 or 3 people.
- Hip-lock is uncommon in the mare, and there is usually no need to rotate foetus.
- Mutation and forced extraction may be undertaken with the mare under general anaesthesia and with her hind limbs mechanically raised.

Foetotomy
- Complete foetotomy is required only rarely in the mare. In most cases, a partial foetotomy allows removal of impediment to delivery.
- Epidural anaesthesia and sedation are required to minimize straining.
- When more than one or two cuts are anticipated, general anaesthesia is indicated.
- Sequelae to foetotomy: retained placenta, metritis, septic peritonitis, laminitis, vaginal and cervical lacerations and delayed uterine involution. The severity of sequelae is proportional to the number of cuts required and the duration of labour prior to presentation.

Caesarean section

Indications: transverse presentation, uterine torsion, malposture of living foetus that cannot be corrected rapidly, oversized foetus (rare because mare controls foetal size, not stallion) and maternal pelvic deformities.

The usual approach is by midline celiotomy. Until the foetus is removed, general anaesthesia should be maintained in a light plane to avoid foetal respiratory depression. The uterus is incised along its greater curvature. Uterine contents should be treated as contaminated and carefully drained to the outside. The use of a continuous interlocking suture through all the layers of the uterine wall and around the entire margin of the incision controls intraluminal haemorrhage – a significant cause of maternal mortality. The uterus is closed with inverting sutures taking care to exclude any placenta. After surgery, administer 5–10 IU oxytocin IV to aid involution and assist membrane expulsion. A low dose of NSAIDs can be given to reduce pain caused by uterine contractions.

Postoperative complications may include metritis, septic peritonitis, laminitis and retained placenta. Check for early adhesions by palpation per rectum after 4 days – break down manually.

14.5 THE POSTPARTUM PERIOD

1. Uterine involution:
   Occurs by 6–10 days postpartum.
2. Foal heat.
   First oestrus occurs 7–9 days after foaling.
   Individual fertility varies tremendously during this oestrus.

Postpartum complications

Retained foetal membranes (RFM)
- Treatment should be initiated if the placenta is retained more than 3 hours (2–10% of mares). Mares are likely to be less fertile at foal heat if not treated.
- Manual removal of RFM should be avoided or undertaken with extreme care.
- Use 5–10 IU oxytocin IV or IM every 30–60 min or 60 IU in 1 L of sterile, isotonic saline solution infused intravenously over 1 hour.
- Repeat as necessary (usually expelled within 30 min).
- If retention is prolonged (>8 hours) the mare can develop endotoxaemia, laminitis and endometritis. Use more aggressive therapy:
  NSAIDs.
  Broad-spectrum antibiotics.
  Exercise.

Alternatively, the allantochorionic space may be infused with 10–12 L of dilute (0.1%) povidone-iodine solution or sterile, isotonic saline solution. The opening in the foetal membranes is tied shut. The distension of the reproductive tract stimulates uterine contractions (via oxytocin release), and the membranes are usually passed within 30 minutes.

Haemorrhage
- May involve uterine, utero-ovarian or external iliac arteries. Can also have severe intrauterine haemorrhage.

- Mares may exsanguinate. Survival depends on whether haemorrhage is contained within the broad ligament or whether the ligament ruptures.
- Signs of colic with weak, rapid pulse and pale membranes.
- Keep mare in dark, quiet stable.
- Sedatives may help.
- Do not give oxytocin in case clot is disrupted.

Rectovaginal fistula/perineal laceration

- Caused by damage to birth canal by foal's hoof.
- Repair surgically after 30 days when damaged tissue has sloughed and swelling has resolved.

Uterine rupture

- Usually uterine body, but can be tip of the gravid horn.
- May pass blood clots vaginally.
- Diagnosed by manual examination per vagina and peritoneal fluid cytology.
- Mare may be depressed and ill due to blood and foetal fluids in abdominal cavity. Uterus has tense, ridged feel.
- Give oxytocin (10–20 IU) hourly to contract uterus.
- Antibiotics/NSAIDs.
- Surgical repair of uterus may be indicated (esp. large ventral tears).

Endometrial haemorrhage

- Uncommon.
- Usually not serious but may persist for 3–4 days.

Treatment

- Restrict exercise.
- Oxytocin to reduce size of uterus.
- Pack uterus if necessary.
- Antibiotics.
- ? Surgical repair but difficult to adequately expose uterus.
- Lavage uterus after 1 week to break down any adhesions.

Uterine prolapse

- Rare in mare. More commonly, one horn invaginates into the uterine lumen causing pain and straining.
- Usually follows placental retention, dystocia or indiscriminate use of ecbolics.
- May be accompanied by shock.

Treatment

- Tranquillizer (do not administer if mare is in shock) or sedative.
- Epidural or general anaesthesia.
- Clean and replace uterus; infuse the uterus with warm isotonic saline solution to ensure the tips of the uterine horns are not invaginated.
- Systemic administration of antibiotics + low doses of oxytocin (5 IU; IM).
- Exercise.
- If handled quickly and effectively future fertility is good.
- If only part of the horn is invaginated infuse 4–8 L warm saline solution and drain.

Toxic metritis

Some degree of endometritis is normal after foaling. Only treat if the uterus fails to involute or the mare is systemically ill.

Treatment

- Lavage uterus with 3–5 L of sterile saline solution. Recover as much fluid as possible. Repeat as necessary.
- Can give oxytocin to help drain uterus.
- Infuse uterus with appropriate antibiotic.
- Exercise.
- Treatments for endotoxaemia. Laminitis is a possible complication.

Cervical tear

- Occurs not only after dystocia but after apparently normal foaling.
- Diagnosed by manual vaginal examination during dioestrus.

Treatment

Surgical repair.

14.6 THE STALLION

Examination of the stallion for breeding soundness (BSE)

Reasons for performing a BSE:

- Prepurchase.
- Before the start of the breeding season.
- Fertility problems.

The components of the BSE are described below.

Identification

Ensure that a thorough description of the stallion is included in the BSE certificate.

History

- General – illness, use, medications.
- Reproductive:
  Number of mares bred/season.
  Number of pregnancies.
  Fertility of mares.
  Results of previous BSE.
  Performance of other stallions on farm.

General physical examination

- Musculoskeletal.
- Respiratory.
- Eyes.
- Mouth.
- Body condition.

Semen collection and evaluation

If the stallion is available for only one day, collect two ejaculates one hour apart. Otherwise it is more representative to collect daily ejaculates for 5–7 days. Using this method stores of sperm in the excurrent duct system will be depleted, and the last ejaculates will represent daily sperm output (DSO). This figure may be used to calculate the number of mares that a stallion can cover in a season. For semen collection, use an artificial vagina filled with hot water so that its internal temperature is 45–48 °C (113–118 °F). Use a plastic disposable liner. Lubricate with non-spermicidal water-soluble lubricant. Rinse the penis with warm water (42 °C, 107.6 °F) and dry. If excessive smegma is present use a water-soluble low-residue soap and rinse

thoroughly with warm water. After collection maintain semen at 34–37°C (93.2–98.6°F) and protect from light, temperature extremes, chemicals and water. Alternatively, the ejaculate can be diluted with semen extenders and kept at room temperature until all the analyses are completed.

The following features should be evaluated:

Motility. Motility should be evaluated first because it decreases with time. Because of the tendency for stallion sperm to clump, it is sometimes easier to check motility after adding extender to the semen.

- Total motility = percentage of sperm that are moving.
- Progressive motility = percentage of sperm that are moving forward in a relatively straight line.
- Express motility as total/progressive motility.

pH. pH should be evaluated soon after collection as the metabolic activity of the sperm increases the pH with time (normal = 7.2–7.6). High pH may be due to contamination with soap or urine, or infection in the urethra or proximal reproductive tract.

Volume and concentration. Both gel and gel-free volume. Average is 70 mL containing a total of 8×10^9 sperm (total sperm number = volume × concentration). Can measure concentration on a haemocytometer or a spectrophotometer (densimeter).

Morphology.

- Can be performed using phase-contrast microscopy on wet-mount preparation in buffered formol saline solution, or by staining with nigrosin eosin.
- Count 200 sperm. At least 60% should be normal.
- Abnormalities are classified according to specific type (normal sperm, abnormal head, detached head, proximal droplet, distal droplet, abnormal midpiece, abnormal tail). Morphological abnormalities have been divided into primary (arise in testis) and secondary (arise in the excurrent ducts or during storage or ejaculation) abnormalities. As the true origin of abnormalities is not known this classification system has been questioned. In bulls' sperm abnormalities have been categorized as major (abnormal sperm head, proximal droplet and abnormal mid-piece) or minor (detached head, distal droplet and abnormal tail) depending on their association with impaired fertility. However, in the stallion the association between specific abnormalities and reduced fertility is not clear. NB. Abaxial midpieces are considered normal in stallion sperm.

Number. Number of morphologically normal, progressively motile sperm = total number of sperm in ejaculate × % morphologically normal × % progressively motile.

There should be at least 1×10^9 in the second ejaculate. This figure is the most important in calculating the potential fertility of a stallion. The second ejaculate should contain approximately 50% of the number of sperm in the first ejaculate.

Longevity of motility. Aliquots of raw and extended semen are kept in airtight, dark conditions at 4°C (39.2°F) and at 23°C (73.4°F). Motility should be evaluated hourly for 6 hours then at 24 hours. At least 10% sperm should be progressively motile at 6 h in the raw semen sample and at 24 hours in the extended semen. Shortened sperm longevity and increased morphological defects may suggest accumulation of extragonadal sperm from infrequent ejaculation. Evaluation of sperm longevity at 4°C is necessary if the stallion's semen is to be chilled and used in an AI programme. However, the relevance of longevity at 23°C in a laboratory is questionable.

Morphology and motility of stallion semen are not affected by season, but volume of seminal fluid and total sperm numbers increase in spring and summer. Although photostimulation can hasten testicular and libidinal recrudescence early in the year, the period of time of heightened reproductive capacity is not prolonged.

Testicular size and consistency

This is highly correlated with the daily sperm output (DSO).

Length, width and height of each testis are measured using calipers or ultrasonography. Total scrotal width should be greater than 8 cm. However, whatever the size of the testes, stallions with flabby or hard testes are potentially poor producers of sperm. Estimates of testicular volume (TV) may improve accuracy in determining daily sperm output (DSO). DSO = mean daily yield of sperm after extragonadal reserves have been depleted. This figure enables calculation of the number of mares a stallion can breed in a season (may be limited by libido).

DSO = 0.024x − 0.76 where x = testicular volume (TV)

TV = $4/3\pi abc$ where a = testicular height/2, in which b = testicular width/2 and c = testicular length/2

Venereal pathogens

Rectal sleeves should be worn and changed between handling the external genitalia of different stallions. For farms or breeding centres that collect stallions for artificial insemination, it is recommended the use of a disposable plastic liner to cover the breeding phantom to avoid contamination of different stallions using the same facility.

1. Prior to rinsing the penis, swab the following structures:
   Fossa (Figure 14.15).
   Shaft.
   Sheath.
   This identifies the normal flora for the stallion (*Corynebacterium* spp., fungi and small numbers of *Streptococcus* spp.).
2. After rinsing and drying the penis, swab the urethra. This identifies the flora of the terminal urethra. Small numbers of mixed bacteria are normal. There should not be a heavy pure growth of any one bacterial species.
3. Immediately after collecting the first ejaculate, swab the urethra. This identifies the flora of the internal genital tract. There should be no growth from this swab.

Figure 14.15 Swabbing the urethral fossa. The stallion is being teased with a mare to facilitate swabbing of the penis.

A pure growth of *Pseudomonas aerogenes*, *Klebsiella pneumoniae* (capsule types 1, 2 and 5), *Streptococcus* spp. or *Taylorella equigenitalis* is considered abnormal. All are potential venereal pathogens. Not all strains are pathogenic, however. Apart from *Taylorella equigenitalis*, all can be present as incidental contaminants. If a stallion is blamed for infecting a mare with *Pseudomonas* or *Klebsiella*, check capsule or serotype of isolates from both the mare and stallion for confirmation.

Code of practice for CEM and other bacterial venereal diseases (in Great Britain)

All breeding TB stallions must be swabbed on two occasions more than 7 days apart from:

- Urethra.
- Urethral fossa.
- Penile sheath.
- Pre-ejaculatory fluid.

Put swabs in transport medium and keep at 4°C (39.2°F) during shipment. Should reach the laboratory within 24 hours of collection.

Stallions should be swabbed before the start of the breeding season. If the cultures are positive for a venereal pathogen the stallion is designated as unsuitable for breeding until he is treated and three sets of negative swabs are obtained at intervals of >7 days.

Treatment

Infections with *Klebsiella* and *Pseudomonas* are difficult to treat. Wash erect penis with povidone-iodine soap. Dry penis and pack with an aminoglycoside cream or 1% silver sulphadiazine daily for 1–2 weeks. Collect serial swabs over several weeks for cultural examination to monitor success of treatment. Alternatively for *Pseudomonas*, wash erect penis with dilute HCl (10 mL of 38% HCl in 1 gallon of water) daily for 2 weeks. Reduce concentration if skin reaction is noted. Treat after each breeding. For *Klebsiella*, use dilute sodium hypochlorite. Add 40 mL 5.25% sodium hypochlorite/gallon.

Taylorella is sensitive to most antibiotics and antiseptics. Wash erect penis daily for at least 5 days with chlorhexidine solution (>2%) followed by topical application of nitrofurazone or penicillin ointment. Pay particular attention to the urethral fossa where the organism is harboured.

After treatment of infections, cultures of commensals from the penis of a normal stallion have been used to recolonize the treated stallion's penis. However, the efficacy of this technique has not been reported.

Code of Practice for EVA (in Great Britain)

All unvaccinated stallions and teasers should be serologically tested prior to the breeding season. Seropositive stallions should be isolated pending virus detection. Semen used for AI, whether fresh, chilled or frozen, can act as a source of infection of EVA. All stallions used for AI should therefore be tested negative for EVA prior to the breeding season. Mare owners should also establish the antibody status of the donor stallion before importation of semen. If the stallion is seropositive, the semen should not be used unless it can be proved that seroconversion was due to prior vaccination.

Physical examination of the reproductive tract

- Penis: examine during the washing procedure.
- Scrotum and testes:
  Palpation.
  Ultrasonography.
- Internal genital organs:
  Palpation (per rectum).
  Ultrasonography.

Abnormalities of the genital tract

A. Scrotum – acquired

Inflammation or swelling raises the temperature and adversely affects spermatogenesis. It takes approximately 2 months after the lesion resolves for the stallion to produce normal ejaculates, but it can take considerably longer.

Causes of swelling

1. External:
   Dermatitis.
   Trauma.
   Ventral oedema – check for EVA and EIA.
2. Internal:
   Haematocele (Figure 14.16).
   Hydrocele (Figure 14.17).
   Varicocoele (Figure 14.18).
   Herniation.
   Peritonitis.
   Haemorrhage.
3. Diagnosis:
   Ultrasonography is of diagnostic value (needle aspiration of scrotal contents is not necessary or recommended)

Acute trauma

Treatment

Cryotherapy – icepacks with simultaneous massage. Should not exceed 20 minutes; apply at 1- to 3-hour intervals. Systemic anti-inflammatory and antibiotic treatment may be administered. Emollients should be applied to the skin to prevent maceration.

Intrascrotal haemorrhage or haematocele leads to permanent testicular damage due to the insulating properties of the resultant

Figure 14.16 Ultrasound image of scrotal haematocele using a 5-MHz linear transducer. Note the increased amount of fluid in the vaginal space containing fibrin strands and small blood clots.

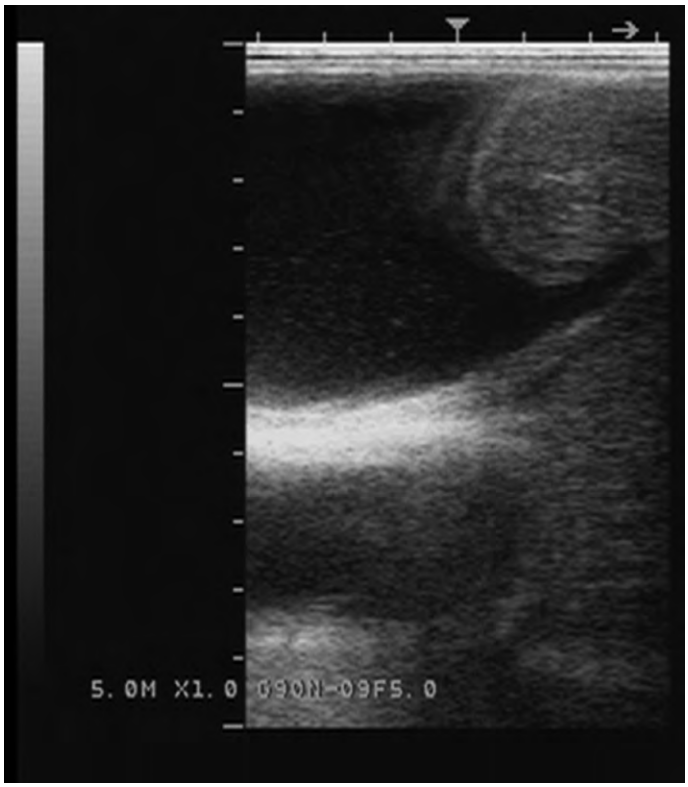


Figure 14.17 Ultrasound image of scrotal hydrocele (clear-amber fluid within the vaginal tunic) using a 5-MHz linear transducer. Note the atrophied testis that has probably resulted from impinging pressure of the enlarged plexus onto the testicular parenchyma.

Figure 14.18 Ultrasound image (5-MHz linear transducer) of varicocele of the pampiniform plexus.

fibrous tissue. The organized blood clot can be surgically removed in an attempt to save the testis.

Scrotal lacerations

Treatment

- Cleanse with isotonic saline solution containing antibiotic.
- Suture parietal vaginal tunic.
- Debride subcutaneous tissues.
- Skin closure with non-absorbable suture.
- Systemic administration of antibiotics and NSAIDs.

Hydrocele

- Abnormal collection of fluid between visceral and parietal vaginal tunics.
- Usually not painful.
- Can accompany scrotal oedema.

Treatment

- Remove underlying cause.
- Exercise.
- If unilateral and persistent, remove affected testis and tunics to save contralateral testis from heat-induced degeneration.

Herniation

See Chapter 8.

B. Scrotum – congenital

Defective scrotal development can occur in intersex horses.

Male pseudohermaphroditism

- Testes are usually intra-abdominal or inguinal.
- Stallion-like behaviour.
- Variable phenotype – underdeveloped penis or enlarged clitoris.
- 64XX or mosaic karyotypes.

Testicular feminization

- XY karyotype.
- Female phenotype.
- Abdominal testes – male behaviour.

X–Y sex reversal

- XY karyotype.
- Female genitalia – small inactive ovaries and hypoplastic tubular genital tract.

C. Testis

Cryptorchidism

See Chapter 8.

Haematoma

Extensive necrosis can occur leading to complete loss of testicular function.

Diagnosis

Ultrasonography.

Treatment

- If haemorrhage is contained within the tunica albuginea, adhesions may not occur. Treat as for acute scrotal trauma.
- If haemorrhage is massive – hemicastration.

Torsion

See Chapter 8.

Neoplasia

Seminoma is the most common tumour.

- Germinal epithelial origin.
- Locally invasive.
- Metastases reported.

Teratoma is found predominantly in cryptorchid testes.

Uncommon tumours

- Interstitial cell.
- Sertoli cell.
- Embryonal carcinoma.
- Lipoma, fibroma, leiomyoma.

Small testes

Hypoplasia

- Relatively common (3% of stallions in one survey).
- Incomplete gonadal development. Usually associated with an underdeveloped epididymis.
- Possibly genetic.

Degeneration

- Major cause of subfertility/infertility.
- Acquired condition.
- Numerous causes:
  thermal injury.
  infection.
  hormonal disturbances.
  malnutrition.
  toxins.
  autoimmune disturbances.
  drugs.

Both conditions can present as:

- Unilateral or bilateral (N.B. Left testis tends to be larger than right in normal stallions).
- Soft or firm consistency.
- Oligo- or azoospermic.
- Round germ cells in ejaculate. May be multinucleate.
- Libido usually unaffected.

History is important in differentiating between hypoplasia and degeneration. Unlike testicular hypoplasia, testicular degeneration may be reversible by removing the factor responsible for changes. Replacement therapy with gonadotrophin-releasing hormone (GnRH) may be more useful in stallions with hypogonadotrophic hypogonadism (defect in pituitary-hypothalamic axis – low LH and follicle-stimulating hormone [FSH]) than in stallions with damage to the seminiferous epithelium (suggested by elevated concentrations of FSH). However, the use of GnRH in stallions remains controversial.

Orchitis – bacterial

This is rare.

Diagnosis

Affected testes are hot, slightly swollen, tense and acutely painful. Fever and scrotal oedema are common.

Treatment

Treatment includes antimicrobial therapy, NSAIDs, hydrotherapy. Prompt removal of a unilaterally affected testis may save the contralateral testis.

Orchitis – traumatic

Diagnosis

- Hot, tense, painful testis.
- Swelling is restricted by the tunica albuginea.
- Increased testicular pressure and temperature lead to marked testicular degeneration.

Treatment

Counteract local inflammation as described above.

D. Epididymis

- Epididymitis. Rare. Usually secondary to orchitis.
- Spermiostasis. In some stallions, sperm accumulate abnormally in the epididymis and deferent ducts during sexual rest. Semen contains a large proportion of detached sperm heads. High sperm numbers in ejaculate and sperm may be clumped. Treat by frequent ejaculation. Can recur if the stallion does not ejaculate regularly.

E. Vesicular glands

Infection of the vesicular glands. There are inflammatory cells in the semen. The stallion may be infertile. Systemic treatment is often unsuccessful. Local irrigation of the glands and infusion of antibiotics via an endoscope may be tried.

F. Ampullae

When the ampullae are blocked spermiostasis occurs and the ampullae feel tense and swollen on palpation. The condition is treated by massage and frequent ejaculation. Oxytocin (10–20 IU) may be administered prior to ejaculation.

G. Prepuce and penis

Paraphimosis

See Chapter 8.

Haematoma

This often follows a kick from the mare at breeding. It usually results from haemorrhage of superficial vessels on the dorsum of the penis. There is rapid swelling, and it can lead to paraphimosis.

Treatment

- Apply compression bandage then treat as for paraphimosis.
- Daily massage to minimize fibrosis.

Coital exanthema

- Caused by EHV-3.
- Pox-like lesions on penis.
- Venereally transmitted.
- Self-limiting.
- Complete resolution in 3–5 weeks.

Treatment

- Sexual rest.
- Daily application of protective emollients.

Squamous cell carcinoma

See Chapter 8.

Squamous papilloma

- Develop only rarely on penis or prepuce.
- Multiple proliferative cutaneous growths that usually regress spontaneously in 1–6 months.

Cutaneous habronemiasis

See Chapter 8.

Altered semen

In urospermia urination occurs during ejaculation.

Treatment

- Train the stallion to urinate prior to collection.
- Fractionation of ejaculate – only useful if the stallion urinates in the final stages of ejaculation.
- Centrifugation and resuspension of sperm in extender.
- Can try diazepam (100–500 mg orally twice a day).

For haemospermia see Chapter 8.

Abnormal reproductive behaviour in stallions

Behaviour-related fertility or management problems comprised over 25% (n = 250) of stallion fertility problems referred to a large American clinic over a 5-year period.

Low libido – causes

- Managerial.
- Musculoskeletal.
- Hormonal.

Managerial

May have preference for:

- Mare.
- Handler.
- Location.

Treatment

- Keep quiet.
- Try outdoor breeding.
- Blinkers.
- Hot compresses applied to base of penis while artificial vagina is on glans.
- Watch another stallion (voyeur effect).
- Use more than one mare.
- Use unrestrained mare in oestrus.

Musculoskeletal

Treatment

- Provide mount mare of appropriate height.
- Position mare on slope downhill from stallion.
- Collect semen with stallion standing on ground.
- Treat pain.
- Ensure good footing.
- Provide lateral support.

Hormonal

1. Impotent stallions tend to have lower blood concentrations of LH and oestradiol-17β whereas concentrations of testosterone tend to be similar to normal stallions.
2. Administration of low concentrations of testosterone (50–200 μg/kg SC every other day) does appear to help slow-starting stallions. Plasma testosterone should be monitored to avoid reaching undesirably high blood concentrations. Increased sexual interest should be apparent within 10 days. Novice stallions generally do not need treatment after the first ejaculation.
3. Treatment of slow stallions with 50 μg GnRH subcutaneously 2 hours and then 1 hour before breeding may increase sexual arousal. Typically, concentrations of testosterone in blood double after this treatment regimen.

Ejaculatory dysfunction

This is a relatively common disorder.

Discharge of semen into the urethra appears to be caused by stimulation of α-adrenergic receptors and by inhibition of β-adrenergic receptors, whereas transport of semen through the urethra is regulated by the somatic nervous system.

1. Often presents as a stallion with good libido and normal copulatory behaviour.
2. May appear to ejaculate (tail flags, urethral pulsations) but does not.
3. May ejaculate after several breeding attempts.

Causes

1. Psychogenic.
   Breeding injury.
   Overuse.
   Aggressive handling.
   Unfamiliar surroundings.
2. Organic.
   Damage to the dorsal nerve of the penis.
   Malfunction of the autonomic nervous system.
   Blocked ejaculatory ducts.
   Retrograde ejaculation (semen expelled into bladder).

Treatment

Treatment depends on cause.

Pharmacological aids

1. α-agonist (L-norepinephrine 0.01 mg/kg IM) 15 minutes before breeding plus β-antagonist (carazolol 0.015 mg/kg IM) 10 minutes before breeding. Used to treat organic ejaculatory failure. Do not use in stallions with recurrent airway obstruction (RAO).
2. Xylazine (α-adrenergic): In normal stallions, spontaneous ejaculation occurs 25% of the time after administration (0.66 mg/kg IV).
3. $PGF_{2\alpha}$: Spontaneous ejaculation in 50% of stallions within 10 minutes of administration (0.01 mg/kg IM).
4. Diazepam: Used in timid stallions at a dose of 0.05 mg/kg (slow) IV 5–7 minutes before breeding.

14.7 REPRODUCTIVE MANAGEMENT

The official breeding season for Thoroughbreds starts on 15 February in the northern hemisphere and 1 August in the southern hemisphere. As many mares only start to cycle later in the spring, manipulation of the breeding season is frequently practised.

Artificial control of photoperiod

- Start 1 December for 15 February breeding season.
- Maintain a ratio of 16 hours light: 8 hours dark. Add light in evening. Continuous light may delay onset of cyclicity.
- A 12 × 12 ft loose box requires a 200 W bulb.

Vernal transition

- Lasts 30–60 days.
- Numerous large follicles (>30 mm) are present on the ovaries, but the mare remains anovulatory during this period.
- Mare remains in oestrus for prolonged periods.

Onset of normal cyclicity may be hastened by:

- Exposure to artificial light by December 1st will hasten the onset of the vernal transition and normal cyclicity.
- Altrenogest daily for 2–3 weeks.
- Twice daily injections of buserelin (10 μg).

Breeding season

- Mares should be teased at least three times per week (preferably daily).
- One follicle, occasionally two, becomes dominant and ovulates on the last or second last day of oestrus. Mean diameter at ovulation is 45 mm.
- Traditionally, mares are bred on day 2 of oestrus or when a 30 mm follicle is present on the ovaries, and then every other day until the end of oestrus. If it is necessary to limit the number of breedings, the ovaries should be palpated and/or scanned daily, and the mare should be bred on the day before ovulation is anticipated. Induction of ovulation with human chorionic gonadotropin or GnRH analogues (deslorelin) may be used for follicles >35 mm; ovulation takes place within 48 hours. Breeding after ovulations should be avoided as the chances of failure to conceive or early embryonic death may increase if breeding takes place at >12 hours after ovulation.

Aberrant patterns of cyclicity

Persistent oestrus

Check behaviour with stallion – many owners misdiagnose true signs of oestrus.

Causes

- Seasonal anoestrus – mares may show weak signs of oestrus in the absence of the strongly inhibiting effects of progesterone.
- Transitional breeding season.
- Granulosa cell tumour.

Treat first two with altrenogest.

Prolonged dioestrus

- Occurs in bred and non-bred mares.
- Failure of luteal regression at end of dioestrus, and therefore mare fails to return to oestrus.
- Up to 25% of cycles may be affected.
- CL may persist for 2 months or more.
- Can result from dioestrous ovulation on day 9–14 (the developing CL is unresponsive to one single injection of PGF2α if administered before day 5 post ovulation) or early embryonic death.
- Treat with $PGF_{2\alpha}$.

Pseudopregnancy

- A syndrome in which non-pregnant, bred mares do not return to oestrus or ovulate, and uterine tone is characteristic of early pregnancy.
- Thought to result from embryo loss but can occur occasionally in non-bred mares (not convincingly documented).

Split oestrus

- In oestrus for several days, then out for 1–2 days, followed by several more days of oestrus.
- Ovulation usually does not occur until the second half.
- More common early in the breeding season.

Double ovulations

- Occurs at about 16% of ovulations.
- Tends to occur repeatedly in certain mares (especially Thoroughbreds, draught breeds and Warmbloods).
- Conception rates are higher than in single ovulating mares, therefore breed the mare.
- Check the mare for twin pregnancy by ultrasonography preferably before day 16 (day of fixation) and crush one conceptus if twins are present.
- If twins have fixed in the one horn (70%) and it is not possible to crush one, natural reduction rates prior to day 40 are 89%. However, in bilateral fixation, only 11% reduce naturally.

Foaling mares

- Foal heat usually occurs at 7–9 days. Second oestrus around 30–35 days (30-day heat).
- Pregnancy rates for mares bred at the foal heat tend to be lower, and embryonic death rates higher than for mares bred at subsequent heats. Select mares with history of normal foaling and that have no fluid in the uterus at time of breeding.
- Mare must conceive by day 25 to foal at the same time the next year.

Lactational anoestrus

- Fifty per cent of pony mares and most Thoroughbred mares have a foal heat after foaling in the anovulatory season.
- Approximately half of these mares return to anoestrus after their first postpartum ovulation.
- The incidence of lactational anoestrus in the breeding season is low. Can stimulate ovulation within 14 days by weaning the foal.

Pregnancy losses (Table 14.1)

Seventy-seven per cent of all detected pregnancy losses occur before day 49.

Manipulation of the oestrous cycle

- Progestagens: Altrenogest – synthetic, oral (Regumate). Oestrus occurs 4–5 days after a 12- to 15-day treatment period.
- Prostaglandin $F_{2\alpha}$: CL must be between 6 and 14 days old to respond to one single injection; multiple injections of $PGF_{2\alpha}$ are needed if luteolysis is attempted before day 5 postovulation. Come into oestrus in 2–5 days. Ovulation is variable (3–12 days) depending on follicular development at time of treatment.

Table 14.1 Pregnancy losses

	Fertile mares	Subfertile mares
Fertilization rate (%)	>90	>80
Embryonic loss rate (%)		
<day 14	9	70
day 0–40	20	>70
>day 60	9	

Side-effect is display of signs of colic (sweating, trembling, muscle cramping) that subside in 30 minutes; it appears to be dose related.

- Progesterone/oestradiol: 150 mg progesterone plus 10 mg oestradiol IM for 10 days followed by $PGF_{2\alpha}$ (10 mg) on day 10. Oestrus occurs on approximately day 16 with ovulation on day 20–22.
- Human chorionic gonadotrophin (hCG): hastens ovulation of a mature follicle; 2500 IU IV or IM causes ovulation of a 35-mm follicle in 36–48 hours.
- Gonadotrophin-releasing hormone: Buserelin (GnRH) does not hasten ovulation unless given twice daily during oestrus. Deslorelin is a GnRH analogue that induces ovulation of a 35-mm follicle at 36–48 hours. It is administered as a short-term implant (2.1 mg deslorelin) or as intramuscular injections (1.5 mg per mare) from preparations made by compounding pharmacies.

Teasing programmes

Mares should be teased daily through oestrus. Teasing after breeding is a good method of detecting early pregnancy loss.

The teaser stallion should be an intact male horse with the following characteristics:

- Good libido.
- Easy to handle.
- Not easily frustrated.

Teasing systems are designed to suit the farm's management.

- The stallion teases the mare over a teasing board or teasing stocks.
- The stallion is placed in a small pen in the mares' pasture.
- The stallion is led to the fence of the mares' pasture.
- The stallion is brought to the door of the mare's loose box.
- The mare is brought to the door of the stallion's loose box.

It is important that each mare be given the chance to exhibit oestrous behaviour – watch for subordinate mares in group-housing situations.

Breeding management

Preparation of the stallion for breeding

- A breeding bridle or a headcollar with a chain through the mouth or over the nose should be placed on the stallion.
- Tease the stallion with a mare in oestrus until he achieves a full erection.
- The penis may be rinsed with warm water and dried with a soft paper towel. Avoid antiseptics.
- Allow stallion to tease the mare and then mount. Usually a groom holds the mare's tail out of the way and another inserts the stallion's penis into the mare's vagina.
- At time of ejaculation, the stallion's tail flags and 7–9 pulsations are detected at the base of the penis.
- A dismount sample can be collected to check for the presence of sperm.
- After the stallion dismounts, the penis may again be rinsed with warm water.

Preparation of the mare for breeding

- Use a bridle or headcollar plus twitch.
- Place padded boots on the hindfeet. Can use breeding hobbles, but the stallion can catch a foot in these. Can tie a forelimb up before stallion mounts (should be released after intromission).
- Bandage tail or place in plastic sleeve.
- Wash vulva, perineal region and hindquarters with mild soap. Rinse and dry.

Minimum contamination technique

In mares susceptible to endometritis, artificial insemination minimizes antigenic challenge to the mare's uterus. If AI is not permissible, then the mare should be bred once only. Scrupulous hygiene should be applied to both the stallion and the mare. Semen extender containing antibiotic (100 mL) can be infused into the mare's uterus immediately prior to breeding. The mare's uterus can either be flushed with buffered saline solution 4 hours after breeding to physically remove contaminants, or the mare can be scanned 12 hours later. If fluid is present, the mare can be treated with an injection of oxytocin followed by daily intrauterine antibiotic infusions for 4 days after ovulation, possibly in combination with uterine lavage.

Artificial insemination (AI)

Semen collection and preparation for AI

Artificial vagina

- CSU Model – expensive, heavy, well-insulated.
- Missouri – inexpensive, light, easy to use, needs constant cleaning, less insulated than the CSU model (Figure 14.19).
- Nishikawa – moderately expensive, tends to leak water.

1. Artificial vagina must be clean and dry. A disposable plastic liner should be used.
2. To clean:
   Soak for 20 minutes in 70% alcohol.
   Hang up to dry.
   Rinse with distilled water.
   Dry.
3. Fill with hot water so that internal temperature is 45°C (113°F).
4. Use non-spermicidal lubricant, e.g. K-Y jelly.

Semen treatment

1. Filter to remove gel.
2. Protect from cold shock, water, sunlight.

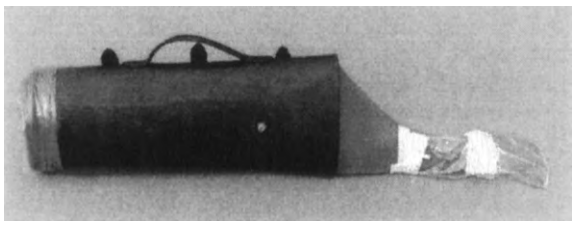


Figure 14.19 Missouri AV assembled ready for use.

3. Evaluate:
   Motility (total and progressive).
   Volume.
   Concentration.
4. Extend 1:1 or 1:2, or, optimally, dilute to 25–50 million per mL for chilled semen.
5. Most commonly used extender is non-fat skim milk powder containing glucose and antibiotics.
   Non-fat dry skim milk: 2.4 g.
   Glucose: 4.9 g.
   Make up to 100 mL with sterile distilled water.
   Add penicillin (150 000 units) + streptomycin (150 000 µg),
   or add ticarcillin (100 mg),
   or add reagent-grade gentamicin (100 mg) + $NaHCO_3$ (2 mL, 7.5%).
6. Mares should be inseminated with a minimum of 300 million progressively motile, morphologically normal sperm.
7. For shipping cooled semen, dilute the ejaculate with extender to reach a final concentration between 25–50 million sperm/mL. That results in a typical insemination dose of 30 to 60 mL that should contain at least 1 billion progressively motile sperm.

AI of the mare

Advantages

- Mares can be bred by a stallion that is geographically inaccessible.
- Mares with young foals are not transported from home.
- Mare can still be ridden.

Disadvantages

- High veterinary expenses.
- If the mare is to remain at home, mare owners must tease mare and communicate with vet and stud farm.
- Semen shipment costs.

Preparation of the mare

- Wrap tail in sleeve or bandage.
- Empty rectum.
- Wash perineal region three times with mild soap, dry.
- Draw semen into Air Tite syringe.
- Wear a clean plastic sleeve and a sterile glove.
- Lubricate hand with sterile, non-spermicidal lubricant.
- Introduce plastic insemination pipette into the uterus; the tip is covered by the hand as it is passed through the vagina. Deposit the semen in the uterine body. Hold cervix shut as the pipette is withdrawn.

Cooled semen

- Before committing a stallion to an AI programme, the stallion's semen must be checked to ensure that it withstands the cooling process satisfactorily.
- Semen should be evaluated for motility and concentration. Extend to 25–50 million progressively motile sperm/mL.
- Prepare two packages of semen for shipment. Keep a sample of the extended semen at the stud for evaluation 24 hours later.
- Semen is double-bagged in Whirl-Pak® bags (Nasco, Fort Atkinson, WI, USA) or placed in 50-mL polypropylene centrifuge tubes). If using bags the samples are placed in a polystyrene cup, and excess space inside the cup is filled with ballast bags containing a blue dye. The cup or the 50-mL centrifuge tubes are then loaded into the Equitainer.
- Should be used within 48 hours of collection.
- Do not open Equitainer until mare is prepared for insemination.
- Inseminate mare (the semen does not need to be pre-warmed).
- Check motility of sperm by placing on a slide at 37°C. Motility improves over a few minutes.
- For mares with normal fertility, optimum pregnancy rates are obtained when ovulation takes place within 48 hours after insemination.
- Check mare for pregnancy 12–14 days after ovulation; if twin pregnancy is diagnosed, manually crush one vesicle at this time.
- Pregnancy rates as good as natural service if semen is of good quality, and mares are fertile.

Frozen semen

- Most commonly stored in 0.5 mL straws or 4 mL macrotubes in liquid nitrogen.
- Quality of semen after freeze–thaw process varies markedly between stallions (only approximately 30% of stallions produce semen that has good motility after thawing).
- Pregnancy rates variable depending on semen quality. The mare must be checked at least every 12 hours and inseminated at the examination before and after ovulation. Alternatively, scan every 6 hours and inseminate at the first examination after ovulation.

Advanced assisted reproduction

Embryo transfer

Advantages

- Embryo can be collected from performance mares and transferred to recipient mare. This means that the donor mare can remain in training.
- Embryos can be collected from mares that are unable to carry a foal to term. Recovery rate of embryos is very poor from mares with endometritis but can be as high as 80–90% from mares without reproductive disease.
- A number of embryos can be collected from a valuable mare over one breeding season.
- It is possible to chill embryos in Equitainers and transport them. At this time embryo freezing is producing poor results.

Problems

- The mare can be superovulated following administration of equine pituitary extract (EPE) or equine FSH (eFSH); however, both drugs are not available commercially. For reasons not yet completely elucidated, mares ovulating more than 4 follicles per cycle have poor embryo recovery rates.
- Due to problems with precise synchronization of ovulation it is necessary to have two recipient mares.
- Embryo transfer is relatively expensive due to mare and veterinary costs.

Procedure

The oestrous cycles of donor and recipient mares are synchronized. The donor mare is bred at oestrus.

Ovulation is monitored in all mares. Optimal pregnancy rates are obtained when embryos are transferred to recipients that ovulate between 3 days before and 1 day after the donor ovulates.

Although embryos can be recovered on days 6 to 9 after ovulation, the optimal time of embryo collection is day 7 or 8. If the goal is

to freeze the embryo, the collection attempt should be made on day 6 after ovulation. The uterus of the donor mare is lavaged on average with 4 litres of complete flush media (several brands are commercially available) that contains a buffered solution, antibiotics, and albumin.

The embryo is identified using a stereomicroscope at 15× magnification, loaded into a 0.5 mL plastic straw and transferred transcervically into the uterus of the recipient mare. Day 8 or 9 embryos may be loaded into an insemination pipette.

Scrupulous hygiene must be maintained to prevent introduction of contamination that could potentially cause endometritis and failure to establish pregnancy. Embryos of good quality transferred to reproductively healthy recipients should result in pregnancy rates of 70–80%.

The donor mare is given $PGF_{2\alpha}$ to bring her back into oestrus.

Oocyte transfer (OT) and gamete intrafallopian transfer (GIFT)

Indicated for infertile, old mares or mares with severe vaginal, cervical or uterine disease that are unable to carry a pregnancy to term or produce an embryo under conventional embryo collection procedures.

Transvaginal ultrasound-guided needle aspiration of preovulatory follicles is used to harvest in vivo-matured oocytes. These oocytes can then be transferred (OT) into a recipient mare's uterine tube that has been inseminated or covered; or, or alternatively, oocytes and low sperm numbers are transferred into one of the recipient mare's oviducts.

Intracytoplasmic sperm injection (ICSI)

Conventional in vitro fertilization (IVF) has not been as successful in horses as it has been in cattle. However, using in vitro manipulation techniques, the injection of a single sperm cell into a mature oocyte (ICSI) has been proved successful. The ICSI is currently available in several equine reproduction laboratories around the world.

After fertilization is documented, the embryo is cultured for another 24–48 hours before being transferred to the uterine tube of a synchronized recipient mare. Recent success has been obtained after culturing the embryo in vitro for several days until it reaches the morula or blastocyst stage, thus allowing the non-surgical direct transfer into the uterus.

Artificial insemination with low sperm numbers

Acceptable pregnancy rates using low sperm numbers (1–5 million sperm) can be achieved if the sperm is deposited at the tip of the uterine horn, ipsilateral to the preovulatory follicle.

Deep horn insemination is carried out by using a special flexible plastic artificial insemination pipette or via hysteroscopic-guided insemination of very small numbers of spermatozoa deposited at the uterotubal junction. Acceptable pregnancy rates can be obtained using low-dose insemination.

Cloning

The cloning of horses using adult somatic cell transfer has been produced by several research groups and it is commercially available in a few countries.

FURTHER READING

Ginther OJ (1992) Reproductive biology of the mare, 2nd edn. Equiservices, Wisconsin

Horserace Betting Levy Board. Common code of practice for control of contagious equine metritis and other equine bacterial venereal diseases and equine viral arteritis. HBLB, 52 Grosvenor Gardens, London SW1W OAU

McKinnon AO, Voss JL (1992) Equine reproduction. Lea & Febiger, Philadelphia

Varner DD, Schumacher J, Blanchard TL, Johnson L (1991) Diseases and management of breeding stallions. American Veterinary Publication, Goleta, CA

APPENDIX 1

Differential diagnoses of reproductive problems in the mare

1. Anoestrus

Season
- autumn
- winter

Failure to tease adequately

Ovarian acyclicity
- granulosa cell tumour
- atrophy
- hypoplasia

Prolonged dioestrus

Pregnancy

Pseudopregnancy

Lactational anoestrus

2. Excessive signs of oestrus

Misdiagnosis of signs of oestrus

Season
- transitional
- winter anoestrus

Granulosa cell tumour

3. Failure to conceive

Breeding at the wrong time

Endometritis

Uterine disease (large cysts, tumours, adhesions, etc.)

Cervical laceration

Cervical adhesions

Urovagina

Poor quality semen

Chromosomal abnormality

Anovulation

Hydrosalpinx

4. Abortion

Infectious
- bacterial

viral
fungal
Non-infectious
twinning
umbilical cord abnormalities
placental insufficiency
uterine body pregnancy

5. Vulvar bleeding

Prepartum
vestibular/vaginal varicosity
placental detachment
Postpartum
uterine rupture
endometrial haemorrhage
vaginal or vestibular damage

6. Vulvar discharge

Endometritis
Cervicitis
Vaginitis
Urovagina
Urological problem

APPENDIX 2

Differential diagnosis of reproductive problems in the stallion

1. Enlarged scrotum

Dermatitis
Trauma
Ventral oedema
Haematocele
Hydrocele
Varicocele
Herniation
Peritonitis
Haemorrhage

2. Defective scrotal development

Male pseudohermaphroditism
Testicular feminization
X–Y sex reversal

3. Enlarged testis

Haematoma
Torsion
Neoplasia
Orchitis

4. Small testis

Hypoplasia
Degeneration

5. Poor quality semen

Season
Urospermia
Haemospermia
Contamination with disinfectants etc.
Poor handling
heat
cold
light
oxygen
agitation
Infection
Testicular hypoplasia/degeneration
Chromosomal abnormality
Impaired epididymal function
Blocked ejaculatory ducts
Elevated scrotal temperature
Hormonal imbalances
Malnutrition
Ageing
Overuse

6. Low libido

Managerial
Musculoskeletal
Hormonal

7. Ejaculatory dysfunction

Psychogenic
Nerve damage or malfunction
Blocked ejaculatory ducts
Retrograde ejaculation

8. Erection failure

Psychogenic
Local vascular or neurological damage

Chapter 15

Orthopaedics 1. Diagnosis of lameness/diseases of joints and bones

Raphael Labens, Michael C.A. Schramme, Alistair R.S. Barr

CONTENTS

15.1 Diagnostic approach to lameness 309
History 309
Physical examination 310
Gait evaluation 310
Provocative tests 311
Regional analgesic techniques 311
Arthrocentesis and analysis of synovial fluid 315
Radiography 316
Ultrasonography 316
Nuclear scintigraphy 318
Thermography 318
Computed tomography (CT) 318
Magnetic resonance imaging (MRI) 318
Diagnostic arthroscopy 318
15.2 Joint diseases 319
Idiopathic synovitis 319
Traumatic arthritis/synovitis 319
Osteoarthritis (degenerative joint disease) 320
Osteochondritis dissecans (OCD) 321
Subchondral bone cysts (osseous cyst-like lesions) 322
Septic arthritis 323
15.3 Fractures 323
Fracture healing 324
Complications of fracture healing 324
Evaluation of equine fractures 324
Emergency stabilization 324
Medication 325
Radiography 325
Emergency transport 325
Surgical techniques 325
Aftercare 326
15.4 Other bone diseases 327
Osteomyelitis 327
Osteitis 327
Physitis 327
Nutritional secondary hyperparathyroidism (bran disease, big head, osteodystrophia fibrosa) 327
Hereditary multiple exostoses (multiple cartilaginous exostoses, osteochondroma) 328
Calcinosis circumscripta 328
Rickets 328
Hypertrophic osteopathy (hypertrophic pulmonary osteoarthropathy, Marie's disease) 328
Osteoporosis/osteopenia 328
Fluorosis 328
Further reading 328

15.1 DIAGNOSTIC APPROACH TO LAMENESS

Diagnosis of orthopaedic problems involves a number of basic steps namely:

- Defining the problem to be investigated.
- Localizing the site or sites of abnormality.
- Characterizing the nature of the pathological change.

The following two chapters describe disorders of the distal and proximal limbs of the horse. However, it is worth noting from the outset that lameness in horses is most commonly a result of abnormalities in the distal limb, i.e. in the carpus or hock and distally, particularly in the foot. The distal limb should therefore always be carefully evaluated first to eliminate it as the source of lameness.

History

1. Essential information includes:
   - Breed.
   - Age.

© 2013 Elsevier Ltd
DOI: 10.1016/B978-0-7020-2801-4.00015-8

- Sex.
- Duration of ownership.
- Use (or intended use) of the horse.
- Nature of the problem (lameness or loss of performance).
- Duration of the problem (recent or chronic).
- Onset of the problem (acute or insidious).

2. Details of recent management may be relevant including exercise, shoeing, housing and feeding.
3. Previous locomotor problems known to the owner should be noted.

Physical examination

Provided that the routine adopted for physical examination is comprehensive in its detail and scope, the precise order in which it is performed is not critical. Every effort should be made to examine the entire musculoskeletal system even in the presence of obvious abnormalities at one site. The examination at rest logically falls into two stages:

1. Overall inspection of the horse from all angles noting particularly:
   - General body condition.
   - Conformation of body, limbs and feet.
   - Posture and weight-bearing on the limbs.
   - Skeletal and soft tissue symmetry.
   - Any localized swellings or thickenings.
2. Detailed evaluation of all individual regions of the limbs by:
   - Inspection, which should reveal deformity, swelling or thickening, skin wounds and muscle wasting.
   - Palpation, which is helpful in detecting heat and pain as well as characterizing the precise location and consistency of any swellings or thickenings.
   - Manipulation of the joints, which allow evaluation of range of movement – detecting restriction, instability, pain or crepitus.

All three procedures should involve comparison of the region under examination with the contralateral limb to detect asymmetries.

Gait evaluation

The aim should be to identify:

1. The presence or absence of a gait abnormality.
2. The limb or limbs involved.
3. The character of any abnormality present.
4. The degree of abnormality.

- The gait is usually best evaluated on a hard level surface. Ideally this should be done in a safe, enclosed area free of distractions and dangers such as traffic and other horses.
- Some forms of lameness are best examined immediately after taking the patient out of its stable (i.e. 'cold lameness'). Chronic low-grade lameness may require a period of sustained exercise to become more obvious.
- Horses should be stripped of all tack, rugs and blankets and should be held by a loose rope that is fixed to a headcollar or bridle.
- Abnormalities of gait are usually most apparent when the horse is moving at the walk or slow trot. Initial evaluation should be performed at the walk to identify those horses which are markedly lame where trotting may be hazardous. Variations in foot placement and limb movement, e.g. shortening of one phase of the stride on one limb, are generally most easily appreciated at the walk when limb movement is slower. In addition, mechanical and neurological causes of lameness are often more apparent at the walk.
- Abnormalities in head and hindquarter movement resulting from pain during weight-bearing are usually most apparent at the slow trot.
- The horse should be observed moving in a straight line and at an even pace from in front, the side and behind.
- Forelimb lameness is best observed while the horse is trotted towards or past the examiner. Hind limb lameness is best observed while the horse is trotted away or past the examiner. Observation from the side is easier if the observer is on the side of the lame limb.
- Lunging the horse in tight circles is helpful in demonstrating more clearly lameness that is subtle or inapparent when the horse is moving in a straight line. If circumstances do not permit such an examination, trotting the horse around sharp corners may accentuate lameness in a similar, though more transient, way.
- It is rare for horses to have to be ridden or driven to make lameness noticeable. Lameness in Standardbreds is an exception to this rule and can often only be observed on the track at full racing speed.
- Canter or gallop is only occasionally of value in the observation of lameness.
- Horses with forelimb lameness due to pain on weight-bearing shift the distribution of weight from the affected limb across to the contralateral forelimb and back to the hind limbs. This is achieved, at least in part, by lowering the head and neck less as the lame forelimb takes weight, and, in compensation, lowering the head and neck more as the sound forelimb strikes the ground. This downward nodding of the head as the sound forelimb strikes the ground is generally the easiest abnormality of movement to appreciate and allows identification of the lame (or lamer) forelimb. The sound limb may also be heard to strike the ground with greater force, particularly if the horse is shod.
- In hind limb lameness the normal symmetrical vertical movements of both quarters, as observed from behind the horse, is disturbed. The quarter on the lame side rises and falls through a greater range of motion than that of the sound limb. This is usually characterized by a long downwards drop during the swing-phase of the stride, followed by a fast, sudden, upwards flick, or hike, during the weight-bearing phase of the stride.
- Head movements are less helpful in the recognition of hind limb lameness. With moderate to severe lameness, the horse may attempt to shift its centre of gravity forward when the lame hind limb starts to bear weight, which can give the misleading impression of a downward head nod during the support phase of the contralateral forelimb, and hence ipsilateral forelimb 'referred' lameness.
- Bilateral forelimb or hind limb lameness is often very difficult to recognize when the horse moves in a straight line. The horse usually shows a stiff, stilted gait with bilaterally shortened stride length. More pronounced unilateral signs of lameness are usually observed when the limbs are subjected to uneven stresses (e.g. by lunging in tight circles).
- Abnormalities of limb movement may also be appreciable in the lame horse:
  - *Alteration in the relative lengths of phases of the stride*. The cranial phase of the stride is that part which occurs in front of the footprint of the contralateral limb, while the caudal phase occurs behind it. If the horse is moving in a straight line, the overall stride length in a pair of contralateral limbs must be even; therefore a reduced cranial phase must always be accompanied by an increased caudal phase. Overall reductions in stride length frequently accompany bilateral

orthopaedic conditions leading to a 'pottery' or restricted gait.

- *Alteration in the arc of foot flight*. Lowering of the arc of foot flight may occur as a compensation to reduce impact when the foot lands or to reduce limb flexion during protraction. If severe, it may lead to dragging of the toes. Exaggerated elevation of a foot due to hyperflexion of the limb joints is occasionally seen in neurological conditions, such as 'stringhalt'.
- *Variations in the path of foot flight and in foot placement*. These may occur for similar reasons to those that cause alterations in the arc of foot flight. The foot may be swung medially or laterally during protraction of the limb. The foot may land asymmetrically contacting the ground first at the toe, heel or on one side.
- *Extension of the metacarpophalangeal/metatarsophalangeal joint*. Pain-related lameness results in a reduction in the extension of the metacarpophalangeal joint providing there is no disruption of the palmar soft tissue supporting structures, hence making this sign a useful predictor of the lame limb.

Certain gait characteristics may be indicative of the site of pain:

- Swinging limb lameness often originates from the proximal limb.
- Supporting limb lameness usually reflects pain in the distal limb.
- In bilateral lameness, the cranial phase of the stride is bilaterally shortened, even in distal limb lameness. This results in a stiff, stilted gait.

It is useful to examine the horse on both soft and hard ground.

- Lameness in the distal limb is often exacerbated on hard ground.
- Proximal lameness may be exacerbated on soft ground.
- Lunging or circling the horse provides further information for the observer:
  Proximal lameness may be exacerbated with the affected limb on the outside of a circle.
  Distal lameness is usually worse with the affected limb on the inside of a circle.

These 'rules' are certainly not absolute. For instance, one must take into account whether the painful locus is located on the medial or lateral aspect of the limb, and certain weight-bearing lamenesses, such as those arising from the proximal suspensory region, are commonly associated with greater lameness with the limb on the outside of the circle.

Records of a lameness evaluation should contain some assessment of the severity of the problem. A variety of scoring systems are employed, e.g. 0 to 5 or 1/10th to 10/10ths, with higher scores representing more severe grades of lameness.

AAEP scale:

0. Lameness not perceptible under any circumstances.
1. Lameness is difficult to observe and is not consistently apparent, regardless of circumstances (e.g., under saddle, circling, inclines, hard surface, etc.).
2. Lameness is difficult to observe at a walk or when trotting in a straight line, but consistently apparent under certain circumstances (e.g., weight carrying, circling, inclines, hard surface, etc.).
3. Lameness is consistently observable at a trot under all circumstances.
4. Lameness is obvious at a walk.
5. Lameness produces minimal weight-bearing in motion and/or at rest or a complete inability to move.

0–10 scale (assessed at the trot):

0. Sound.
1. The minimal degree of lameness detectable which may be inconsistent.
2. A consistent, but mild, degree of lameness – detectable and consistent subtle head-nod.
3. Consistent and obvious head nod/pelvic asymmetry.
4. Pronounced head nod/pelvic asymmetry.
5. Marked head nod/pelvic asymmetry.
6. Very marked head nod/pelvic asymmetry.
7. Difficulty trotting; only just able to place heels to the ground.
8. Minimal weight-bearing, heels not placed on the ground.
9. Only able to touch the limb to the ground.
10. Unable to put limb to the ground.

Provocative tests

Provocative tests may be used for three basic reasons:

1. To demonstrate occult lameness in a horse that appears 'sound' on initial gait evaluation.
2. To exacerbate a mild lameness.
3. To aid localization of the abnormality causing the lameness.

A variety of manoeuvres are employed. The most commonly used are '*flexion tests*':

- A flexion test is performed by holding the joint under consideration in a firmly flexed position for a period (usually 1 minute) and then immediately watching the horse move, usually at the trot, to detect any change in gait compared to that observed before performing the test. The response to a flexion test should be interpreted in the light of other findings, and it is wise to avoid using it as the sole criterion upon which to base a firm diagnosis.
- As far as possible, the examiner attempts to flex the suspected joint only. Since some joints are inherently linked together in flexion and extension (i.e. hock and stifle; phalangeal joints and fetlock), exact differentiation of pain responses between these joints is not possible.
- Extension of the distal interphalangeal (DIP) joint may be performed using a wedge-shaped piece of wood with a 20° inclination, which is placed under the weight-bearing foot to raise the toe and increase the load on the deep digital flexor tendon (DDFT), the navicular bone and its ligaments. After 2 minutes, the horse is trotted away. Unfortunately, the response to this test is inconsistent, even in confirmed cases of navicular syndrome.
- Response to localized pressure over exostoses, tendon swellings, splints, etc., can be assessed. Localized pressure is maintained for 1 minute over the suspected site of pain, after which the horse is immediately trotted away.
- In all tests, the examiner looks for any significant exacerbation of lameness. Only by experience can one learn where the boundaries of normality lie for the amount and duration of tension applied. A sound horse should not show consistent lameness following any of these tests.

Regional analgesic techniques

'Nerve blocking' is time-consuming, invasive, and sometimes hazardous to both horse and examiner. It relies on subjective evaluation of gait for its interpretation. Despite these disadvantages, it is often the only way to determine the answer to the question 'where does it hurt?'.

Local analgesic solutions can be used in a variety of ways to localize the source of pain responsible for lameness.

- The use of local analgesia relies on accurate placement of local analgesic into or around the structure to be desensitized, followed by evaluation of its effect on the gait.
- The results of regional analgesia are most easily and reliably interpreted in horses with an obvious and consistent degree of lameness.
- Interpretation is more difficult and less reliable in horses with slight or inconsistent lameness.
- If the lameness is chronic and low-grade, it may be helpful to exacerbate the problem by exercising the horse for a few days to make the lameness more apparent prior to the use of local analgesia.
- Conversely, caution should be exercised with the use of regional analgesia in acutely lame horses if there is a possibility that the cause may be an injury which could be exacerbated by injudicious use of a limb rendered pain free by the use of local analgesia. An example would be a horse with an undisplaced fracture that may displace with increased weight-bearing. Initial radiographic and/or scintigraphic examination may be prudent if such injuries are suspected.

Local analgesia may be used for:

1. Perineural infiltration around specific nerves to desensitize regions of the limbs supplied by those nerves distal to the site of injection.
2. Intrasynovial analgesia of joints, tendon sheaths or bursae.
3. Direct local infiltration over suspect superficial lesions.
4. Field analgesia, performed by circular injection around the suspected site of disease, thereby blocking all nerve fibres entering the area.
    - All local analgesics prevent depolarization of the nerve by changing the permeability of the cell membrane to sodium ions (Na^+). The analgesic cation binds to the nerve cell membrane anion. This results in blocking of the Na^+-channels in the cell membrane.
    - The preparations used for nerve blocks in lameness examinations should be intermediate in duration of action (60 to 90 minutes). Lidocaine hydrochloride is commonly used but may produce more soft tissue swelling than mepivacaine which causes minimal tissue reaction and has a slightly longer duration of action.
    - The site of injection should be clean. The hair need not be clipped for nerve blocks or for intrasynovial centesis. Strict adherence to aseptic technique is required for all intrasynovial injections and it is good policy for all regional injections as many of these are close to synovial cavities, and so inadvertent penetration is possible.

For perineural analgesia a minimal volume that will still reliably block the target nerve is deposited as accurately as possible adjacent to it. The more proximal nerve trunks are thicker and more deeply situated within the tissues making accurate placement more difficult. The volume of local anaesthetic solution used is therefore generally greater for more proximal nerve blocks. For proximal blocks requiring use of large needles, it can be helpful to place a small bleb of local anaesthetic subcutaneously first using a fine-gauge needle before positioning the larger needle.

The peripheral nerves generally run in association with an artery and a vein as a neurovascular bundle. If a needle inadvertently enters a blood vessel on initial placement it should be withdrawn slightly and repositioned (usually slightly more caudally).

Distal limb blocks usually take effect within 5 to 10 minutes; because of the increased thickness of proximal nerve trunks, more proximal blocks take longer to become effective and should generally be given at least 20 minutes to act. The efficacy of distal perineural analgesia can be tested to some extent by evaluating skin sensation distal to the block. This is best done using a blunt point such as a ball point pen. The response should be compared to that in the equivalent region of the contralateral limb, assuming that this limb has not been previously blocked.

Perineural analgesia should be carried out in a sequential manner starting distally and working proximally until lameness is eliminated. If a specific joint is suspected to be the source of lameness from the initial physical examination, it is better to perform an intra-articular block of this joint first, rather than a regional block proximal to the joint, because the joint block will not interfere with subsequent more distal regional blocks if the response is negative.

When performing intrasynovial analgesia the most reliable sign that the needle has been accurately placed within the synovial cavity is the appearance of synovial fluid in the hub of the needle. This does not, however, always occur immediately for a number of reasons:

1. Some of the smaller joints and bursae contain only a very small amount of synovial fluid (e.g. the navicular bursa or the distal intertarsal joint).
2. Synovial villi may be sucked into the end of the needle and prevent any synovial fluid escaping through it.

Initial lack of resistance to injection followed by an increase in pressure with injection, illustrated by backflow of injected solution into the syringe when pressure on the plunger is released, also suggests accurate needle placement within a synovial cavity. There is no reliable way of testing whether an intrasynovial block has been effective, and thus it is important to be sure that needle placement is accurate from the outset.

In those cases where a positive response is seen, the time taken to achieve soundness varies from as little as 5 minutes for small distal joints such as the distal interphalangeal joint to up to an hour for large complex joints such as the stifle.

Peripheral nerve blocks of the lower limb

Anatomy

Two distinct systems of innervation have been identified in the lower limbs: the superficial and deep innervation systems.

- The superficial innervation of the forelimb is supplied by the lateral and medial palmar nerves, which course distally between the suspensory ligament (SL) and the DDFT. These nerves form a continuation of the median and ulnar nerves.
- The deep innervation of the forelimbs originates from the lateral palmar nerve (palmar branch of the ulnar nerve) which forms the lateral and medial palmar metacarpal nerves. These nerves course distally between the splint bones, the SL and the metacarpus. Distal to the button of the splint bone these nerves regain a subcutaneous location, turn to the dorsal aspect of the fetlock, and end halfway down the dorsal aspect of the pastern.
- The pattern of distribution of the deep and superficial innervation of the lower hind limb is similar to that of the forelimb, apart from one major difference – the presence of a dorsal deep innervation system that originates from the deep peroneal nerve. The lateral and medial dorsal metatarsal nerves course distally all the way down into the laminar corium of the foot.
- Variations on this pattern of nerve distributions are commonly found in the forelimbs.
    - In 30% of horses, an intermediate branch originates from the dorsal aspect of the palmar digital nerve, and forms the intermediate digital nerve.
    - In up to 50% of horses, the medial palmar metacarpal nerve continues down to the coronary corium.

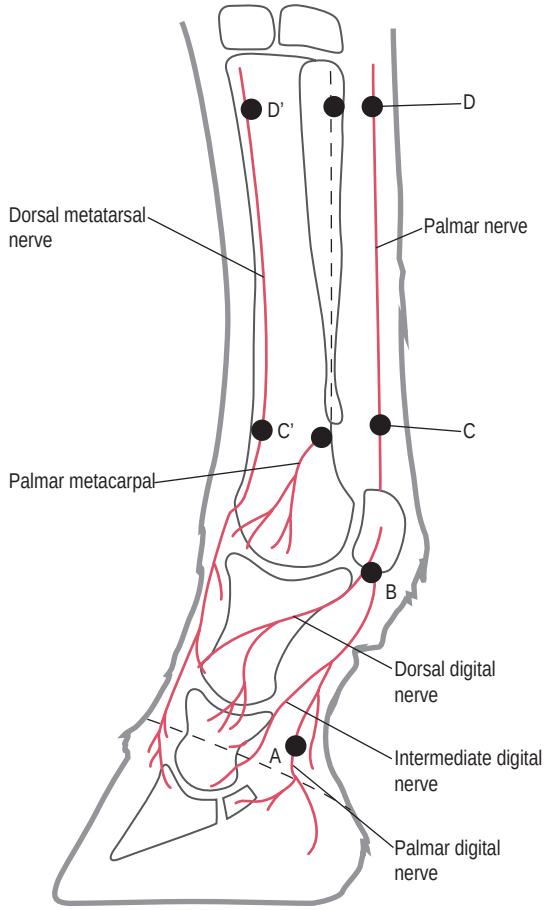


Figure 15.1 Schematic representation of the nerve blocks of the lower limb. A, B, C and D mark the sites for the palmar digital, abaxial sesamoid, low 4-point and high 4-point injections. C′ and D′ mark the additional sites for the low and high 6-point injections in the hind limb.

Perineural analgesia of the distal limb

Palmar/plantar digital nerve block (PDNB) (Figure 15.1)

The Palmar/plantar digital nerves are palpated along the lateral and medial borders of the DDFT, palmar/plantar to the accompanying artery. One to two millilitres of local analgesic solution are injected in the angle between the ungular cartilages of the distal phalanx and the palmar/plantar aspect of the pastern, on the palmar/plantar border of the neurovascular bundle. This results in desensitization of the entire foot including the navicular bone, the navicular bursa, the suspensory ligaments of the navicular bone, the distal part of the DDFT, the digital cushion, the corium of the frog, the solar and laminar dermis, the DIP joint and the distal phalanx. The only part of the foot that is not desensitized by a PDNB is the dorsal aspect of the coronary band and the sensitive laminae of the dorsal wall of the toe. The understanding that the PDNB only desensitizes the palmar/plantar half of the foot is obsolete. Absence of skin sensation over the heels indicates that the block has taken effect.

Abaxial sesamoid nerve block (ASNB) (Figure 15.1)

The dorsal and palmar/plantar digital nerves are part of the neurovascular bundle which is easily palpated over the abaxial surface of the proximal sesamoid bones. The needle is inserted in a distal direction, just palmar/plantar to the neurovascular bundle, by the base of the proximal sesamoid bone, and 2–3 mL of local analgesic solution is injected on both sides. Correct injection leads to desensitization of the entire foot, the DIP joint, the distal sesamoidean ligaments and flexor tendons distal to the site of injection, the proximal interphalangeal (PIP) joint, and often part of the fetlock joint. The skin around the coronary band should be entirely desensitized. A ring block of the pastern may be a useful alternative to the ASNB, because it avoids the sometimes confusing partial desensitization of the fetlock joint that is frequently achieved with an ASNB.

Low 4-point nerve block (low palmar and palmar metacarpal nerve block) (L4-PNB) (Figure 15.1)

The palmar nerves are desensitized by injection of 3 mL of local analgesic solution subcutaneously on either side of the limb, between the SL and DDFT at the level of the distal buttons of the splint bones.

Local analgesic (2 mL) is injected over each palmar metacarpal nerve where these emerge to a subcutaneous location, just distal to the buttons of the splint bones.

This nerve block desensitizes all structures distal to the level of injection, although some skin sensation may still be present over the dorsal aspect of the fetlock due to additional innervation of this area by branches of the cutaneous antebrachii medialis nerve, which is a dorsal branch of the ulnar nerve.

In the hind limb, this block is referred to as the low 6-point nerve block, because some local anaesthetic solution has to be injected further dorsally from the plantar metatarsal nerves to desensitize the dorsal metatarsal nerves.

Significant proximal diffusion of local anaesthetic solution can occur with use of an ABSNB or L4(6)-PNB. Therefore the interpretation of results should include assessment of the time to effect. Improvement may be observed when lameness originates from the proximal metacarpal/metatarsal region especially if more than 15 minutes have elapsed following injection.

High 4-point nerve block (high palmar and palmar metacarpal nerve block) (H4-PNB) (Figure 15.1)

The palmar nerves are desensitised between the SL and the DDFT, deep to the heavy fascia of the flexor retinaculum, 2 to 3 cm distal to the level of the carpometacarpal joint.

The palmar nerves tend to be closely associated with the dorsal surface of the DDFT, although occasionally the lateral palmar nerve is found further palmarly, between the superficial digital flexor tendon (SDFT) and the DDFT. The palmar metacarpal nerves lie between the SL, the palmar surface of the metacarpus and the axial surface of the splint bone.

Since the local anaesthetic solution must be deposited deep to the retinaculum, the skin should not bulge during or after injection. Local anaesthetic (3 mL) is injected over each of the four sites, which should lead to desensitization of all structures distal to the level of injection, apart from the skin over the metacarpus, the most proximal part of the suspensory ligament and the proximal ends of the splint bones.

Proper desensitization is monitored by squeezing the body of the SL as metacarpal skin sensation is usually not completely desensitized by this block.

For reasons mentioned before, this nerve block becomes a 6-point block in the hind limb.

Desensitization of the origin of the suspensory ligament (SL)

To desensitize the most proximal part of the SL in the forelimb, the following techniques can be used:

- 'Subcarpal' block.
  - Infiltration of local analgesic solution only in the region of the palmar metacarpal nerves – i.e. half of the high four-point block.
  - The lateral palmar nerve block:
    performed just below and halfway along the palmar border of the ligament between the accessory carpal bone and the proximal end of the lateral splint bone.
    performed at the medial aspect of the distal third of the accessory carpal bone in the vertical groove close to the palmar border of the bone.
    a 25-gauge, 5/8 inch needle is inserted perpendicular to skin in a mediolateral direction down to its hub, and 3 mL of analgesic solution is injected.
- Lateral palmar nerve block – palmaromedial to accessory carpal bone.
  - In the palmaromedial groove of the accessory carpal bone.
  - 25 G – 5/8 inch – 2 mL of local analgesic solution.
  - No risk of entering the carpal sheath in one published study.
- Direct infiltration from both medial and lateral aspects of the limb over the proximal aspect of the SL with the limb raised.
- The ulnar nerve block (see below).
- In the hind limb the proximal part of the SL is desensitized either by direct infiltration or by desensitizing the deep branch of the lateral plantar nerve axial to the proximal aspect of the lateral splint bone. With the hind limb off the ground, a 21-gauge, 1-inch needle is introduced just distal to the head of the lateral splint bone just off the lateral border of the superficial digital flexor tendon, perpendicular to the long axis and in the sagittal plane of the limb. The needle is introduced down to its hub to a depth of 21-gauge, 1-inch needle, and 3–4 mL of local analgesic solution is injected.

Peripheral nerve blocks of the upper limb

Median and ulnar nerve blocks

Median and ulnar nerve blocks desensitize the carpus and most structures distal to it. As with tibial and peroneal blocks in the hind limb, skin desensitization is not complete distal to the nerve block but only affects certain areas (see below). This makes it advisable to perform these proximal limb blocks on a different occasion to the more distal regional blocks to be able to demonstrate that appropriate skin desensitization has occurred.

Median nerve block

- Needle: 19-gauge, 2-inch (5-cm).
- Volume: 15–20 mL.
- Site: The caudomedial border of the radius just distal to the superficial pectoral muscle. The nerve lies cranial to the median artery and vein. Skin is desensitized only at the medial aspect of the pastern.

Ulnar nerve block

- Needle: 20-gauge, 1-inch (2.5-cm).
- Volume: 15–20 mL.
- Site: In the muscular groove between the ulnaris lateralis and the flexor carpi ulnaris muscles on the caudal aspect of the antebrachium, 10 cm proximal to the accessory carpal bone, at a depth of one to two cm. Skin desensitization occurs on the proximodorsolateral aspect of the metacarpus.

Tibial and peroneal nerve blocks

Tibial and peroneal nerve blocks eliminate deep sensation from the hock and structures distal to it. As with median and ulnar blocks in the forelimb, loss of skin sensation is limited to certain areas and may be inconsistent.

Tibial nerve block

- Needle: 20-gauge, 1-inch (2.5-cm).
- Volume: 20 mL.
- Site: Just caudal to the deep digital flexor tendon and cranial to the common calcaneal tendon about 10 cm proximal to the tuber calcis on the medial aspect of the limb deep to the fascia. Skin sensation is usually lost over the bulbs of the heel.

Peroneal nerve block

- Needle: 19-gauge, 1.5-inch (3.7-cm).
- Volume: 15 mL total (10 mL deep and 5 mL superficially).
- Site: In the muscular groove between the long and lateral digital extensor muscles on the lateral aspect of the crus, 10 cm proximal to the lateral malleolus. The peroneal nerve has deep and superficial branches. Ten ml are injected around the deep branch, about 3–5 cm deep and 5 mL around the subcutaneous superficial branch during withdrawal of the needle. Skin desensitization usually occurs on the proximolateral aspect of the metatarsus.

Intrasynovial analgesia

Analgesia of the navicular bursa

- Needle: 20-gauge, 3.5-inch (9-cm).
- Volume: 3–4 mL.
- Site: The needle is inserted 1 cm proximal to the coronary band on the midline between both heel bulbs, along the sagittal plane of the foot, towards an imaginary point, situated halfway along and just distal to the coronary band. The needle hits the flexor surface of the navicular bone at this point, and 3 to 4 mL of local analgesic solution is injected.

Analgesia of the distal interphalangeal joint

- Needle: 23-gauge, 1-inch (2.5-cm).
- Volume: 6 mL.
- Site: The needle is inserted 1 cm proximal to the coronary band on the dorsal midline, at an angle of 15 to 45° to the horizontal, and advanced through the common digital extensor tendon.
- Extra-articular structures (e.g. the navicular bone) are also desensitized by this injection even though the DIP joint and the navicular bursa do not physically communicate.

Analgesia of the proximal interphalangeal joint (PIP)

- Needle: 20-gauge, 1.5-inch (3.7-cm).
- Volume: 5 mL.
- Site: A needle is introduced through the skin on the lateral aspect of the limb, between the lateral margin of the DDFT and the palmarolateral border of the proximal phalanx and advanced distally in a lateromedial plane towards the palmar border of the middle phalanx until the palmar pouch of the PIP joint is entered.

Anaesthesia of the metacarpo/metatarsophalangeal (fetlock) joint

- Needle: 23- or 21-gauge, 1-inch (2.5-cm).
- Volume: 10 mL.
- Site: Introduce the needle through the collateral sesamoidean ligament, between the palmar surface of the metacarpal condyle and the dorsal articular surface of the proximal sesamoid bone, with the limb flexed.

- Alternative site – dorsally in the weight-bearing limb, to one side of the digital extensor tendons, proximal to the articular margin of the proximal phalanx.

Analgesia of the digital synovial sheath

- Needle: 20-gauge, 1-inch (2.5-cm).
- Volume: 10 mL.
- Site: Synoviocentesis is best performed by introducing the needle into the distal pouch of the sheath, between the bifurcating branches of the SDFT and the lateral or medial margin of the DDFT in the palmar aspect of the pastern.
- It can also be performed proximal to the proximal sesamoid bones, between the DDFT and the SL, close to DDFT. This may be very difficult if synovial effusion is absent.
- Another alternative approach describes the insertion of a 21-gauge, 1-inch needle, through the palmar/plantar annular ligament of the fetlock and along the lateral or medial border of the SDFT with the limb off the ground and in partial flexion.

Analgesia of the middle carpal joint

- Needle: 20-gauge, 1-inch (2.5-cm).
- Volume: 10 mL.
- Site: With the carpus flexed, on the dorsal surface of the joint just lateral to the extensor carpi radialis tendon, between the proximal and distal rows of carpal bones. The midcarpal joint usually communicates with the carpometacarpal joint, which will, therefore, also be desensitized by this procedure.

Analgesia of the antebrachiocarpal joint

- Needle: 20-gauge, 1-inch (2.5-cm).
- Volume: 10 mL.
- Site: With the carpus flexed, on the dorsal surface of the joint just lateral or medial to the extensor carpi radialis tendon, between the distal radius and the proximal row of carpal bones.

Analgesia of the elbow joint

- Needle: 19-gauge, 2-inch (5-cm).
- Volume: 15 mL.
- Site: Just cranial or caudal to the lateral collateral ligament. The joint space can usually be appreciated by careful palpation.

Analgesia of the shoulder joint

- Needle: 19-gauge, 3.5-inch (9-cm) spinal needle.
- Volume: 20 mL.
- Site: Advance the needle between the cranial and caudal prominences of the lateral tuberosity of the humerus, at a 45° angle to the long axis of the horse and slightly proximodistally until the tip of the needle hits bone. Aspirate to assess intra-articular needle placement.

Analgesia of the tarsometatarsal joint

- Needle: 20-gauge, 1-inch (2.5-cm).
- Volume: 5 mL.
- Site: Proximal to the proximal margin of the 4th metatarsal bone, between it and the 4th tarsal bone. Needle angled at 30–45° distally and slightly axially.

Analgesia of the distal intertarsal joint

- Needle: 25-gauge, 5/8-inch (1.6-cm).
- Volume: 5 mL.
- Site: Interosseous space between the 2nd and 3rd tarsal bones and the central tarsal bone on the medial aspect of the hock, just distal to the cunean tendon. Advance the needle in a mediolateral direction in the proximal extent of the interosseus space.

Analgesia of the tarsocrural joint

- Needle: 20-gauge, 1-inch (2.5-cm).
- Volume: 15 mL.
- Site: Into the dorsomedial pouch of the joint, just medial or lateral to the saphenous vein.

Analgesia of the femoropatellar joint

- Needle: 20-gauge, 1.5-inch (3.5-cm).
- Volume: 20 mL.
- Site: Medial or lateral to the middle patellar ligament just distal to the distal border of the patella. The needle is directed caudoproximally. The femoropatellar joint communicates with the medial femorotibial joint in at least 65% of horses.

An alternative approach involves injection into the lateral cul-de-sac of the femoropatellar joint just caudal to the lateral patellar ligament and 5 cm proximal to the lateral condyle of the tibia.

Analgesia of the medial compartment of the femorotibial joint

- Needle: 20-gauge, 1.5-inch (3.5-cm).
- Volume: 20 mL.
- Site: Between the medial patellar ligament and the medial collateral ligament into the medial synovial sac of the joint, just proximal to the proximal border of the medial meniscus.

Analgesia of the lateral compartment of the femorotibial joint

- Needle: 20-gauge, 1.5-inch (3.5-cm).
- Volume: 20 mL.
- Site: Between the tendon of origin of the long digital extensor and the lateral collateral ligament of the femorotibial joint, just proximal to the proximolateral margin of the tibia.

Arthrocentesis and analysis of synovial fluid

Joint fluid is a dialysis product of plasma to which hyaluronic acid (HA) is added by synoviocytes. Its composition is determined by the permeability of the joint membrane. Inflammation of a joint leads to depolymerization of normal HA, and the permeability barrier is disturbed resulting in joint effusion. Changes in joint fluid reflect the degree of synovial membrane inflammation.

1. Gross appearance of fluid.
   - Normal: pale yellow, translucent and free of debris.
   - Trauma: haemorrhagic, dark yellow or amber, with flocculent material.
   - Infection: serofibrinous, fibrinopurulent, turbid, cloudy.
   - Osteoarthritis (OA or DJD): minimal changes.
2. Volume.
   - Trauma: increased.
   - Chronic OA/DJD: increased, or rarely decreased.
   - Infection: increased.
3. Clotting test in plain tube.
   - Normal: no clot formation because lack of fibrinogen.
   - Abnormal: speed of clot formation relates to the degree of inflammation.

4. Total protein.
   - Normal: 18 to 20 g/L.
   - Inflammation: 20–40 g/L.
   - Infection: > 40 g/L.
5. Viscosity.
   - Normal: 5- to 7-cm string from syringe; 2.5- to 5-cm string between fingers.
   - Abnormal: from reduced viscosity to watery.
6. Cytology.
   - WBC count.
     - Normal: 0.2×10^9/L; < 10% neutrophils, some lymphocytes and mononuclear cells.
     - Trauma: $0.5–10 \times 10^9$/L; neutrophils increased.
     - OA/DJD: $0.5–1 \times 10^9$/L.
     - Infection: $>30 \times 10^9$/L; >90% neutrophils.
7. Culture. In approximately 50% of cases of septic synovitis, no organisms are found in synovial fluid because bacteria are sequestrated in the synovial membrane, joint fluid is bactericidal, or antibiotics have been previously administered. A higher culture rate can be obtained by using enrichment broths, anaerobic as well as aerobic cultures, and culturing synovial membrane biopsies as well as synovial fluid samples.
8. pH. The pH is the only synovial parameter in infectious joint disease that remains unaffected by previous administration of intra-articular corticosteroids.
   - Normal: >6.9.
   - Infection: <6.9.
9. Markers of cartilage degeneration. The search for a quantitative marker of OA has led to the attempt to analyse joint fluid for the presence of cartilage particles after filtration of the synovial fluid sample. Although this has proved to be an inconsistent technique, new promising biochemical markers of proteoglycan and collagen breakdown are presently under investigation for clinical use.

Radiography (see Chapter 25)

Following identification of the site of pain during the clinical examination, the next step in a logical sequence is to image the area of interest.

Foot

A complete radiographic examination of the equine foot usually consists of four different views:

- Lateromedial (LM).
- Two dorsoproximal-palmarodistal oblique (DPr-PaDiO) ('upright pedal' or 'high coronary route') (i) centred and exposed for the distal phalanx (ii) centred, collimated, and exposed for the navicular bone.
- Palmaroproximal-palmarodistal oblique (PaPr-PaDiO) ('flexor' or 'skyline' view).

Other oblique views can be useful when fractures of the wings of the distal phalanx are suspected, or abnormalities of the hoof wall are investigated.

Pastern

- Lateromedial (LM).
- Dorsopalmar (DPa).

Oblique views are occasionally obtained to highlight the palmarolateral and palmaromedial borders of the proximal phalanx, onto which the distal sesamoidean ligaments insert.

Fetlock

- Dorsopalmar (DPa).
- Lateromedial (LM).
- Dorsolateral-palmaromedial oblique (DL-PaMO).
- Dorsomedial-palmarolateral oblique (DM-PaLO).

These four standard projections can be supplemented with different specialized views if a particular area of the fetlock requires more highlighting.

Carpus

- Lateromedial (LM).
- Dorsopalmar (DPa).
- Dorsolateral-palmaromedial oblique (DL-PaMO).
- Dorsomedial-palmarolateral oblique (DM-PaLO).
- Flexed lateromedial (flexed LM).
- Dorsoproximal-dorsodistaloblique (skyline) (DPr-DDiO) – taken at varying angles to skyline the distal radius or proximal or distal rows of carpal bones.

Elbow

- Flexed mediolateral (flexed ML).
- Craniocaudal (CrCd).

Shoulder

- Extended mediolateral (extended ML).
- Craniomedial-caudolateral oblique (CrM-CdLO).

Hock

- Lateromedial (LM).
- Dorsoplantar (DPl).
- Dorsolateral-plantaromedial oblique (DL-PlMO).
- Dorsomedial-palmarolateral oblique (DM-PaLO) or
- Plantarolateral-dorsomedial oblique (PlL-DMO).

Additional view: Plantaroproximal-plantarodistal oblique (PlPr-PlDiO) – 'skyline' of the sustentaculum tali.

Stifle

- Lateromedial (LM).
- Caudocranial (CdCr).

Additional views:

- Craniproximal-craniodistal oblique (CrPr-CrDiO) – 'skyline' of the patella.
- Oblique variations on the CdCr or LM.

Hip

- Ventrodorsal.
- Standing oblique latero-lateral.

Ultrasonography (see Chapter 25)

The most commonly used equipment for examining equine tendons and joints is a 7.5- to 10-MHz sector or linear array probe.

Technique

Ultrasonographic examination of tendons and ligaments in the distal fore- and hind limbs must follow a methodical approach that allows

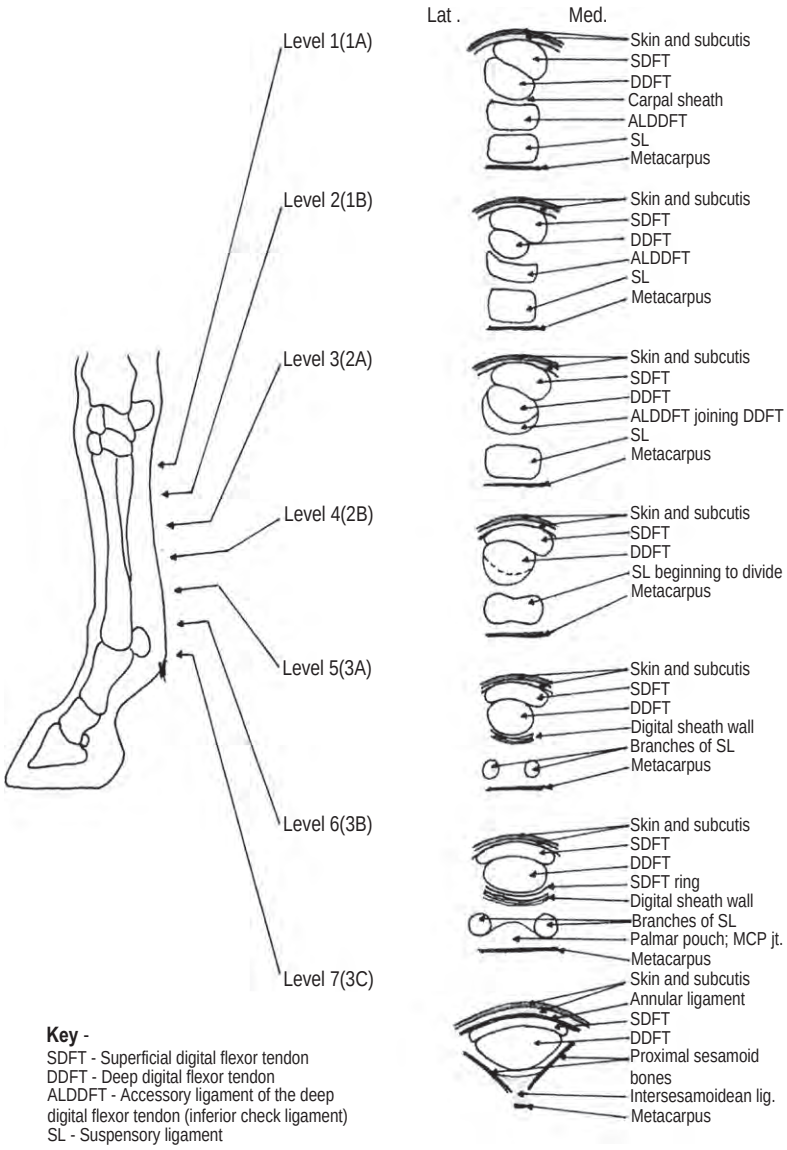


Figure 15.2 Ultrasonographic appearance of the soft tissues on the palmar aspect of the distal limb (Courtesy of Smith RK, Webbon PM 1994 In: Hodgson DR, Rose RK (eds) The athletic horse, principles and practice of equine sports medicine. W.B. Saunders Co, Philadelphia, p 300–302. *With permission from Elsevier Ltd and Professor D.R. Hodgson.*). SDF = superficial digital flexor; DDF = deep digital flexor; SL = suspensory ligament; ALDDFT = accessory ligament of the deep digital flexor tendon (inferior check ligament).

comparative views to be taken for contralateral comparison and for follow-up examination.

Metacarpal region (Figure 15.2)

The probe is applied to the palmar aspect of the metacarpus in a horizontal (transverse) fashion. Seven equidistant levels are chosen between the accessory carpal bone and the ergot of the fetlock (levels 1 to 7). This allows sequential identification of the SDFT, the DDFT, the accessory ligament of the DDFT (inferior check ligament), the SL and its branches, and the annular ligament of the fetlock.

Three longitudinal views (levels 1 to 3) are then taken with the probe in line with the tendons.

Pastern region (Figure 15.3)

The probe is applied to the palmar/plantar aspect of the pastern and three transverse views are assessed (levels 1 to 3). Identifiable structures are the proximal digital annular ligament (if enlarged), the branches of the SDFT, the DDFT and the distal sesamoidean ligaments (oblique and straight). One longitudinal view (PL) is also usually assessed.

Interpretation

Significant alterations in the ultrasonograms of tendons include:

1. Changes in echogenicity:
   - Hypoechoic/anechoic areas ('black holes') in acute tendon injuries represent the presence of fluid, granulation tissue, or young fibrous tissue (in order of increasing echogenicity).
   - Hyperechoic ('brighter') areas in a healed tendon injury represent fibrosis.
   - Calcification may cause acoustic shadowing. The calcified area reflects almost all of the sound.
2. Changes in cross-sectional size of tendons/ligaments. The size should be compared to that of the same structures in the unaffected leg.

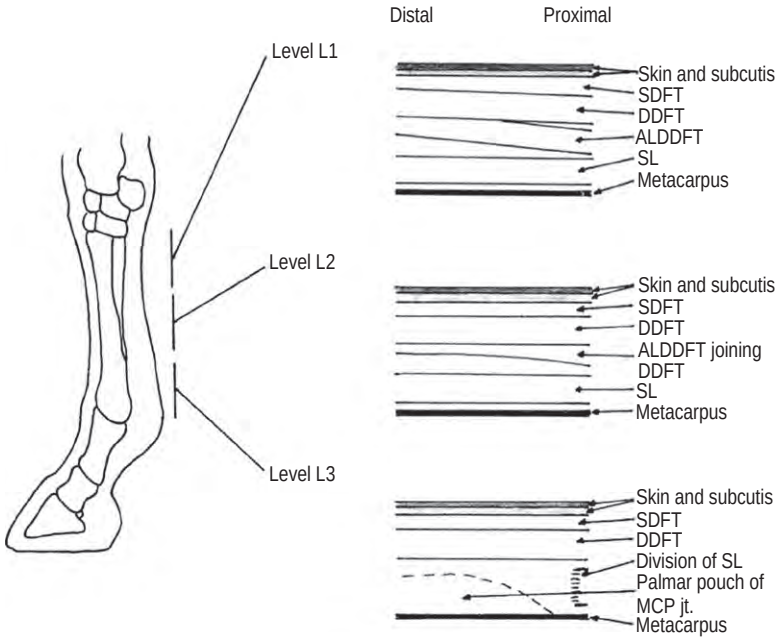


Figure 15.3 Ultrasonographic appearance of the soft tissues on the palmar/plantar aspect of the pastern (Courtesy of Smith RK, Webbon PM 1994. In: Hodgson DR, Rose RJ (eds) The athletic horse, principles and practice of equine sports medicine. W.B. Saunders Co, Philadelphia, p 300–302. *With permission from Elsevier Ltd and Professor D.R. Hodgson.*). SDF = superficial digital flexor; DDF = deep digital flexor.

Key -
SDFT - Superficial digital flexor tendon
DDFT - Deep digital flexor tendon
ALDDFT - Accessory ligament of the deep digital flexor tendon (Inferior check ligament)
SL - Suspensory ligament
MCP jt. - Metacarpophalangeal joint

3. Changes in shape/position.
4. Changes in margination/outline which may indicate the formation of peritendinous adhesions.

Indications for the use of ultrasonography

1. Diagnosis. Ultrasonography of swollen palmar metacarpal soft tissues allows identification of the structure(s) involved and enables more accurate assessment of the severity of the damage.
2. Prevention of more severe injury. Ultrasonography allows an accurate assessment of the subtle signs that are harbingers of severe injury. If these are recognized the horse can be withdrawn from strenuous exercise and treated before the tendon fails.
3. Ultrasonographic monitoring of healing is useful in the management of tendon injuries.
4. Ultrasonography is occasionally used in the assessment of joint disease.

Nuclear scintigraphy (see Chapter 25)

The use of bone scintigraphy in the horse lies mainly in the recognition of stress injuries to the skeleton (i.e. fatigue fractures) and of acute or chronic entheseous or articular sites of bone remodelling (e.g. sacroiliac region).

Thermography

Thermography is a non-invasive diagnostic technique that allows quantitative visualization of changes in skin surface temperature. The infrared radiation that is emitted from the skin is converted into electrical impulses, which are converted into computer images. The major diagnostic value of thermography is its ability to detect early soft-tissue inflammation (e.g. early tendinitis) before clinical signs develop although false positives and false negatives are common.

Computed tomography (CT) (see Chapter 25)

CT is a cross-sectional imaging method created by multiple X-ray beams. As contrast in a CT image is determined by the variable attenuation of the X-ray beam by tissues of different density, CT scans are considerably quicker to obtain and easier to interpret than MR images. CT is ideally suited for cross-sectional analysis of complicated bone disease. It requires general anaesthesia, but the resolution of cartilage, soft-tissue contrast and detection of medullary fluid is inferior to MRI.

Magnetic resonance imaging (MRI)

(see Chapter 25)

MRI has high intrinsic contrast and resolution, resulting in good anatomic separation between different tissues (tendon, ligament, cartilage, bone, joint fluid). In particular the superior soft-tissue contrast and anatomical detail have high diagnostic value. Besides supplying anatomic information, MRI also displays information that is primarily physiological or pathophysiological. As a cross-sectional imaging modality, MRI is able to scan an object in any plane.

Diagnostic arthroscopy

Arthroscopy allows direct inspection of synovial cavities including joints and tendon sheaths. It allows evaluation of the non-osseous tissues of the joint, i.e. cartilage, synovial membrane and intra-articular ligaments and menisci, all of which may be difficult to assess by other means.

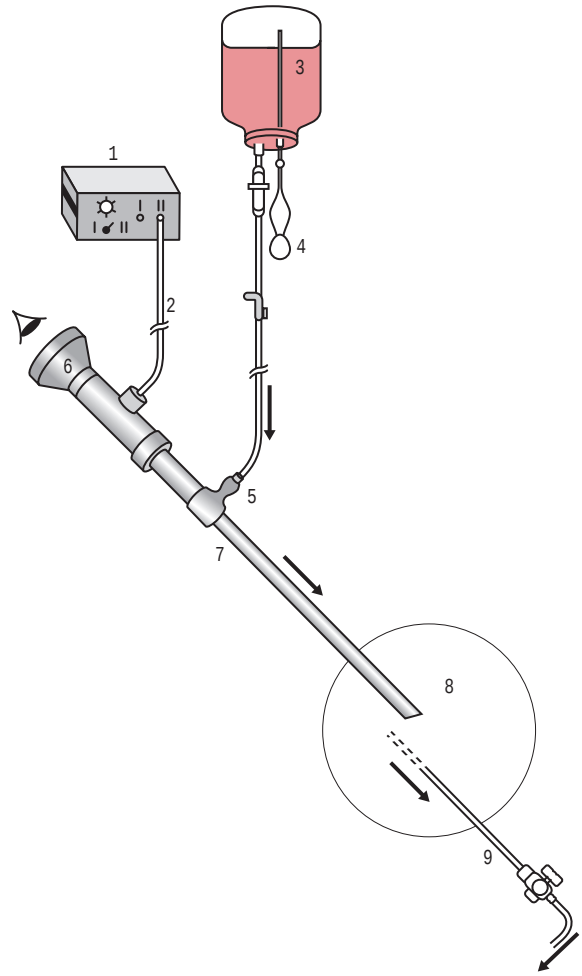


Figure 15.4 Basic set-up for diagnostic arthroscopy. 1. Light source. 2. Light transmitting cable. 3. Fluid source. 4. Pressure bulb. 5. Inflow stop cock. 6. Telescope (4 mm). 7. Telescope sleeve (5 mm). 8. Distended joint. 9. Drainage cannula.

- Equipment most frequently consists of a 4-mm wide telescope with a 25° forward angle of view. The telescope is connected to a fibreoptic light cable. This scope is inserted into the joint through a 5-mm arthroscopic sleeve. This sleeve has a connection for fluid inflow into the joint.
- The technique (Figure 15.4) relies on distension of the joint with sterile balanced electrolyte solution. Once distended, it becomes possible to push the narrow sleeve through a stab incision in the tense joint capsule with a conical trocar with little risk of damaging the intra-articular structures (i.e. cartilage). Only a small stab incision in the skin is needed, which is later closed with a single suture. After the position of the sleeve is ascertained inside the joint, the trocar is exchanged for the telescope. Inflow of fluid is necessary to maintain joint distension and visualization during the procedure.
- Applications in the horse are numerous. All equine joints, tendon sheaths and bursae are fully or partially amenable to arthroscopic examination.
- Indications for diagnostic arthroscopy in the horse include persistent intra-articular pain and/or abnormalities seen on radiographs. The technique is now widely regarded as the preferred method for joint surgery.

15.2 JOINT DISEASES

Idiopathic synovitis

Definition

Chronic synovial effusion of a joint or tendon sheath of uncertain pathogenesis and often not associated with pain or lameness. Common examples include articular and tendinous 'windgalls' of the fetlock (distension of the metacarpo/metatarsophalangeal joint or digital synovial sheath respectively) and 'bog spavin' in the hock (distension of the tarsocrural joint).

Aetiopathogenesis

Presumed to be low-grade synovitis as a result of minor trauma or abnormal stresses due to poor conformation. Idiopathic synovitides need to be differentiated from synovitis caused by a primary injury which may not be immediately apparent but which requires detailed imaging. May be self-perpetuating once present, due to stretching of the synovial membrane and joint capsule. Usually no radiographic abnormalities are present.

Treatment

- Treatment is usually not necessary if there is no functional disturbance or evidence of an underlying problem (such as OCD in young horses).
- In young horses the swellings may spontaneously resolve as the horse reaches maturity.
- Drainage of the synovial fluid is possible but must be done under aseptic conditions; rapid refilling of the synovial cavity with excess fluid is common.
- Prevention of recurrence with bandaging or injection of corticosteroids or hyaluronic acid can sometimes be successful.

Traumatic arthritis/synovitis

Aetiology

- Single-event trauma.
- Chronic repetitive trauma.

Pathogenesis

See osteoarthritis – the dividing line between traumatic arthritis and osteoarthritis is really one of degree, chronicity and reversibility, i.e. if the disease becomes persistent and progressive it should be referred to as osteoarthritis.

Pathology

The following pathological conditions may be present individually or in varying combinations.

- Synovitis.
- Capsulitis.
- Articular cartilage damage.
- Articular fracture.
- Ligamentous injury.
- Luxation/subluxation.
- Subchondral bone haemorrhage, oedema, sclerosis or lysis.

Clinical signs

- Lameness.
- Joint swelling.
- Synovial effusion.
- Periarticular soft-tissue swelling.
- Pain on joint manipulation – particularly forced flexion.

- Heat – increased surface temperature over joint.
- Instability/crepitus – if severe ligament or bone damage is present.
- Chronic thickening and reduced range of motion in later stages.

Further tests

- Radiography (see Chapter 25).
  - For fractures.
  - Immediately and 7–10 days later if results inconclusive.
  - For evidence of pre-existing or secondary osteoarthritis.
- MRI.
  - In particular for detection of bone oedema, sclerosis or cartilage erosions.
- Synovial fluid analysis.
  - In particular for the detection of sepsis.
- White blood cell total and differential.
  - Total protein.
- Diagnostic arthroscopy – for synovitis, cartilage damage, intra-articular fractures and ligamentous injury.

Treatment

- Box rest and later a progressive programme of controlled exercise.
- Cold application locally over first 24–48 hours.
- Support bandaging.
- Non-steroidal anti-inflammatory drugs (NSAIDs).
- Single dose of short-acting corticosteroid in selected cases of synovitis/capsulitis.
- Polysulphated glycosaminoglycans.
- Hyaluronic acid.
- Joint lavage.
- Specific surgery for primary disease such as fractures etc.

Osteoarthritis (degenerative joint disease)

Definition

Progressive destruction of articular cartilage accompanied by changes in bones (marginal osteophytes, subchondral sclerosis or lysis) and soft tissues (capsular fibrosis, villous hypertrophy) of a joint.

Classification

Equine osteoarthritis (OA) has been classified in a variety of ways.

1. Primary OA.
   - Probably due to chronic repetitive trauma in most cases.
   - Risk factors may be inherent e.g. breed, age, sex, conformation; or external, e.g. occupation.
   - Systemic predisposition may relate to properties of the tissues involved, e.g. cartilage and bone; while expression, site and severity may be determined by use (i.e. local biomechanical factors).
2. Secondary OA – the consequence of other joint diseases including:
   - Traumatic arthritis.
   - Articular fracture.
   - Osteochondrosis.
   - Septic arthritis.

Pathogenesis

The pathogenesis of OA involves both *mechanical* disruption of the articular surface and the destructive effects of a number of *chemical mediators*.

Chemical mediators of OA:

1. Cytokines, e.g. interleukin 1 (IL-1), tumour necrosis factor alpha (TNFα).
   - May act as messengers from synoviocytes or leucocytes to activate chondrocytes to produce destructive enzymes.
   - Reduce proteoglycan synthesis by chondrocytes.
2. Enzymes.
   - Extracellular neutral metalloproteinases, e.g. proteoglycanase (stromelysin).
   - Collagenase.
   - Gelatinases.
   - Lysosomal enzymes – cathepsins, glycosidases etc.
3. Inflammatory mediators.
   - Eicosanoids (prostaglandins, thromboxane).
   - Leukotrienes.
   - Kinins.
   - Histamine, 5HT.
   - Nitric oxide.
4. Free radicals.
   - Superoxide.
   - Hydroxyl.
5. Antibodies – complement factors.

The initial result of these mechanical and chemical insults is breakdown and loss of proteoglycans from the articular cartilage. The cartilage thereby loses its stiffness and becomes soft, which predisposes it to further mechanical disruption. It also increases its permeability to destructive enzymes and impairs chondrocyte nutrition. Proteoglycan loss *per se* is probably reversible provided the chondrocytes and collagen framework remain intact. Collagen disruption is seen only at a later stage accompanied by gross evidence of fibrillation.

The causes of pain in OA are unclear, and possibly include raised intramedullary bone pressure and synovitis.

Osteophytes form at the marginal transition zone, i.e. at the junction of articular cartilage, synovial membrane and periosteum. They are probably produced by mesenchymal cells in response to substances released in inflamed or degenerate joints. Osteophytes may represent an attempt by the joint to extend its surface area to reduce concussion and improve stability but at the expense of loss of range of movement.

Pathology

1. Cartilage:
   - Gross:
     Yellow discoloration.
     Softening.
     Dullness.
     Surface fibrillation.
     Erosion and ulceration.
   - Histological:
     Chondrocyte death.
     Fibrillation.
     Reduced metachromasia.
   - Biochemical:
     Proteoglycan depletion.
     Increased water content.
     Collagen disruption.
2. Soft-tissue:
   - Congestion and thickening.
   - Mononuclear infiltration.
   - Villous hypertrophy.
   - Capsular oedema and later fibrosis.
3. Bone:
   - Marginal osteophytes and remodelling.
   - Subchondral sclerosis or lysis.
   - Articular fractures.
   - Increased subchondral bone pressure.

- Enthesiophytes associated with inflammation and disruption of the fibrous joint capsule and collateral ligaments.

Although articular cartilage changes are the primary pathological characteristic of OA they are poorly correlated to the severity of clinical signs or radiographic changes. As in other systems and diseases the pathological changes seen are a combination of the effects of the primary insult and the body's reactions and attempts at repair.

Clinical signs

- Lameness.
- Pain on manipulation.
- Positive response to joint flexion.
- Thickening.
- Reduced range of movement.
- Crepitus.

Note – there are frequently few localizing clinical signs in chronic OA in horses, and local analgesia to eliminate lameness is often the only way to identify the joint(s) concerned. Scintigraphy may also be of use in this regard.

Diagnosis

Imaging techniques.

Radiology (see Chapter 25)

- Periarticular new bone formation (osteophytes and enthesiophytes; periosteal new bone in very severe cases such as a consequence to chronic sepsis).
- Subchondral sclerosis/lysis.
- Narrowed joint space (only in severe cases).
- Radiological changes occur relatively late and may correlate poorly with clinical signs.

Scintigraphy

- Increased nucleotide uptake may predict radiographic changes.

MRI

- Subchondral bone abnormalities.
- Cartilage abnormalities.
  - The sensitivity of MRI for detection of periarticular osteophytes may be inferior to radiography.

Arthroscopy (Figure 15.5)

- Cartilage softening, fibrillation and loss.
- Osteophytosis.
- Accompanying subchondral bone abnormalities.

Treatment

Treatment of established OA is often unrewarding in terms of sustained return to athletic activity.

- Controlled light exercise/swimming etc.
- Correct overweight and any foot imbalance.
- Intra-articular medication:
  - Corticosteroids (palliative only).
    PSGAGs.
    Hyaluronic acid.
    Interleukin-1 receptor antagonist protein (IRAP®).
- NSAIDs.
- Surgical arthrodesis of low-motion joints, e.g. small intertarsal and pastern joints.

Osteochondritis dissecans (OCD)

Osteochondrosis involves defective endochondral ossification leading to failure of conversion of cartilage into bone. In dogs and horses the condition particularly affects the articular surface resulting in a specific manifestation of osteochondrosis referred to as osteochondritis dissecans (OCD). As the articular surface cartilage fails to ossify in its deepest layers, it becomes thicker than normal, and the chondrocytes become further removed from their source of nutrition – the synovial fluid. Eventually they die. The deep layers of cartilage then undergo necrosis, and fissures form, which may then extend to the articular surface to form a cartilage flap. The flap may undergo partial ossification to form an osteochondral fragment.

Osteochondritis dissecans is one of a number of conditions (including osseous cyst-like lesions, angular and flexural limb deformities, physitis and the 'wobbler' syndrome) that have been grouped together under the heading of 'developmental orthopaedic disease' because osteochondrosis potentially plays a role in the aetiopathogenesis of all of them. Because the initial lesion of 'osteochondrosis' occurs in

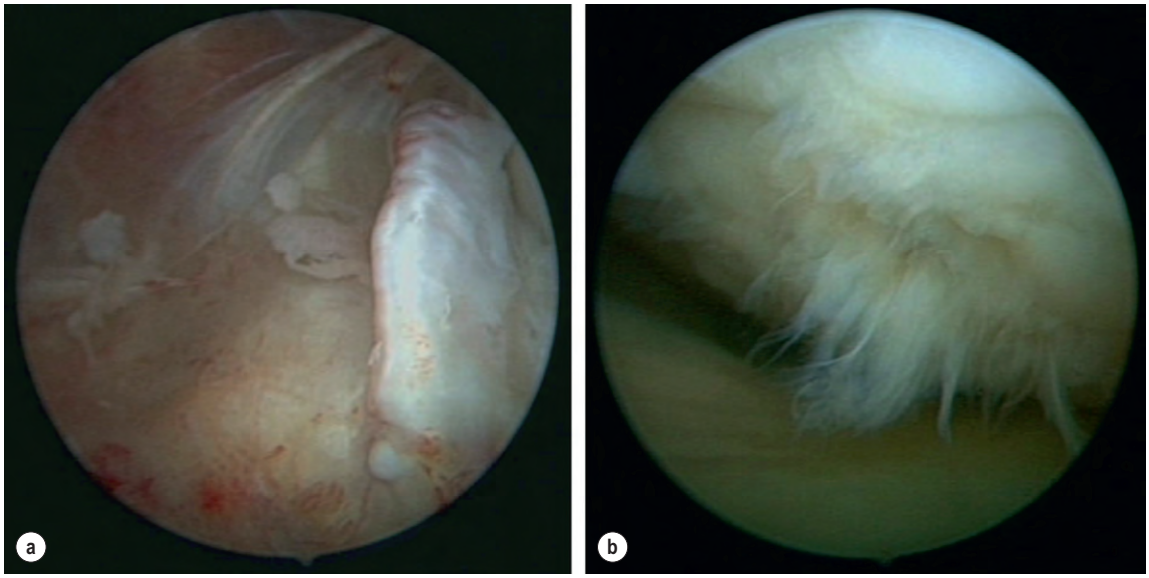


Figure 15.5 Arthroscopic appearance of osteophytosis on the medial intercondylar eminence of the tibia (a) and cartilage fibrillation on the medial femoral condyle (b) of a horse with osteoarthritis of the medial compartment of the femorotibial joint.

cartilage rather than bone the term 'dyschondroplasia' has been suggested as a potentially better name.

The aetiology of osteochondrosis is poorly understood and probably multifactorial. Important factors probably include rapid growth, high dietary energy levels and mineral imbalances, endocrinological dysfunction, biomechanical trauma and heredity.

1. Rapid growth rate – particularly spurts of growth which may follow a growth setback, for example a setback in growth caused by a period of poor nutrition. Growth rate is, in turn, determined by:
   - Genetic factors.
   - Intensive feeding (especially excess soluble carbohydrates).
2. Mineral imbalance.
   - High phosphorus diets.
   - Copper deficiency.
   - Possibly high zinc or cadmium diets.
3. Mechanical trauma.
   - May account for site specificity within joints.
   - May cause separation of already defective cartilage (pathological fracture).
   - Excessive weight increases trauma from use.
   - Conversely exercise may have a protective effect against osteochondrosis in the face of high-energy diets.
4. Hormonal factors.
   - Growth hormone, insulin, thyroid hormone, sex hormones.
   - High postprandial insulin concentrations may inhibit growth hormone which is necessary for cartilage maturation.
5. Heredity.
   - There are reported increases in incidence in the offspring of some stallions. However, clinically and radiologically normal stallions may produce affected foals making the implementation of screening programmes difficult.

Epidemiology

Clinical signs usually arise from birth to 2–3 years of age. Sometimes however the condition may not become clinically obvious until affected horses are put into work.

The epiphyseal osseous development is not complete until approximately 9–10 months of age. In younger horses therefore, radiographic abnormalities consistent with osteochondrosis may still resolve. This has been shown for lesions affecting the tarsocrural joint which may resolve until the horse is 5–6 months old. For lesions of the femoropatellar joint, resolution of lesions may be possible until the horse is 8–9 months old.

Most case series show a predominance of male horses (2:1 male:female).

There are breed variations in overall and individual joint incidence, e.g. OCD of the distal intermediate ridge of the tibia in the hock seems to be most common in Standardbreds. Ponies seem to have a low incidence of osteochondrosis.

Clinical signs

- Lameness, stiffness, positive response to flexion.
- Joint effusion is common especially in the stifle and the hock.

Diagnosis

- Radiography (see Chapter 25).
  - Subchondral defects with or without adjacent (or displaced) osseous fragments (see Figure 15.6).
- Arthroscopy.

Treatment

Both conservative and surgical methods of treatment are used. Conservative treatment consists primarily of rest and correcting dietary input to achieve even and appropriate growth rates. If no free floating, separate osteochondral fragments are present, conservative treatment may be given preference over surgery until joint development is complete (approx 9–10 months of age).

Figure 15.6 Caudolateral-craniomedial oblique radiograph of the stifle of a horse with osteochondrosis affecting the lateral trochlear ridge. Note the defect and the adjacent osseous fragments.

Surgery comprises debridement and removal of separated or partially attached osteochondral fragments and abnormal subchondral bone. This is usually done under arthroscopic visualization.

Subchondral bone cysts (osseous cyst-like lesions)

Radiographically detectable cystic lesions in the subchondral bone which often communicate with the articular surface have been reported in a number of joints including the medial compartment of the femorotibial joint, carpus, pastern, coffin, fetlock, elbow, shoulder and hock.

Aetiopathogenesis

There are two possible causes for the development of subchondral bone cysts.

1. They may be a manifestation of osteochondrosis with thickened necrotic cartilage persisting as a localized defect within the subchondral bone.
2. They may result from localized trauma damaging primarily either:
   - The articular cartilage, or
   - The underlying subchondral bone.

Clinical signs

Lameness localisable to a joint cavity – often few other signs.

Diagnosis

Radiography, CT, MRI (see Chapter 25).

Treatment

Both conservative (rest) and surgical treatments have been described.

Surgery usually consists of corticosteroid injection of the cyst via an articular (arthroscopic) or extra-articular approach depending on the joint involved. Alternatively the cyst cavity can be debrided. Additional surgical procedures such as forage of the bone surrounding the cyst or packing the cyst with an autogenic cancellous bone graft have been reported but are now infrequently used due to their limited success. More recently cartilage resurfacing techniques using autogenic chondrocyte or stem cell implantation or autogenic bone-cartilage transplants have been reported.

Septic arthritis

Aetiology

Organisms involved include:

- Coliforms.
- *Staphylococcus aureus.*
- *Streptococcus.*

In foals *Salmonella*, *Rhodococcus equi* and *Actinobacillus* are also found.

Pathogenesis

In adult horses infection is usually the result of a penetrating wound involving the joint or extension from a site of local infection close to a joint. Kick wounds adjacent to the hock or elbow are a common cause. Infection may be iatrogenic following arthrocentesis or surgery.

In foals infection may spread to joints via the haematogenous route, and multiple joints may be involved. The initial source of infection may be the umbilicus, pneumonia or enteritis. Failure of passive transfer of immunoglobulins may predispose. The same organisms may also colonize metaphyseal or epiphyseal bone leading to osteomyelitis.

Articular cartilage is rapidly destroyed by enzymes from bacteria, white blood cells and inflamed synovial membrane. These cause proteoglycan and collagen breakdown. Cartilage nutrition is compromised by extensive fibrin clots that form in the joint.

Clinical signs

- Lameness – often severe especially in adult horses.
- Joint swelling, heat and pain.
- May be an accompanying regional cellulitis that masks the specific joint effusion.
- Variable systemic signs of pyrexia, leucocytosis, neutrophilia etc. – not always present.

Diagnosis

1. Synovial fluid analysis.
   - Increased volume, turbid, clots, low viscosity.
   - Increased WBC count >30 × 10^9/L; >90% neutrophils.
   - Protein >40 g/L.
   - Culture – into blood culture medium initially.
2. Radiography (see Chapter 25).
   - Soft-tissue swelling.
   - Joint space narrowing (in chronic cases).
   - Subchondral bone destruction and periarticular new bone (in adult horses these changes occur late and the joint is usually beyond redemption by this stage).
   - Osteomyelitis of the epiphysis or metaphysis accompanying septic arthritis is quite common in foals.

Treatment

1. Drainage and lavage of the joint, with several litres of sterile polyionic fluid (e.g. Hartmann's solution) during arthroscopic evaluation. The use of continued drainage via implanted suction drains or open arthrotomies remains controversial but has been reported to be successful. It may be particularly applicable to chronic or recurrent cases. It may be combined with repeated lavage via a tube drain or separate arthrocentesis puncture.
2. Partial synovectomy and removal of fibrin clots. Both of these can be done arthroscopically in many joints – synovial membrane biopsies can also be obtained for culture at the same time. These procedures may need to be repeated if the response to initial treatment is unsatisfactory.
3. Prolonged intensive antibiotic therapy preferably based on culture and sensitivity. Initial therapy with penicillin/gentamicin is often used until results become available. Antibiotic administration may be performed via systemic, regional intravenous, intraosseous and direct intrasynovial routes via repeat arthrocentesis or a constant-rate infusion catheter to maximize intra-articular and intraosseous concentrations.
4. Analgesia with NSAIDs, e.g. flunixin meglumine or phenylbutazone.

Treatment should be monitored in terms of improvement in clinical signs, i.e. reduction in lameness, joint swelling, heat, pain on palpation/manipulation and reduction in discharge where appropriate. Sequential synovial fluid sampling may also be helpful in determining whether the inflammatory response is diminishing due to elimination of the infection.

Prognosis

The prognosis for established synovial infection is usually regarded as guarded. If treated *early* and *aggressively* the results can be rewarding particularly in adult horses where survival of up to 85% of cases has been recorded. The prognosis in foals is generally less good.

15.3 FRACTURES

Classification

Fractures may be classified in a variety of ways with reference to the characteristics listed below. This allows a concise description which has relevance in relation to management and prognosis.

1. Direction and anatomical location of fracture line.
   - Transverse.
   - Oblique.
   - Longitudinal.
   - Spiral.
   - Comminuted – three or more major fragments present.
   - Multiple – more than one bone fractured.
   - Diaphyseal.
   - Metaphyseal.
   - Physeal/epiphyseal (Salter-Harris subclassification).
   - Articular – involving a joint surface.
2. Relative displacement of fragments.
   - Overriding.
   - Distracted.
   - Impacted.
   - Compression – usually applied to vertebral fractures.
   - Folding.
   - Depression – usually with reference to skull fractures.
3. Complete/incomplete.
4. Open (compound)/closed.
5. Stable/unstable.
6. Monotonic/fatigue/pathological.
7. Complicated – involving damage to other organs or systems.

Clinical signs

- Pain (and therefore lameness).
- Swelling.
- Deformity.
- Abnormal mobility (unstable).
- Loss of function (non-weight-bearing).
- Crepitus.

It is important to note that incomplete undisplaced fractures, which are relatively common in the horse compared to other domestic species, will not cause evidence of deformity, instability or crepitus and that in some situations swelling may also be minimal. In these cases signs are therefore confined to lameness. If lameness is acute in

onset and severe, the possibility of a fracture should not be dismissed merely because the other signs are not present.

Fracture healing

Secondary bone union – healing by callus

1. Inflammatory phase.
   - Vascular damage results in haemorrhage and haematoma formation.
   - Osteocytes at the bone ends are deprived of nutrition and die.
   - An acute inflammatory response ensues.
     - Fibrin plug.
     - Platelet adherence.
     - White blood cells phagocytose debris.
2. Repair phase.
   - Haematoma organizes as granulation tissue and serves as a scaffold for repair.
   - Pluripotential mesenchymal cells found perivascularly and from the periosteum, endosteum and muscle differentiate to form:
     - Fibrous tissue.
     - Cartilage.
     - Bone.
   - Cartilage progresses to form bone by endochondral ossification.
   - Reparative phase ends when the callus forms a rigid functional union.
3. Remodelling.
   - Resorption and replacement of bone to restore normal architecture.

Primary bone healing

No fibrous tissue or cartilage is formed – repair is directly to bone which occurs when the gap between the bone ends is less than ~ 1mm and movement at the fracture ends is minimal.

1. Gap healing – newly formed bone lamellae are at right angles to parent bone and remodelling with aligned osteones occurs later.
2. Contact healing – primary osteonal repair across the fracture site.

Complications of fracture healing

1. Infection – particularly following open reduction and internal fixation.
2. 'Fracture disease'.
   - Muscle atrophy.
   - Soft-tissue adhesions.
   - Joint stiffness.
   - Osteoporosis.
3. Delayed union/malunion/non-union.
4. Laxity of the supporting soft tissues in immobilized limbs in foals.
5. Growth disturbance – in young horses – angular limb deformities in the contralateral limb due to excessive weight-bearing.
6. Laminitis of the contralateral foot in adult horses.

Evaluation of equine fractures

Equine fractures vary in their severity and implications and range from catastrophic comminuted proximal limb bone fractures, for which the only recourse may be euthanasia, to small chip fractures that can be removed during routine, elective surgery and the removal of which provides a good prognosis for return to full athletic activity. Certain fractures appear to be occupational hazards, particularly in the racehorse, and may be a consequence of pre-existing physiological or pathological processes that predispose to failure (stress or fatigue fractures). These fractures often have a predictable location and configuration, and examples include third carpal slab fractures and sagittal fractures of the proximal phalanx. Direct external trauma is another common cause of fractures (traumatic fractures). In small animals, road traffic accidents are by far the commonest cause of such injuries. However, in horses, falls, collisions with fences and kicks from other horses account for the majority of traumatic fractures.

Before embarking on treatment it is important to have a clear idea of the likely prognosis in terms of the intended use of the horse, the likelihood of achieving that endpoint and whether fallback positions are acceptable. This requires a thorough evaluation of the fracture, the patient and also of the owner's aspirations. The financial implications of major surgical intervention and the attendant costs in aftercare in the short- and long-term also need to be discussed at an early stage.

Initial evaluation should attempt to answer the following questions:

1. Is a fracture present?
2. Which bone is fractured?
3. What is the likely configuration of the fracture? Try to evaluate the degree and direction of instability if this is present. Knowledge of the common fracture configurations in specific locations is useful.
4. Is the location and likely configuration of the fracture such that the prognosis is definitely hopeless, and euthanasia should be performed without delay?
5. Are there any additional injuries that need to be taken into account – in particular are there associated wounds that might, or definitely have, resulted in contamination of the fracture site?

If treatment is still a possibility then priority should be given to *emergency stabilization* of the fracture followed by *comprehensive radiographic examination* to allow full evaluation.

Emergency stabilization

Objectives

1. Minimize additional damage to:
   - Soft tissues – skin, blood vessels, nerves. It is essential to preserve soft-tissue cover to prevent contamination and preserve potential blood supply to the fracture site.
   - Bone ends – movement between fracture ends causes bone eburnation which makes accurate reduction more difficult at surgery.
2. Allow horse to regain control of limb thereby reducing tendency to panic.
3. Provide a degree of pain relief.
4. Limit soft-tissue swelling.

If skin penetration has occurred, clean the wound and cover with water soluble antibiotic ointment and a sterile dressing before immobilizing.

Forelimb

1. *Phalanges and distal metacarpus*. Stable (e.g. incomplete) fractures are probably best immobilized in a normal weight-bearing position. Unstable fractures tend to bend at the fracture site rather than the fetlock and so should be stabilized with the fetlock straight by splinting along the dorsal surface of the limb from carpus to toe so that the dorsal cortices of the bones are in a straight line. This ensures axial loading and negates the tendency to bending at the fracture site. A splint should be

Figure 15.7 A Kimzey splint applied to the distal limb of a horse with a suspected proximal phalanx fracture.

taped to the dorsal surface of the limb over a light bandage, and the limb cast or substantially bandaged to just below the carpus. Commercial splints (eg Kimzey splints) provide a simple and effective option to achieve the same end result (see Figure 15.7).

2. *Metacarpus, carpus and distal radius*. Splints extending from elbow to ground caudally and laterally over a substantial Robert Jones bandage.
3. *Mid and proximal radius*. As above but extend lateral splint up side of chest to prevent abduction of the distal limb and consequent penetration of the skin by bone fragments on the medial aspect of the radius (where there is little protective muscle).
4. *Elbow and proximally*. In the proximal limb the bones are generally well covered with muscle and splinted by the body wall. Attempts at additional splinting are often ineffective and possibly counterproductive by increasing the weight (and thus pendulum-like effect) of the distal limb. Lightly splinting the carpus in extension (straight alignment) has been suggested to help by enabling the horse to use the leg for balancing.

Hind limb

1. Distal metatarsus and phalanges. As for forelimb.
2. Mid and proximal metatarsus. Caudal and lateral splints over a Robert Jones bandage. Caudal splint placed to top of tuber calcis.
3. Hock and tibia. Contoured lateral splint (e.g. aluminium tubing) from foot to lateral thigh applied over a Robert Jones bandage. As with radius, try to prevent abduction and consequent skin penetration medially.
4. Femur. No splinting necessary because of muscle cover.

Medication

1. Start intravenous broad-spectrum antibiotic therapy.
2. Analgesics.
3. Probably best not to sedate.

Radiography (see Chapter 25)

Try to obtain at least two views of the whole fractured bone at right angles to each other. It is often necessary to obtain *multiple oblique views* of a complex fracture to gain a full understanding of its configuration prior to deciding prognosis and treatment.

If the horse is to be transported to another clinic for treatment, consultation with surgical colleagues prior to referral most logically takes place *at this stage* when *the fracture is stabilized* and *the radiographs have been taken*.

Emergency transport

1. Minimize distance horse has to walk.
2. Van-type horsebox probably preferable to trailer because it is more stable. However, it may be more difficult for the horse to enter and leave.
3. Position with good limbs at front to counteract braking.
4. Confine tightly and provide support with bales. Leave head and neck loose to aid balancing.
5. Restrain foals manually.

Surgical techniques

Surgical techniques for fracture management are continually changing and improving. The advent of arthroscopy allows both the removal of

small articular 'chip' fractures, e.g. in the carpus and fetlock, and the accurate reduction of major displaced articular fractures, e.g. third carpal slab fractures and displaced metacarpal condylar fractures prior to screw fixation.

Major displaced fractures usually require stabilization with some form of internal fixation device, usually screws (commonly inserted as lag screws) or combinations of screws and bone plates. Techniques for the use of external fixation in the horse have also been described. The principles governing such fixation are:

- Aseptic technique – infection remains one of the most devastating complications of equine fracture surgery, and every effort should be made to attain the highest possible standards of surgical asepsis.
- Reduction – accurate anatomical reduction, particularly of articular surfaces.
- Immobilization – rigid stabilization – often by interfragmentary compression using the lag-screw principle and/or ASIF dynamic compression plates. The newer locking compression plates offer even more rigid fixation.
- Minimize soft-tissue damage before, during and after surgery.
- Early controlled exercise to avoid the complications of fracture disease outlined above.

It is impossible to document here the methods used for internal fixation of every individual fracture encountered in the horse, but some of the commonly treated fractures are listed below:

- Sagittal and frontal plane proximal phalanx fractures – lag screws.
- Apical- and mid-body proximal sesamoid bone fractures – lag screw (Figure 16.12) or circumferential wiring.
- Condylar fractures of the distal third metacarpal/metatarsal bone – lag screws.
- Transverse/oblique fractures of the third metacarpal/metatarsal bone – compression plate.
- Carpal slab fractures – lag screw.
- Olecranon fractures and long bone fractures – compression plate (Figure 15.8).
- Chip fractures of the fetlock and carpus – arthroscopic removal.

Not all equine fractures need surgery. Some minimally displaced or incomplete fractures heal satisfactorily with conservative treatment – examples include certain 'stress' fractures seen particularly in young racehorses (e.g. third metacarpal bone, tibia, humerus, pelvis), some incomplete proximal phalangeal and lateral condylar fractures, many splint bone fractures, some accessory carpal bone fractures and non-articular distal phalanx fractures.

Proximal limb bone fractures in adult horses are very difficult to reduce and stabilize adequately at surgery. Sustained traction with ropes and pulleys or an overhead hoist may aid reduction of overriding fractures. Technical advances and increasing experience will undoubtedly improve success rates, but euthanasia still remains the only realistic option for many horses with a long-bone fracture.

Fractures in small horses, i.e. foals/yearlings and small pony breeds, are easier to treat in mechanical terms though 'youthful exuberance' postoperatively can complicate management and disrupt fixation. Most reported cases of successful proximal longbone fracture in the horse have been in young horses.

Aftercare

External support

Following fracture treatment most surgical wounds should at least be covered in the immediate postoperative period to prevent contamination. Bandaging may help to limit soft-tissue swelling in response to the surgery and when combined with splints may provide some support for a fracture. Casting is appropriate to some major distal limb fractures for the recovery process, e.g. major fractures of P2, P1, Mc/Mt III, carpus and possibly distal radius and hock. Fractures proximal to these sites do not benefit from cast support, and casting may actually be detrimental.

Figure 15.8 Radiograph demonstrating internal fixation techniques involving multiple dynamic compression plates and a dynamic hip screw to fix a radial fracture in an adult horse. The advent of new implants has provided greater opportunity to fix the more complex and more proximal fractures in the horse although surgical fixation of the proximal long bones in adult horses, some weighing in excess of 500 kg, is still a formidable challenge.

Recovery

Anaesthetic regimens that lead to a quiet and coordinated recovery should be used. Sedation in the recovery box may also help. Assisting recovery with ropes attached to the head collar and tail can help to stabilize horses in the standing position and prevent multiple attempts at standing which can overload implants. Dedicated pool recovery systems can also be used but are not commonly available.

Antibiosis

Antibiotic cover should be provided for all fracture cases where implants are used.

Analgesia

Horses undergoing fracture fixation usually require perioperative analgesia.

Confinement and exercise

Most horses undergoing fracture treatment require a period of post-operative box rest, the duration of which varies according to the specific fracture. Exercise should start with short periods of walking in hand which are gradually increased in duration and eventually replaced with ridden walking and trotting exercise. Controlled ridden exercise is better than 'turning the horse out' to convalesce.

15.4 OTHER BONE DISEASES

Osteomyelitis

Osteomyelitis is an infection involving the cortex and medullary cavity of bones.

Aetiopathogenesis

Routes of infection:

- Haematogenous – usually in foals less than 4 months of age.
- Penetrating wounds.
  - Traumatic or surgical.
  - Particularly in association with internal fixation of fractures where the presence of implants tends to promote the persistence of infection.
- Extension from a contiguous infected focus.

Organisms are usually similar to those listed for septic arthritis, i.e. coliforms, *Staphylococcus* and *Streptococcus*.

Haematogenous localization usually occurs in the metaphysis or epiphysis due to sluggish blood flow within the large venous sinusoids. Infection may spread from the bone into adjacent soft tissues or into the joint.

Diaphyseal osteomyelitis is usually a consequence of open reduction and internal fixation of fractures.

Treatment

- Surgical drainage of exudate.
- Removal of infected or necrotic material.
- Stabilization – in cases of fractures with internal fixation – provided that implants are maintaining stability they should be left until such time as the fracture has healed at which time they should be removed.
- Antibiotic therapy.
- Bone grafting.

Prognosis

Established osteomyelitis is a very serious condition with a guarded prognosis. Infected fracture sites containing implants may still heal provided stability can be maintained. The infection then often resolves once the implants are removed. If infection results in instability due to major implant loosening or breakage, healing does not occur, and unless stability can be achieved by further intervention the horse is permanently incapacitated.

Osteitis

Osteitis is an infection of the cortical layer of bone.

Aetiopathogenesis

Usually a consequence of traumatic open wounds, particularly in the metacarpus/metatarsus. Such trauma exposes cortical bone, disrupts the periosteum and compromises the blood supply to the outer cortex. Such wounds are inevitably contaminated, and the vascular disruption allows infection to become established. The devascularized outer cortex separates from the underlying bone to form a sequestrum, and infection persists.

Treatment

Traditionally, surgical debridement and removal of necrotic and infected tissue – particularly dead bone (sequestra) – is performed.

Medical management with antibiotics and local wound irrigation until the sequestrum has been resorbed may also be successful but may take a long time.

Physitis

There is enlargement of the physeal region of longbones in growing horses.

Aetiopathogenesis

The aetiology is unclear. It is seen in rapidly growing young horses and may be related to other developmental orthopaedic disorders including osteochondrosis.

Clinical signs

There is widening of the bone at the level of the physis. The metaphysis thus flares out adjacent to the physis, particularly at the level of the distal radius, tibia and metacarpus/metatarsus. Variable lameness/stiffness may occur. It may be seen in conjunction with other developmental orthopaedic diseases including angular limb deformities, OCD, flexural deformities and wobbler syndrome.

Radiology (see Chapter 25)

Flaring of the metaphysis and epiphysis adjacent to the physis may be seen, and also irregularity of the physis and sclerosis of bone surrounding the physis.

Treatment

Treatment comprises reduction of energy content of ration to slow growth, ensuring correct mineral balance in ration, rest, and administration of a NSAID.

Nutritional secondary hyperparathyroidism (bran disease, big head, osteodystrophia fibrosa)

Aetiopathogenesis

- Due to relative calcium deficiency and excess phosphate.
- Low calcium leads to increased secretion of PTH and bone resorption.
- Exclusively grain diets (e.g. bran) without hay or mineral supplementation may have such a calcium/phosphorus imbalance.
- The flat bones of the jaw and skull appear to be preferentially affected in the horse.

Clinical signs

- Symmetrical enlargement of the mandible and facial bones.
- May lead to loosening of teeth.

Treatment

Treatment consists of dietary correction and mineral supplementation.

Hereditary multiple exostoses (multiple cartilaginous exostoses, osteochondroma)

This is a rare inherited congenital condition involving multiple abnormal bony projections from long bones, ribs and pelvis. Solitary osteochondromas also occur especially on the caudal aspect of the distal radius (see carpus). The exostoses usually do not continue to grow after the horse is skeletally mature. Single lesions may be amenable to surgical removal.

Calcinosis circumscripta

Calcified, granular amorphous deposits occur in the subcutaneous tissues usually near joints or tendon sheaths, particularly on the lateral aspect of the stifle where it is frequently firmly attached to the joint capsule. If associated with lameness they may be surgically removed, but as this may involve removing a portion of joint capsule, there are associated risks of wound breakdown, poor healing and septic arthritis.

Rickets

Rickets is caused by vitamin D deficiency but is *very rare* in horses. Vitamin D is normally ingested in plant material or formed in the skin in response to sunlight. Deficiency is therefore uncommon. It results in metaphyseal flaring and bowing of the extremities, but other conditions such as physitis and angular limb deformities are much more common causes of such signs.

Hypertrophic osteopathy (hypertrophic pulmonary osteoarthropathy, Marie's disease)

Aetiopathogenesis

It is classically associated with space-occupying pulmonary lesions, e.g. tumours, abscesses, tuberculosis, and has also been reported in association with abdominal lesions, e.g. ovarian dysgerminoma.

Suggested disease mechanisms include neural pathways involving the vagus nerve and humoral factors such as elevated oestrogen concentration.

Clinical signs

- Symmetrical limb swelling.
- Oedema.
- Minimal lameness.

Radiology

Periostitis particularly at the proximal and distal metaphyseal regions of the long bones.

Osteoporosis/osteopenia

Aetiopathogenesis

Osteoporosis is usually seen as a consequence of disuse after severe protracted lameness or after application of external immobilization in the form of a cast. It occurs most dramatically in foals after casting and can result in pathological fracture if severe.

Radiology

Bones appear generally less radiodense than normal with punctate lucencies, and cortices appear thin. In the distal limb the proximal sesamoid bones are often the most dramatically affected (sometimes referred to as 'ghost' sesamoids).

Treatment

Treatment comprises gradual removal of support and return to normal activity. Casts should be removed as soon as possible in foals and replaced with bandages and splints which are progressively removed to reduce the risk of pathological fracture.

Fluorosis

Aetiopathogenesis

Ingestion of toxic amounts of fluorine in food or water – usually a result of industrial contamination via effluent or fumes.

Clinical signs

- Generalized stiffness, lameness and unthriftiness.
- Exostoses particularly on the metacarpus/metatarsus.
- Mottling of the teeth.

Radiology

Osteoporosis and exostosis formation.

Treatment

Removal from source of contamination.

FURTHER READING

Auer JA, Stick JA (2012) Equine surgery, 4th edn. Elsevier, St Louis

Baxter GM (2011) Adams' and Stahak's lameness in horses, 6th edn. Lippincott, Williams & Wilkins, Baltimore

Butler JA, Colles CM, Dyson SJ, Kold SE, Poulos PW (2008) Clinical radiology of the horse, 3rd edn. Wiley-Blackwell, Oxford

Nixon A (1996) Equine fracture repair. Saunders, St Louis

Ross MW, Dyson SJ (eds) (2003) Diagnosis and management of lameness in the horse. Saunders, St Louis

Wyn-Jones G (1988) Equine lameness. Blackwell Scientific Publications, Oxford

Chapter 16

Orthopaedics 2. Diseases of the foot and distal limbs

Michael C.A. Schramme, Raphael Labens

CONTENTS

16.1 Diseases of the foot 330
Cracks of the hoofwall 330
Seedy toe/separation of the hoofwall 330
Hoofwall avulsions/separations 331
Laminitis 331
Pododermatitis (bruising and abscess of the foot) 334
Nailbind 335
Keratoma 335
Canker 336
Thrush 336
Penetrating wounds to the sole 336
Quittor/necrosis of ungular cartilage 338
Lameness due to foot imbalance 338
Fractures of the distal phalanx 339
'Pedal osteitis' 340
Bone contusion of the distal or middle phalanx 340
Osseous cyst-like lesions of the distal phalanx 340
'Sidebone' 341
Fractures of the navicular bone 341
Navicular disease 341
Distal tendinitis of the deep digital flexor tendon (DDFT) 344
Collateral desmitis of the distal interphalangeal (DIP) joint 345
Traumatic and degenerative arthritis of the DIP joint 346
Septic arthritis of the DIP joint 346
Flexural deformity of the DIP joint 346
16.2 Diseases of the pastern and fetlock region 347
'Ringbone' 347
'Subluxation' of the pastern joint 348
Fracture of the middle phalanx 348
Fractures of the proximal phalanx 348
Desmitis of the distal sesamoidean ligaments 350
Distal and proximal digital annular ligament desmitis 351
Villonodular synovitis 351
Luxation of the fetlock joint 351
Collateral ligament desmitis of the metacarpo(tarso) phalangeal joint 352
Desmitis of the intersesamoidean ligament 352
Subchondral bone injury in the fetlock region 352
Osteochondritis dissecans (OCD) and osseous cyst-like lesions in the fetlock joint 353
Osteoarthritis of the fetlock joint 353
Sesamoiditis 354
Fracture of the proximal sesamoid bone 354
Breakdown of the suspensory apparatus 355
Annular ligament syndrome 356
Tenosynovitis of the digital flexor tendon sheath and longitudinal tears of the superficial or deep digital flexor tendon 357
Septic tenosynovitis of the digital flexor tendon sheath (DFTS) 357
Flexural deformity of the fetlock joint 358
Angular deformity of the fetlock 359
16.3 Diseases of the metacarpus and metatarsus 359
Condylar, metaphyseal and diaphyseal fractures of the third metacarpal/metatarsal bone 359
'Bucked shins'/'sore shins'/dorsal metacarpal disease 360
Proximal palmar/plantar metacarpal/metatarsal pain 360
'Splints' 361
Fractures of the splint bones 361
Superficial digital flexor tendinitis 363
Spontaneous rupture of the superficial digital flexor tendon 364
Deep digital flexor tendinitis 364

© 2013 Elsevier Ltd
DOI: 10.1016/B978-0-7020-2801-4.00016-X

Tendon lacerations 365
Desmitis of the suspensory ligament 365
Desmitis of the accessory ligament of the deep digital flexor tendon 367
Further reading 368
Appendix 368

16.1 DISEASES OF THE FOOT

Cracks of the hoofwall

Classification

Vertical defects in the hoofwall occur along the direction of the lamellae of the hoofwall. They can occur in the toe region (toe cracks), quarters (quarter cracks) and heels (heel cracks). Superficial cracks do not involve the sensitive laminae, but deep cracks do. Deep cracks are often accompanied by localized infection of the dermis. Cracks can originate from the coronary band or from the solar margin. They can be partial or complete (from coronary band to solar margin).

Aetiology

- Overgrown hoofwall may lead to splitting extending proximally from the solar margin.
- Dry, poor quality horn and poor foot balance increase the incidence of cracks.
- Defects caused by tearing away of a shoe and part of the wall.
- Chronic traumatic defects in the coronary band (false quarters)
- High prevalence in the toe region of Draft horses and the medial quarter of Standardbred racehorses.

Clinical signs

- Obvious hoofwall defect with or without local discharge, heat, or lameness.
- Hoof testers and a uni-axial diagnostic analgesia are used to determine clinical significance of the crack.
- Pain associated with deep cracks is caused by irritation of the laminar and coronary dermis by differential movement of both sides of the horn defect.
- Haemorrhage from repetitive irritation.
- Purulent discharge from secondary infection of the exposed dermis.

Treatment

- Prevent with regular good foot care to eliminate foot imbalances.
- Application of oil to the hoof to prevent excessive dryness.
- Oral administration of biotin and methionine to promote good quality horn growth.
- Groove or burn transversely down to healthy continuous horn at the proximal limit of incomplete cracks to prevent progression.
- Adequate immobilization of the crack to eliminate irritation and lameness and to allow healing from the coronary dermis down.
  - A full bar shoe with clips drawn on either side of the crack.
  - The hoofwall can be lowered either palmar/plantar to the defect (quarter cracks) or directly distal to the defect (toe cracks) to eliminate upward pressure and irritation of the coronary dermis during weight-bearing.
  - Heartbar shoes and frog pads are used to transfer the load of weight-bearing from the walls to the frog.
  - Hoof binding resins, acrylics and prosthetic repair materials. These materials should only be used after thorough debridement and elimination of deep-seated infection.
  - Transversely placed nails, screws and wire, screws and metal plate, or lacing with various materials (e.g. wire, polyester).
- Partial hoofwall resection may be needed for complicated cracks that prove refractory to stabilisation techniques.

Prognosis

Favourable for most cracks. However, cracks associated with chronic coronary band defects are more difficult to eliminate and have a tendency to recur.

Seedy toe/separation of the hoofwall

Definition

'Seedy toe' describes a condition in which the dermal and epidermal layers are separated in the toe region of the foot. Newly formed horn follows the line of separation between layers and perpetuates the defect. After some time the separation is visible in the white line in the toe region, and the dermal structures are exposed to ascending infection.

Aetiology

- Focal haemorrhage, seroma formation, or inflammation between the layers of the hoof wall. This is followed by a failure of the damaged laminae to produce keratin.
- Rotation of the distal phalanx in complicated laminitis.
- Onychomycosis has been related to the presence of fungal infection of the hoof wall.

Clinical signs

- Presence of brown, crumbly horn-like matter along the white line in the toe region.
- Non-keratinized void extending from the white line proximally between the layers of the hoof wall. The defect may extend as far proximally as 2/3 of the distance between the solar margin and the coronary band.
- Lameness in severe cases.
- Percussion of the dorsal hoofwall may produce a characteristic hollow sound.

Diagnosis

- Careful exploration of the undermined wall with a blunt radiopaque probe allows radiographic documentation of the extent of separation.
- Gas shadow in the soft tissues dorsal to the distal phalanx on lateromedial radiographs of the foot.

Treatment

- Regular cleaning of the defect and shoeing with wide-webbed, flat shoes to increase the base of support for the foot are sufficient in mild cases.
- In more severe cases, all separated horn must be removed from the solar margin of the toe to a level where normal interlaminar bond is present. The exposed laminae are medicated under a dressing. This allows newly formed horn to grow distally in a normal direction and restores a normal interlaminar bond.

The use of antifungal medication and foot soaks has been advocated (e.g. White Lightning-Grand Circuit Products LLC, Louisville, Kentucky.)

Hoofwall avulsions/separations

Aetiology

- Incomplete avulsions can be caused by complicated cracks, infection with separation of the white line, penetration of the white line by a foreign body, chronic foot imbalance, overgrown feet and improper shoe removal.
- Complete avulsions usually result from lacerations caused by sharp objects.

Clinical signs

- Moderate lameness is typical following an acute hoof wall avulsion. Severe lameness is indicative of damage to underlying bony, synovial, tendinous or ligamentous structures. Occasionally, a foreign body may be lodged deep between the epidermal and dermal laminae.
- If lameness persists, the clinician should be suspicious of deep seated soft tissue or synovial infection or a phalangeal fracture.

Diagnosis

- Following meticulous cleaning and aseptic preparation the injury should be examined carefully for damage to structures deep to the avulsed hoof wall.
- Plain and contrast radiography, computed tomography (CT) or magnetic resonance imaging (MRI) may help to identify the source of persistent pain.

Treatment

Avulsions without damage to coronary dermis are managed by careful debridement, removal of all loose horn, topical application of antiseptic medication and bandaging until the defect is keratinized. A mixture of sugar and betadine solution works well both as antiseptic and astringent.

- Avulsions with coronary band involvement are treated following the same principles. The coronary band must be meticulously reconstructed by suturing. If the wound is severely contaminated or has lost blood supply, delayed primary or secondary closure may be more appropriate.
- Complete immobilization of the foot in a distal limb cast for up to 4 weeks is advisable following reconstruction of acute avulsions to allow for primary healing of the coronary dermis.
- A foot-pastern cast works well to protect the exposed dermal tissue against further contamination and provides stability during healing. Once the defect is keratinized with a superficial layer of horn, the cast can be replaced with a shoe, and the defect can be repaired with a hoof resin.
- If a substantial portion of hoof wall has been excised, a full bar shoe can be applied to provide stability.

Laminitis

Definition

Laminitis is a complicated interrelated sequence of inflammatory and vascular events that affect the laminar tissues of the foot and result in varying degrees of breakdown of the interdigitation of the primary and secondary epidermal and dermal laminae of the foot.

Pathogenesis

The pathogenesis remains largely unknown, but the disease is thought to result in hypoperfusion, ischaemia and necrosis of the laminae. Four mechanisms have been suggested:

1. Endotoxin-induced laminar microthrombosis.
2. Shunting of laminar blood flow through laminar arteriovenous anastomoses.
3. Vasoconstriction and perivascular laminar oedema.
4. Activated enzyme destruction of the laminar basement membrane.
    - Laminitis-trigger-factors lead to local enzymatic activation in the horse's foot, in particular of metalloproteinases 2 and 9 (MMP 2 and 9). The MMPs have been shown to be instrumental in the process of destruction of the basement membrane that is the key structure that bridges the epidermis of the hoof to the connective tissue of the distal phalanx.
    - In addition it is also thought that alterations in vaso-active mechanisms (mainly vasoconstriction and AVA shunting), induced by imbalances of vaso-active amines, also produce laminar ischaemia and necrosis in the dermal structures of the hoof wall.
    - In experimental studies intra-cellular glucose deprivation in response to insulin resistance has also been observed to cause laminar detachment by incapacitating cellular desmosomes.
    - The dorsal coronary dermis, dorsal laminar dermis and dorsal solar dermis have a much less extensive collateral circulation than the palmar dermal tissues in the foot, and are therefore more susceptible to ischaemic necrosis.
    - The resulting pain causes further release of catecholamines, which cause vasoconstriction and increase ischaemia in the dermal vasculature.
    - The epidermal-dermal junction becomes oedematous and weakened with loss of interlaminar bond. If this damage extends over a sufficiently large area, suspensory support of the distal phalanx inside the hoof capsule is lost. A combination of the forces of weight-bearing, the pull of the DDFT and dorsal pressure of space-occupying soft tissues (dermal congestion, oedema, seroma and epidermal hyperplasia) result in mechanical separation of the distal phalanx from the hoof wall.
    - If, as in most cases, mechanical separation is localized to the toe region of the foot, the distal phalanx rotates away from the dorsal hoof wall. This displacement is referred to as *palmar rotation* as the heels remain suspended by their laminar attachments.
    - In exceptional cases, the lateral or medial wall may be worst affected, in which case the lateral or medial wall of the distal phalanx may displace distally within the hoof capsule without evidence of rotation of the distal phalanx away from the dorsal wall. This is referred to as *lateromedial rotation*.
    - In severe cases in which the suspensory support of all the laminae is lost, the entire distal phalanx drops distally within the hoof without evidence of rotation. This displacement is referred to as 'sinking'. 'Sinkers' have a much worse prognosis than horses with dorsopalmar or lateromedial rotation.
    - Systemic changes in laminitis can be found in the cardiovascular (tachycardia, hypertension, alterations intrinsic coagulation system) and endocrine systems (increased catecholamines, cortisol, testosterone and plasma renin, and decreased thyroxine). Renal damage (glomerulonephritis, medullary necrosis) and liver disease have also been reported.

Aetiology

The aetiology of laminitis is multifactorial, because many different predisposing factors can trigger the same vascular or inflammatory mechanisms. However, in many horses with systemic disease these

mechanisms may be caused by the presence of circulating endotoxins.

- Nutritional carbohydrate (grain) overload may lead to dysbacteriosis of the intestinal flora, overgrowth of Gram-negative bacteria and absorption of endotoxins from the bowel.
- Overeating of lush grasses high in fructan may result in hindgut fermentation and lactic acid production, resulting in grass founder.
- Systemic infections (e.g. endometritis, pneumonia, enteritis).
- Horses recovering from colic surgery are at risk of developing laminitis, possibly because of intestinal dysbacteriosis and the risk of absorption of endotoxin and the frequent presence of blood hypercoagulopathies.
- Ingestion of large amounts of cold water.
- Electrolyte imbalances (Ca/P and Na/K).
- Chronic overloading of the sound limb in severe unilateral lameness.
- Strenuous and often prolonged work on hard surfaces.
- Long-term or high-dose corticosteroid administration. Corticosteroids increase the vasoconstrictory response of the digital vasculature to catecholamines and serotonin in circulation.
- High testosterone concentration has been associated with coagulopathy in the dermal vasculature and may explain why stallions appear to be at higher risk for laminitis.
- High oestrogen concentrations have been associated with laminitis in mares with anoestrus or continuous oestrus. Oestrogens are abundantly present in some grasses or legumes.
- Exposure to black walnut wood shavings.
- Hypothyroidism.
- Equine pituitary pars intermedia dysfunction (PPID) (Cushing's disease) (see Chapter 9).
- Equine metabolic syndrome (EMS) (see Chapter 9).

Classification

The clinical presentation of laminitis can be classified into three different categories:

1. Acute laminitis: acute stage of systemic illness with accompanying lameness.
2. Chronic laminitis not associated with a clinically identifiable episode of acute systemic illness and characterized by milder and inconsistent lameness due to low-grade laminal pain and disrupted normal laminar junction.
3. Refractory laminitis: residual lameness due to structural foot changes (rotation or sinking) following resolution of the acute systemic illness.

Clinical signs

- Horses with acute laminitis show signs of systemic disease and local signs of foot disease. Systemic signs include elevation of heart rate, increased respiratory rate, hypertension (100–200 mmHg) and anorexia. Fever may be present if the horse is septicaemic or endotoxaemic. Evidence of moderate to severe pain is present (sweating, flared nostrils, muscular tremors). Horses are reluctant to move, and if severely affected, may become recumbent.
- Laminitic ponies on lush spring pasture have a large neck crest that consists of adipose tissue. This crest tends to become firm to palpation in the early stages of the disease. This form of fat distribution is frequently associated with endocrine abnormalities like PPID or EMS.
- A mild form of laminitis in racing Thoroughbreds on a chronic overdose of dietary carbohydrates (i.e. glycogen overload) is characterized by signs of indigestion, low faecal pH (<6.2), poor performance, generalized stiffness and warm feet.
- Signs indicative of foot disease are present in horses with laminitis. Most commonly, both front feet are simultaneously affected (except for cases of contralateral overload). The horse has a bounding digital pulse, often in all four limbs. The hoofwall feels abnormally warm, especially in its dorsal half. In severe cases, close examination may show evidence of a depression at the proximal dorsal margin of the coronary band ('sinker') or exudative discharge from a separated coronary band. Investigation of the sole may show a convex profile just dorsal to the point of the frog if the distal phalanx has rotated. This same part of the sole may be discoloured by haemorrhagic effusion or even show ulceration with prolapse of the solar dermis (Figure 16.1). The white line may also show evidence of widening, blood infiltration, haemorrhage or separation.
- Application of hoof testers or percussion over the sole (dorsal to the point of the frog) and the dorsal wall is usually strongly resented. Response to application to hoof testers is variable when tissue death leads to local loss of sensation.
- Horses with laminitis display a typical stance with the forelimbs stretched out in front and the hind limbs placed beneath the body as close as possible to the centre of gravity. The gait is characterized by a typical 'heel before toe' foot placement and obvious lameness.
- The clinical severity of laminitis may be graded using the Obel grading system. These grades corresponded well to the degree of histopathological damage.
  - Obel Grade 1: shifting weight.
  - Obel Grade 2: typical laminitic gait (stiff and stilted as if 'walking on egg shells'). Lifting of each front foot and resting on the contralateral foot are possible.
  - Obel Grade 3: reluctance to walk with typical laminitic gait. Lifting of each front foot and resting on the contralateral foot are not possible.
  - Obel Grade 4: mostly recumbent.
- Horses with chronic laminitis show signs of foot lameness similar to those of horses with acute laminitis. However, when

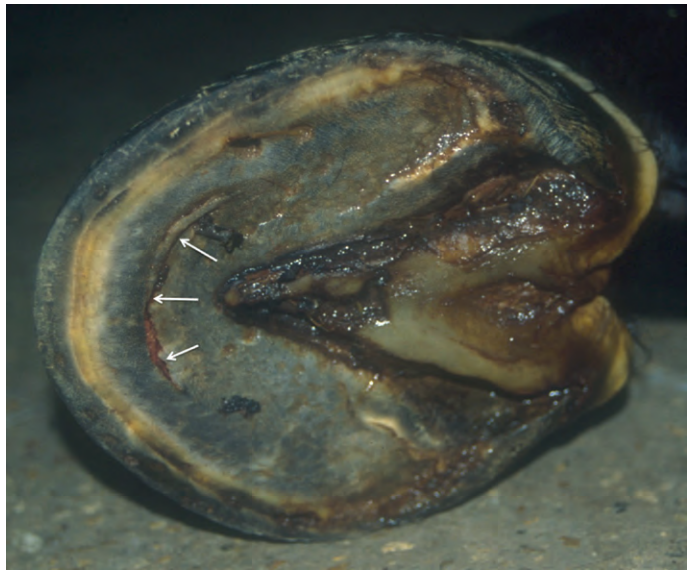


Figure 16.1 Solar penetration in laminitis. Note the semi-circular area, just dorsal to the frog, where the solar margin of the distal phalanx is penetrating the sole (arrows).

the distal phalanx becomes stable, lameness improves, and the foot starts to develop some evidence of long-term deformation. Rings parallel to the coronary band reflect temporary alterations in hoof growth. In laminitis, these rings tend to be closely packed in the toe region and diverge towards the heels, reflecting the discrepancy in rate of hoof growth in these different areas of the foot. The result of decreased toe growth is ultimately a 'slipper' foot. Convexity of the sole accompanied by ulceration and prolapse and necrosis of the dermis may be the result of sustained pressure of the tip of the rotated distal phalanx on the solar dermis and its blood supply in this area.

- The dead space caused by separation and rotation between the distal phalanx and the dorsal hoof wall in the toe region is gradually filled by epidermal scar tissue. This wedge of epidermal hyperplasia maintains the malalignment of both structures and can be observed as a wide and distorted white line on the solar surface of the foot.
  - Secondary infection is a common feature of chronic laminitis. These infections can range from simple subsolar and laminal abscesses to more extensive infections that can lead to septicaemia. Infection characteristically causes a sudden exacerbation of lameness.
  - Endocrine function tests may be necessary to identify the presence of endocrine abnormalities like PPID or EMS.

Radiology

- Radiographs are necessary to identify the degree of phalangeal displacement, the presence of infection and chronic bony remodelling of the distal phalanx (Figure 25.6).
- Measurements that are useful to determine whether displacement of the distal phalanx has occurred must be performed on a perfect lateromedial radiograph of the foot. Distal phalangeal rotation is assessed by subtracting the angle between the dorsal wall and the sole from the angle between the dorsal surface of the distal phalanx and the sole. Quantification of the degree and progression of distal phalangeal displacement, identified by sequential radiographs, is important prognostically.
- Other useful measurements include the proximal and distal dorsal hoof wall thickness (maximum 18 mm for normal Thoroughbreds) and the founder distance (the vertical distance between the level of the dorsoproximal aspect of the hoof wall and the level of the extensor process of the distal phalanx) which should be less than 10 mm in normal horses. The founder distance has been described as the most accurate prognostic indicator for horses with complicated laminitis that have suffered displacement of the distal phalanx within the hoof capsule.
- Sinking may be difficult to identify radiographically because the dorsal wall and the distal phalanx remain parallel. The presence of sinking can usually be confirmed only by comparing the dorsal hoof wall thickness and founder distance on a marked lateromedial radiograph with normal values or if disease is unilateral by comparing images to the contralateral foot.
- The presence of gas opacities on foot radiographs may be indicative of local infection.
- Attention should also be paid to evidence of remodelling of the toe of the distal phalanx. Fractures, upwards curling of the apex due to new bone production ('slipper bone'), irregular radiolucencies and extensive osteolysis with alterations in shape of the apex of the distal phalanx justify a guarded prognosis.
- Production of new bone along the dorsal margin of the distal phalanx, with or without rotation, is usually subtle and indicates the presence of laminar tearing.
- Radiographic examinations should also include a dorsopalmar view. While all of the above abnormalities are usually recognized in lateromedial views, rarely lateral or medial laminar detachment exceeds dorsal laminar damage and leads to asymmetric sinking of the distal phalanx (lateromedial rotation), which can only be accurately recognized in dorsopalmar projections.
- Digital venograms have been used to identify perfusion deficits, which usually indicate a poor prognosis.

Treatment

Acute laminitis should be treated as an emergency. Destruction of the laminar bed starts several hours before the first signs of lameness are apparent, and continues to progress if treatment is not initiated.

1. Removal of initiating cause.
   - Orally administered liquid paraffin, or mineral oil, block absorption of endotoxins from the GI tract.
   - Horses should be removed from pasture and fed hay and water only.
   - Systemic infections should be treated vigorously with antibiotics. Antibiotics are also advisable as prophylaxis against secondary sepsis of the foot.
   - Prior to the administration of antibiotics, anti-endotoxic medication may be considered, as the bactericidal effect of antibiotics may result in an abundance of available bacterial cell wall fragments (i.e. endotoxin) for absorption. Polymyxin-B and endotoxin-specific antibodies are used clinically. These agents have shown the greatest effect when given prior to the release of endotoxins in circulation.
   - Feeding virginiamycin to horses on a high-grain and pellet feed diet has been proposed as a preventive measure to inhibit caecal lactic acid production and dysbacteriosis.
2. Treatment of peripheral vascular disease. The application of ice on horse's feet remains controversial. Its presumed beneficial effect lies in causing vasoconstriction during the prodromal stage of laminitis which may reduce the delivery of vasoactive and inflammatory substances to laminar capillaries, which in turn, reduces the activity of tissue-degrading enzymes. Icing following clinical manifestation of disease may reduce oedema formation but potentially aggravates any ongoing ischaemic vascular events. To reduce tissue temperature in the horse's feet effectively, ice boots should incorporate the entire distal limb up to the carpus/tarsus.
   - Acetylpromazine to reduce vasoconstriction and hypertension.
   - Isoxsuprine hydrochloride may counteract early vascular changes in laminitis.
   - NSAIDs to control digital pain and to interrupt the pain–hypertension cycle.
   - Potassium chloride is supplemented to correct depletion of potassium.
   - Sodium is removed from the diet.
   - Pentoxifylline alters the rheological properties of erythrocytes and improves circulation and oxygen delivery.
   - Topical application of nitroglycerine may improve local circulation in the foot.
3. Mechanical support of the diseased laminae.
   - Perineural analgesia and forced exercise MUST be avoided.
   - The horse should be bedded on a deep soft bed (e.g. shavings, sand, peat) that conforms easily to the shape of the sole and frog.
   - Pre-shaped frog pads (e.g. Theraflex pads, Lily pads), Styrofoam pads or plaster of Paris slippers help transfer the load of weight-bearing from the walls to the frog (Figure 16.2).

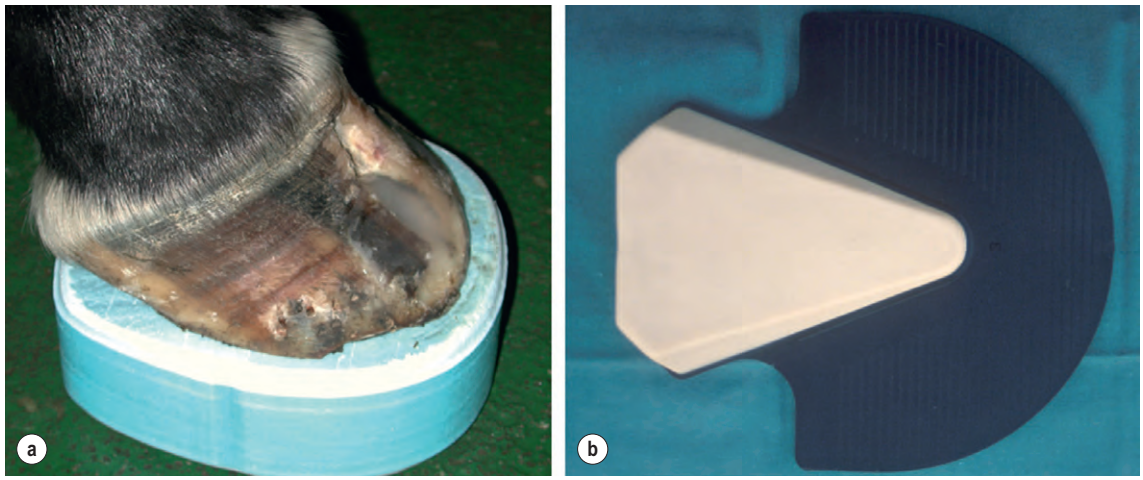


Figure 16.2 Methods of reducing the load of weight-bearing through the hoof wall in horses with laminitis. (a) The Styrofoam™ pad – the weight of the horse compresses the foam so that it conforms to the undersurface of the foot, providing a spread of the weight-bearing load. (b) The Theraflex™ pad – this consists of a hard plastic plate (black) with a firm rubber wedge (cream-coloured) that is positioned over the frog to act as a frog support. Rolled bandages can be used in the same capacity, but the firmness of the wedge is more efficient at protecting the solar surface from load.

In chronic laminitis, treatment is aimed at correcting residual foot problems and preventing recurrence of systemic disease.

1. General management of the horse.
   - Limited intake of carbohydrates to control obesity.
   - Supplement the ration with 30 g of potassium chloride daily.
   - Supplementation with oral methionine and biotin daily.
   - Pergolide, cyproheptadine or trilostane for horses with Cushing's disease.
2. Corrective foot trimming.
   - Guided by good lateromedial foot radiographs.
   - Removal of all excess horn in the toe region and trimming of the heels to restore normal spatial alignment between the distal phalanx and the hoof.
   - Dorsal wall resection may be indicated for severely affected horses. Dorsal wall resection is more effective in chronic than acute laminitis. This should never be taken lightly by the treating clinician. Once the dorsal wall is removed, the horse requires close follow-up and intensive aftercare!
3. Therapeutic shoeing for laminitis.
   - A wide-webbed, seated-out, bar or egg-bar shoe of thick steel with a rolled toe, increases the base of support, minimizes the amount of pressure on the sole, and facilitates breakover of the foot.
   - A heart-bar shoe may support and immobilize the distal phalanx sufficiently to prevent further rotation and allow for undisturbed revascularization of diseased laminae and restoration of the interlaminar bond.
   - Derotation or rail shoes have been used to help re-align the distal phalanx inside the hoof capsule. Typically these shoes are placed on the trimmed heel and avoid contact with the dorsal half of the sole and solar margins.
   - Various types of impression compounds have been used to pack the pain-free part of the solar surface of the foot and spread the load of weight-bearing away from the solar margin.
4. Deep flexor tenotomy.
   - Tenotomy is performed in the mid-metacarpal region and should be combined with an extended heel derotation shoe.
   - This procedure may produce significant pain relief and may prevent further dorsal laminar detachment and rotation of the distal phalanx.
   - Tenotomy appears more effective in chronic laminitis than in horses with acute laminitis, sinking or foot sepsis.
   - Tenotomy should be regarded as a salvage procedure for valuable breeding horses. The beneficial effects may only be temporary. The procedure can be repeated distally to the previous tenotomy site.
   - Similar relief has also been described following inferior check ligament desmotomy.
   - In one study, 59% of horses were alive for at least 2 years after a tenotomy.

Prognosis

Most horses with acute laminitis recover with conservative treatment, without the use of radical trimming or shoeing.

- If a horse with acute laminitis shows no rotation by day 10, it can come off medication by day 20 and can usually be safely considered as recovered by day 60.
- The prognosis becomes unfavourable when:
  - the acute phase continues for more than 10 days.
  - digital pain is uncontrollable and the horse is mostly recumbent.
  - blood pressure remains higher than 200 mmHg for more than a few hours.
  - secondary infection is present.
  - displacement of the distal phalanx has occurred. Full athletic recovery is still possible for horses in which rotation does not exceed 5.5°.
  - the founder distance has exceeded 15 mm.
- Prognosis becomes hopeless in cases of:
  - sinking of the distal phalanx accompanied by a founder distance that has exceeded 20 mm.
  - exungulation of the hoof.

Pododermatitis (bruising and abscess of the foot)

Definition

Pododermatitis is traumatic inflammation of the solar dermis with or without secondary infection.

- Subsolar bruising can occur in any portion of the solar surface of the foot.

- Corns are pressure-induced bruises in the angle of the wall and the bar (usually medially).
- Chronic subsolar bruising is caused by chronic, repetitive crushing of the solar dermis and is seen most commonly in thin-soled, flat-footed horses.
- 'Pus in the foot' is used to describe both subsolar abscesses, secondarily infected corns, bruises or penetrating wounds of the sole, and infections of the 'white line' (dermal-epidermal junction).
- 'Gravel' is a layman's term to describe a draining tract at the coronary band caused by eruption of a subsolar/submural abscess.

Aetiology

- Bruising of the dermis is caused by external pressure, either from the horn itself or from hard foreign bodies (e.g. irregular ground surface, stones, poorly fitted shoes).
- In laminitis with rotation of the distal phalanx, severe bruising can be caused by downward pressure of the apex of the distal phalanx.
- Thin-soled, flat-footed horses (mostly Thoroughbreds) and horses with long-toe/low-heel conformation are predisposed to solar trauma.

Clinical signs

- Varying degree of lameness. Frequently, the horse is bearing minimal weight, often on the toe only, and resting the foot continuously when standing.
- Increased warmth of the foot, with a bounding digital pulse and enlargement of the palmar and palmar digital arteries.
- Pain on percussion of the sole or walls.
- Exploration of the suspected part of the sole/white line may reveal dark red discoloration ('dry corn') or a seroma ('moist corn'), or lead to a sudden release of black (superficial infection; Figure 16.3) or yellow (deep necrotizing infection) purulent material.
- Expansion of the purulent infection may result in partial or complete undermining of the frog and solar horn. If pus can not escape via the sole or white line, proximal expansion can occur between the hoofwall and laminar dermis, resulting in eruption of infection between the coronary dermis and the proximal wall (gravel).

Treatment

1. Removal of the shoe may be sufficient treatment for corns.
2. Prevention by proper, regular trimming and wide shoeing.
3. Surgical shoes to render a horse with a bruise usable:
   - full bar shoe.
   - set or dropped heel shoe.
   - silicone rubber sole filler or leather anti-concussion pads.
4. Thinning the solar horn over the bruised site, without exposing the dermis. The horse should be rested and left barefoot on a thick, soft bed until sound. The use of NSAIDs may be appropriate in severe cases.
5. When infection is present, all the underrun solar horn must be removed to provide drainage and to allow topical application of medication and natural debridement of necrotic dermis. The edges of the horn defect should be thinned gradually. The foot should be medicated daily with an antiseptic solution and kept bandaged to protect it from external contamination.
6. Tetanus antitoxin or a toxoid booster should always be administered to any horse with a subsolar infection if immunization against tetanus is not current.
7. In cases of 'gravel', the communicating solar abscess should be exposed to establish distal drainage. Part of the separated laminar horn may have to be removed to expose the necrotic laminar dermis. Avoid removing the wall. Proximodistal irrigation with an antiseptic solution is useful to eliminate submural infection.

Figure 16.3 The release of pus from a subsolar abscess (arrow). The horse usually improves very quickly after draining the subsolar abscess, especially if there is considerable pressure in the abscess.

Nailbind

Definition

Direct injury or bruising of the solar or laminar dermis by a shoe nail.

Clinical signs

The horse may haemorrhage from the nail hole and become instantly lame, but more commonly, lameness develops over a few days, as a result of intermittent trauma to the dermis during exercise. External contamination through the nail hole often results in secondary septic pododermatitis. Nail removal and localized pressure may cause drainage of black purulent material from the nail hole.

Treatment

Shoe removal and widening of the nail hole to provide adequate drainage.

Keratoma

Definition

Localized, cone-shaped or cylindrical wedge or cystic structure of abnormal horn between the laminar horn and dermis.

Aetiology

1. Uncommom tumour derived from the keratin-producing epidermal cells of the stratum germinativum of the epidermis.

2. Chronic injury or irritation of the stratum germinativum by chronic hoof wall cracks, submural foreign bodies or nail bind.

Clinical signs

- Intermittent lameness due to recurring purulent infections draining from the white line or coronary band. Once the keratoma has grown out distally, a break in the continuity of the white line/sole junctions allows bacteria to enter the hoof between the laminar horn and dermis.
- Acute lameness in the presence of infection. Lameness usually disappears with resolution of infection.
- Sometimes insidious, chronic, low-grade lameness due to sustained pressure on the laminar dermis and distal phalanx in the toe region.
- Sustained pressure from the space-occupying keratin mass results in focal osteolysis of the distal phalanx, most commonly in the region of the toe (Figure 25.37).
- Careful inspection of the white line after trimming may reveal an island of abnormal horn with widening or deformation of the white line at the toe.

Treatment

- Surgical excision by partial or complete resection of the hoofwall.
- Fewer complications can be expected to occur following partial hoof wall resections, and the horse is also more likely to return to its previous levels of exercise faster.
- The foot is wrapped with a sterile pressure bandage with an antiseptic/astringent dressing to counteract prolapse of the swollen dermis. After 4–6 weeks, a firm, dry cornified cuticle forms, and bandaging can be discontinued.
- The bandages can be changed every 2 to 3 days, or, alternatively, a plaster of Paris cast can be applied over a bandage for 1–2 weeks at a time, to keep the foot dry and clean.
- After a firm cornified cuticle has formed, the defect can be filled with prosthetic hoof material to protect the laminae until normal horn grows distally to cover the defect.
- A bar shoe with stabilizing clips on either side of the surgically created defect in the hoof wall must be maintained for several months.

Prognosis

Favourable, but up to 12 months may pass before a normal foot has regrown.

Canker

Definition

Canker is a septic process that results in the development of a chronic, hypertrophic, moist pododermatitis of the frog and sole, and deficient formation of solar horn.

Aetiology

- Unhygienic stabling.
- Horses in semitropical climate of southern US.
- Unknown infectious agent (?).
- Individual or breed predisposition. Seen more commonly in Draught horses.

Clinical signs

- Characteristic foul odour.
- Caseous white exudate resembling cottage cheese.
- Ragged, filamentous appearance of frog horn.
- Usually hind limbs affected.
- Proliferative swollen dermis covered with caseous exudate is found when loose horn is removed.
- Lameness only in advanced cases.

Treatment

- Extensive surgical debridement of all affected tissues. Debridement can cause extensive haemorrhage and is best performed with the horse under general anaesthesia and following placement of a tourniquet.
- Careful prolonged post-operative management with daily bandaging, application of topical therapeutic dressings (e.g. benzoyl peroxide in acetone and metronidazole) until the defect is dry and covered with a layer of new horn. Additional debridement of necrotic dermis may be required during bandage changes.

Prognosis

Prognosis is always guarded. Treatment is long and intensive, and recurrence is likely.

Thrush

Definition

Degenerative condition of the central and collateral sulci of the frog, characterized by disintegrating horn and the presence of grey to black material in the affected areas.

Aetiology

- Unhygienic, moist stabling conditions with poor foot care.
- Infection by keratolytic organisms: multifactorial, but often *Fusobacterium necrophorum* is involved.

Clinical signs

- Black discharge in the sulci of the frog with very offensive odour.
- Lameness only when sensitive tissues involved.
- Mainly hind limbs affected.

Treatment

- Debridement of all infected and separated horn. Usually must be debrided several times before infection is controlled.
- Daily topical astringent medication under dressing. Sterile bandaging after each debridement. Continue treatment under shoe with hospital plate.

Prognosis

Guarded to favourable. Worse if sensitive tissues are affected. Prolonged treatment is often necessary.

Penetrating wounds to the sole

Definition

Puncture wound through the sole or frog horn involving the dermis or the deeper structures of the foot.

Aetiology

- Standing on sharp objects, mainly nails.
- Kicking against sharp objects such as pitch fork, nail in wall, etc.
- These wounds invariably become secondarily infected.

Localization

1. Puncture of solar dermis.
   - Septic pododermatitis.

2. Puncture into digital fat cushion.
   - Heel abscess, septic pododermatitis.
3. Puncture into distal phalanx.
   - Mostly when penetration occurs in toe region or peripheral areas of the sole.
   - Commonly results in bacterial osteitis with sequestration of necrotic bone fragment.
   - Marginal or pathologic fractures of the distal phalanx occur rarely.

The most dangerous area for penetration is the middle to palmar/plantar third of the frog and its sulci, in which case the following structures are at risk of injury:

4. Deep digital flexor tendon (DDFT).
   - Results in localized necrosis of tendon with severe lameness.
   - Necrosis has a tendency to spread within the tendon and erode into deeper structures ('melting tendinitis).
5. Navicular bursa (penetration may occur secondary to necrosis of the DDFT).
   - Septic navicular bursitis.
   - Navicular bone osteomyelitis.
   - Secondary infection of the DIP joint may also occur.
6. Impar ligament and DIP joint.
   - Septic DIP joint (in addition to septic tendinitis and bursitis).
7. Digital flexor tendon sheath (DFTS).

Penetrating objects in the palmar/plantar third of the sole may enter the digital flexor tendon sheath, giving rise to septic tenosynovitis and possibly tendinitis of the DDFT.

Clinical signs

- Object may still be embedded in the foot; register location, depth and direction of penetration.
- Acute onset lameness that improves following removal of foreign body. With primary fracture of the distal phalanx or navicular bone, no improvement is seen following removal of the penetrating object.
- Non-weight-bearing lameness commonly occurs 1 day after penetration due to secondary infection of the penetrated tissue.
- Purulent discharge from solar defect. Entry site is sometimes difficult to find when sealed, especially when penetration has occurred through pliable frog tissue.
- Palpation with hoof testers may determine site of puncture as focus of pain.
- Often swelling and heat as far proximal as pastern and fetlock.
- Sometimes the horse displays systemic signs of infection (e.g. septicaemia, increased temperature, anorexia).
- Sometimes a stringhalt-like gait may occur in hind limb following penetration.

Diagnosis

- Hoof testers.
- Careful paring of sole to find site of penetration; establish direction of puncture with blunt sterile probe after thorough cleaning.
- Synoviocentesis and fluid analysis of DIP joint, navicular bursa and DFTS.
- Radiography.
  - If possible, take a foot radiograph with object in place before removal. However, leaving a sharp foreign body in situ during walking and transport can result in greater damage and therefore measures should be taken to minimize this possibility. Alternatively, assessing the direction and penetrative depth of the foreign body when removed is useful. It must be remembered, however, that the final resting position of the foreign body may not be the same as its maximal penetration because some foreign bodies can be partially withdrawn when the foot is lifted after penetration.
  - Plain radiographs may show fractures, osteitis.
  - Positive-contrast radiographs with radiopaque probe inside tract shows direction and depth of penetration.
  - Positive-contrast arthrography, bursography or tenosynography may show communication with solar defect.
  - Positive-contrast sinography of solar defect may show communication with synovial structures but can introduce contamination.

Treatment

1. Acute presentation.
   - Systemic administration of antibiotics and tetanus antitoxin (or a toxoid booster).
   - Establish length, nature and direction of penetrating object and tract.
   - If no synovial, osseous or tendinous involvement, clean the sole thoroughly, pare widely around the site of penetration down to sensitive laminae (1–2 cm diameter), remove all contaminated tissue, provide adequate drainage and treat with daily antiseptic dressings.
   - If synovial, osseous or tendinous involvement is suspected (especially when with penetration of middle third of the frog), emergency surgical debridement under general anaesthesia is warranted ('Street nail procedure').
2. When synovial penetration or infection is confirmed:
   - Navicular bursoscopy, in combination with a focal debridement of the penetrating tract, is the treatment of choice for punctures involving the navicular bursa.
   - Arthroscopic lavage of the DIP joint or DFTS in combination with navicular bursoscopy if either cavity involved.
   - Alternatively, a 'Street Nail' procedure consists of solar fenestration of all affected tissues under general anesthesia to allow for aggressive surgical debridement and adequate drainage.
   - Aftercare consists of daily antiseptic dressings under sterile bandage. If the infection is eliminated, purulent discharge ceases, and the defect gradually seals with healthy, pink granulation tissue.

Prognosis

- Prognosis depends on the extent of the injury and the surgical procedure performed. Prognosis is always guarded if there is synovial or tendinous involvement.
- In one report 95% of horses with punctures outside the frog or sulci returned to full athletic function. On the other hand, only 50% of horses survived if a puncture of the sole had occurred within the boundaries of the frog.
- Following bursoscopic lavage and debridement, survival may be as high as 80% and a return to previous levels of exercise as high as 60% for horses with puncture wounds of the navicular bursa.
- The 'street nail' operation should be regarded, at best, as a salvage procedure to achieve pasture soundness. In one report, only 30% of horses survived following a 'street nail' operation. Horses presented within 7 days of injury and those with a hind limb rather than forelimb injury had a better prognosis.

Figure 16.4 'Quittor' – necrosis of the ungular cartilages. Note the presence of a discharging, non-healing ulcer proximal to the coronary band. This should be distinguished from discharge at the coronary band which is more usually due to extension of a subsolar abscess.

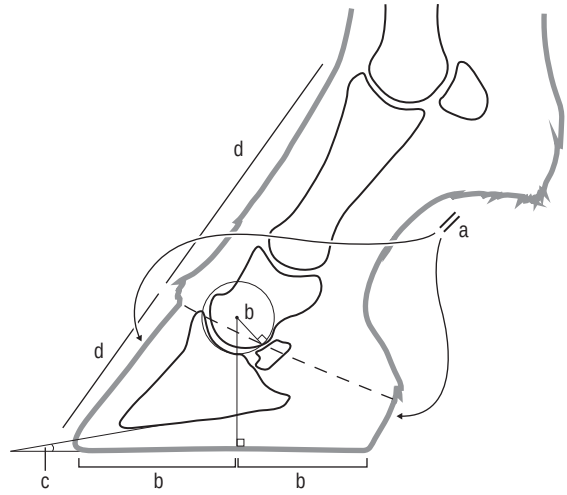


Figure 16.5 Line drawing showing the 'ideal' dorsopalmar foot balance. (a) The angles of the heel and dorsal hoof wall are parallel. (b) The centre of a circle drawn around the distal interphalangeal joint bisects the weight-bearing surface of the foot. (c) The downward angle of the distal phalanx should be 2–3°. (d) The angle of the pastern should be parallel to the angle of the dorsal wall of the hoof ('foot-pastern axis').
Courtesy of Ware R and Colles C.

Quittor/necrosis of ungular cartilage

Definition

Localized necrosis within an ungular cartilage of the distal phalanx. Purulent discharge and sinus formation proximal to the coronary band ensues.

Aetiology

- External trauma (laceration, puncture, bruises from interference) to the lateral heel region of the foot proximal to the coronary band.
- Possible extension of subsolar or submural abscess.

Clinical signs

- Chronic, suppurative draining tracts in the heel region proximal to the coronary band that heal intermittently (Figure 16.4).
- Localized pain, heat and swelling in the region of an ungular cartilage.
- Lameness may occur intermittently in the acute stage of infection.
- Chronic fibrosis of the heel and deformity of the hoof wall may occur.

Treatment

Surgical excision of necrotic ungular cartilage via a skin flap above the corornary band and a hole through the hoof wall distal to the coronary band. Care must be exercised to avoid opening and contaminating the palmar pouch of the DIP joint during resection.

Prognosis

Favourable, although recurrence due to incomplete resection is possible.

Lameness due to foot imbalance

Normal conformation

The weight of the horse is transmitted through the limb to the distal phalanx. The distal phalanx is suspended, via interlaminar bonds, from the inner aspect of the hoof wall. To assure even weight distribution around the interdigitating laminae of the distal phalanx and the hoof wall, it is important that the foot is balanced and that the hoof capsule is evenly positioned around the centre of weight-bearing of the foot. This centre of weight-bearing has been equated with the centre of rotation of the DIP joint or with the midpoint of the roughly circular shape of the hoof wall around the distal phalanx (Figure 16.5).

Dorsopalmar imbalance – 'long-toe/low-heeled foot'

(Figure 25.35)

- A 'long-toe/low-heel foot' is a common conformational defect, especially in Thoroughbreds. Because of this inherent conformation, but also as a result of inadequate trimming of the toe, insufficient length to the branches of shoes and long intervals between shoeing, a long-toed foot tends to rock back onto its heels. Consequently, the centre of weight-bearing of the affected foot migrates in a palmar direction towards the heels which increases the relative vertical load on the heels. The heels cannot support this extra weight and collapse under the foot. The heels are no longer parallel with the toe, and the foot adopts a typical 'long toe/low heel' appearance.
- This abnormal foot shape results in abnormal biomechanical forces causing tearing of the dorsal laminae and straining of the DDFT and the supporting ligaments of the navicular bone.
- Typically bilateral forelimb lameness ensues, especially when the horse is moving on hard ground or in circles.
- Treatment of this condition is aimed at restoring even weight-distribution throughout the laminae of the hoof wall. Trimming the toe adequately and regularly avoids abnormal leverage arising from an abnormally long hoof wall. Shoeing with an egg-bar shoe or a seated-out, wide-webbed, open shoe with extra length and width at the heels gives support to overloaded heels.
- Severe imbalance with chronic heel collapse is difficult to correct and carries a poor prognosis for long-term soundness.

Overlong feet

- Long, boxy feet are often seen in European Warmblood horses. If the foot is allowed to grow too long, the centre of weight-bearing gradually moves palmarly towards the heels. This leads to excessive loading of the heels and may cause lameness.
- Treatment consists of regular trimming and shoeing with good heel support (egg-bar shoes or wide-webbed, open shoes with extra heel-length).

Lateromedial imbalance – 'sheared heels'

- 'Sheared heels' result from uneven trimming of the heels or from poor conformation. One heel (usually the medial one) strikes the ground before the other. This results in proximal displacement of this heel. The wall on this side becomes short and upright. The opposite wall expands and flares outwards.
- Continuous tearing and bruising of the sensitive laminae in the compressed heel and breakdown of tissues that connect both heels may result in lameness.
- This condition is best treated with a straight bar shoe. The branches of the shoe are positioned and secured where the hoof wall should be. The centre line of the limb must be situated exactly over the centre of the shoe. The shoe is fitted wide on the compressed wall and tight on the expanded, flared wall. Gradually, with each shoeing, the sole is brought back perpendicular to the long axis of the limb until the ground impact is redistributed evenly over both heels.
- A straight bar shoe minimizes relative movement between both heels and allows the supporting structures to heal.

Fractures of the distal phalanx

Classification

Type I: non-articular fracture of palmar/plantar process.
Type II: sagittal articular fracture from the DIP joint to medial or lateral solar margin.
Type III: midsagittal articular fracture.
Type IV: fracture of the extensor process.
Type V: comminuted fractures or fractures secondary to foreign body penetration or osteomyelitis.
Type VI: non-articular marginal fractures.
Type VII: non-articular palmar/plantar process fractures in foals, originating and ending at the solar margin.

Aetiology

- Concussion combined with twisting as the foot lands, most commonly during flat racing on a hard surface.
- Kicking unyielding objects.
- Stepping on uneven surfaces (e.g. curb, rock, stones).
- Foreign body penetration with/without infection.
- Fractures of the extensor process may be caused by excessive tension on the extensor tendon (i.e. avulsion fracture), direct trauma or impaction of the process against the distal end of the middle phalanx during hyperextension of the DIP joint.
- A separate centre of ossification or osteochondrosis lesion of the extensor process has been proposed. Friesian horses are predisposed to large osteochondral fragments or an ununited extensor process.

Clinical signs

- Articular fractures cause the most severe signs: acute, severe lameness, heat, pain on hoof testers and percussion, increased digital pulses and effusion of the DIP joint.
- Horses with non-articular fractures usually present with less severe lameness and more localized response to hoof testers. Lameness may improve quickly with rest.
- Pain from Type I fractures can be alleviated with a unilateral PDNB. Pain from most other types of distal phalanx fracture can be alleviated only by a bilateral ASNB.
- Haemarthrosis of the DIP joint may suggest the presence of an articular fracture.
- Radiographs are necessary to confirm the diagnosis and determine the type of fracture (Figure 25.20).
- The extensor process of both front feet may have a separate centre of ossification, which may appear radiographically as a fracture fragment (Figure 25.38). A rounded appearance to the fragment suggests a non-traumatic origin. A separate centre of ossification is rarely clinically significant, except when the fragment is very large.
- Fractures of the extensor process may result in enlargement of the extensor process and malformation of the foot (i.e. buttress foot).
- Most type VI fractures occur in conjunction with remodelling of the distal phalanx of horses with chronic laminitis.

Treatment and prognosis

Type I. The foot should be immobilized with a bar shoe with quarter clips or a cast, and the horse stall rested (Figure 16.6). Healing of this fracture takes 3 to 9 months depending on the size of the fragment. The prognosis for soundness is good.

Types II & III

- About 50% of horses treated conservatively with stall rest, foot or distal limb casts, bar shoes with quarter clips or a full-rim shoe return to soundness after 6 to 12 months.
- Surgical treatment by lag-screw fixation may produce better results than conservative treatment, especially in horses over 3 years of age. Time to healing is significantly shorter (3–6 months) following surgical treatment.

Type IV. Surgical removal is indicated if the fragment is the cause of lameness. Arthroscopic removal provides an excellent prognosis if

Figure 16.6 A foot cast used to treated distal phalanx fractures and instability of the hoof capsule. The inner layer consists of a resin-based casting material applied directly to the foot, taking care to avoid impinging on the soft parts of the heels, and the coronary band, and the outer later is a harder-wearing thermoplastic casting material.

secondary OA has not yet developed. Large osteochondral fragments may be stabilized with a lag-screw, but even large fragments of up to 15–20% of the distal phalanx have been removed successfully without complications.

Type VI and VII. Treatment consists of stall rest (4 to 8 weeks) and surgical shoeing (wide-webbed shoes with anti-concussion pads. In foals (<6 months of age) stall rest or small round pen turnout is typically sufficient to allow healing. Glue-on cuff shoes may be used but must be monitored carefully due to the potential for rapid development of an abnormal foot shape.

Successful fracture healing is accompanied by an improvement in lameness and response to hoof testers. Radiographic follow-up examinations may provide limited information as fractures of the distal phalanx frequently heal by fibrous union. Persistent lameness can be managed by palmar/plantar digital neurectomy.

'Pedal osteitis'

Definition and aetiology

- The term 'pedal osteitis' defines a reaction of the distal phalanx in response to a primary insult. Such potential insults to the bone are penetrating injuries to the foot with or without infection, subsolar bruising, septic pododermatitis, laminitis, keratomas and surgical implants.
- Radiographic characteristics of osteitis include localized osteolysis, sclerosis, bone fragmentation, bone sequestration and new bone formation.
- Generalized demineralization of the distal phalanx, widening and increase in number of the vascular channels and roughening of the solar margin have also been associated with pedal osteitis in horses with flat and thin soles. This foot conformation is not uncommon in Thoroughbreds or Thoroughbred crossbreeds. The significance of these radiographic findings in not always clear as it has been shown that individual variation exists in the number and size of vascular channels of the distal phalanx.

Clinical signs

- Persistent lameness in spite of treatment of the primary insult. The character and severity of lameness are determined in part by the underlying condition (e.g. penetrating injury to the foot, subsolar bruising, septic pododermatitis, laminitis, keratoma, implant intolerance) and in part by the amount of secondary damage to the distal phalanx.
- Horses with persistent lameness following treatment of penetrating injuries to the foot, subsolar bruising, septic pododermatitis, laminitis, keratomas and implant surgery, should be evaluated radiographically to determine if the distal phalanx shows remodelling changes.

Treatment

- Treat the primary cause.
- Sequestered fragments from the distal phalanx should be surgically removed. Aftercare consists of daily antiseptic dressings under a sterile bandage until the infection is cleared and the defect has filled and keratinized.
- Removal of up to 25% of the distal phalanx has been described without negative side-effects.
- Anti-concussive shoeing and management.

Prognosis

- The prognosis is usually favourable for horses with chronic remodelling of the distal phalanx due to chronic subsolar infection.
- Flat-footed horses with chronic foot soreness and chronic remodelling of the distal phalanx may require life-long attention to minimize concussion (anti-concussive pads, selection of exercise surface, etc.)

Bone contusion of the distal or middle phalanx

Definition

Bone contusion or bruising is an MRI diagnosis characterized by the presence of a fluid-like signal in bone. The aetiology is uncertain, but impact trauma is considered to be the most likely cause and may be one of the 'pedal osteitis' pathologies.

Location

Bone contusions of the phalanges occur mostly in the region of the palmar process of the distal phalanx and the dorsodistal aspect of the middle phalanx. Bone bruising of the dorsodistal aspect of the middle phalanx usually involves a well-circumscribed area of cancellous bone adjacent to the dorsal half of the distal interphalangeal joint and can be located axially or abaxially in the middle phalanx.

Clinical signs

- Lameness is usually acute in onset.
- Severity of lameness may vary between mild and severe.
- Lameness is abolished with a PDNB (distal phalanx) or ASNB (middle phalanx). Lameness from bone contusions of the middle phalanx may also improve significantly following intra-articular anesthesia of the DIP joint.

Diagnosis

Bone contusions are identified on fat-suppressed MR images as intraosseous signal hyperintensity. There is usually associated increase in radiopharmaceutical uptake on scintigraphic images. Results of other imaging modalities are normal.

Treatment

Treatment should consist of stall rest, administration of NSAIDs and potentially tiludronate to limit bone resorption and suppress bone inflammation. The prognosis is usually excellent if the subchondral bone plate is not involved.

Osseous cyst-like lesions of the distal phalanx

Aetiology

The aetiology of osseous cyst-like lesions is unclear. Both traumatic and developmental theories have been suggested.

Clinical signs

The affected horse may have a history of acute-onset, severe lameness (most frequently in a forelimb) or of moderate chronic, insidious lameness. Lameness tends to improve with rest but recurs with exercise.

- Substantial improvement with PDNB or intra-articular anaesthesia of the DIP joint and sound with ASNB.
- Radiographs show a circular radiolucent lesion with a sclerotic border adjacent to the subchondral bone plate of the DIP joint. The cyst-like lesion usually communicates with the DIP joint.

Treatment

- Conservative treatment with box rest is usually unrewarding.
- Intra-articular medication with a corticosteroid may result in temporary resolution of lameness.

- Surgical treatment consisting of extra-articular debridement through the hoof wall has been successful in select cases, but requires intra-operative imaging and the morbidity of the procedure can be high.
- Much better results have been reported following arthroscopic debridement of the cyst-like lesion in young horses if it can be reached athroscopically.
- Alternatively the cystic cavity can be injected with a corticosteroid under arthroscopic guidance. The more palmar/plantar the cyst is located on the articular surface of the distal phalanx, the more difficult it is to reach it with a needle or curette.

'Sidebone'

Definition

Sidebone is a term used to describe ossification in the ungular cartilages of the foot.

Aetiopathogenesis

- Ossification of ungular cartilages may be associated with poorly balanced feet and uneven loading of the heels.
- It is thought to have a genetic predisposition in Icelandic horses.
- Acute trauma or chronic repetitive trauma (e.g. lacerations, punctures, chronic bruising from interference) may lead to chronic inflammatory changes and osseous metaplasia of the cartilage.
- Ossfication is common in cob types and horses with a low height to bodyweight ratio.
- Ossification usually proceeds from distal to proximal. Uncommonly ossification starts within the ungular cartilage and progresses centripetally to form a separate centre of ossification. Usually the lateral ossified ungular cartilage is larger than the medial one.
- Fractures involving sidebones are rare and if present usually occur at their distal extent, close to the palmar/plantar process of the distal phalanx. An ununited, large separate centre of ossification can resemble a fractured sidebone.
- In a recent retrospective study, severe sidebone formation was associated with concurrent collateral ligament desmitis of the DIP joint.

Clinical signs

- Lameness is rare but may occur in the early phase of ossification. Lameness may also be seen following local injury or fracture of a sidebone.
- There is a prominent, hard, rigid and bony enlargement proximal to the coronary band at the level of the quarter.
- Radiography shows the extent of ossification (Figure 25.39). A radiolucent line may be present between the palmar process of the distal phalanx and the base of the ossified cartilage and should not be interpreted as a fracture line. Nuclear scintigraphic examination may aid in the differentation between a separate centre of ossification and a fractured sidebone.

Treatment

- Treatment is usually unnecessary because it is not usually a cause of lameness.
- The feet should be kept properly balanced to distribute weight-bearing evenly.
- A bar shoe may be helpful if lateromedial foot imbalance is present.

Fractures of the navicular bone

Incidence and location

Three types of fractures may affect the navicular bone:

- Chip fracture involving the proximal or distal border or the wings. Osseous fragments of the distal border may also be characterized as separate centres of ossification or dystrophic mineralization within the impar ligament.
- Simple sagittal fracture close to the sagittal ridge (Figure 16.7).
- Comminuted fractures are uncommon and are usually displaced.
- Congenital non-fusion of centres of ossification of the navicular bone may be confused with fracture. The navicular bone may have 2 or 3 separate centres of ossification.

Aetiology

- Sagittal and comminuted fractures result from acute trauma to the foot such as from running on hard unlevel ground, stepping on to a curb stone or kicking at stall walls with the hind limbs.
- Chip fractures may be avulsion fractures, located within the impar ligament or navicular suspensory ligaments.

Clinical signs

- Sagittal or comminuted fractures cause sudden-onset, severe lameness.
- Walking improves greatly within 2 weeks with stall rest.
- Positive response to a PDNB.
- Radiography provides a certain diagnosis (Figure 25.21). Fractures are easily confused with packing defects of the frog clefts. Always include a flexor view (Figure 16.7).
- The fracture normally develops fibrous union or non-union and, therefore, remains permanently visible.
- Radiographic features of a chronic fracture are remodelling along the proximal border and osteolytic changes along the fracture line often of cyst-like appearance. As the DIP joint is involved, OA of the DIP joint becomes established early on, and permanent lameness ensues. The prognosis for soundness is poor.

Treatment and prognosis

- Conservative treatment using bar shoes, quarter clips, raised heels and stall rest (6 months) is generally unsuccessful as it results in non-union and osteoarthritis of the distal interphalangeal joint.
- The only way to obtain bony union is by interfragmentary compression, using lag-screw fixation. The more recent the fracture, the better the prognosis for soundness. Internal fixation may avoid the onset of OA of the DIP joint.
- Pain caused by chip fracture of the tip of the wing of the navicular bone, can be alleviated by a navicular suspensory ligament desmotomy.
- Palmar digital neurectomy may be useful in horses that fail to regain soundness after internal fixation. It does not provide long-term resolution of lameness in conservatively treated horses because of the onset of osteoarthritis of the distal interphalangeal joint.

Navicular disease

Definition

Navicular disease is a chronic degenerative disease of the navicular bone, the fibrocartilage of its flexor surface, the *ligamenta sesamoidale collateralia* (navicular suspensory ligaments) and the *ligamentum distale impar* (impar ligament). Concurrent degenerative change of the DDFT and the navicular bursa is common.

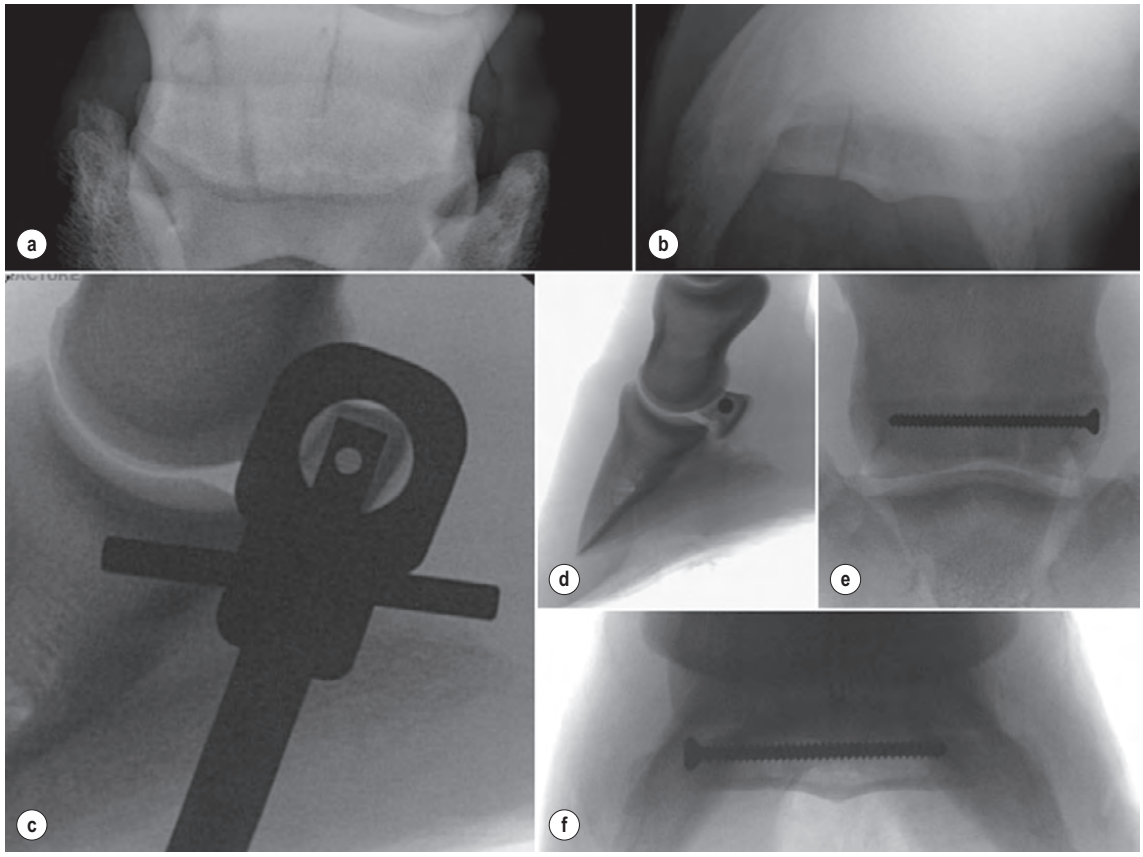


Figure 16.7 Lag-screw fixation of a navicular bone fracture. (a) Dorsoproximal-palmarodistal oblique and (b) palmaroproximal-palmarodistal oblique radiographs of a parasagittal fracture of the navicular bone. The latter view is essential to avoid the misdiagnosis of the presence of a fracture due to an overlying frog shadow on the dorsoproximal-palmarodistal oblique view. (c) Intra-operative fluorographic image of the correctly positioned jig for insertion of the lag screw. Post-fixation – (d) lateromedial, (e) dorsoproximal-palmarodistal oblique and (f) palmaroproximal-palmarodistal oblique fluorographic images showing accurate placement of the lag screw which is compressing the fracture.

Pathology

There are four basic manifestations of navicular disease based on pathological changes and the MRI appearance of lesions:

1. 'Classical' navicular disease: degenerative changes are present in the palmar fibrocartilage and flexor cortex of the navicular bone. This is usually accompanied by degenerative changes of the bursal part of the superficial layer DDFT and inflammatory changes of the navicular bursa. Secondary medullary changes include trabecular remodelling, focal osteolysis, medullary fibrosis, congestion and oedema.
2. Distal border fragments and navicular bone remodelling: osseous fragments occur at the lateral or medial angles of the distal border. Remodelling of the parent navicular bone usually results in lameness. Not all distal border fragments result in navicular disease and lameness.
3. Medullary necrosis: rare cases of primary medullary injury with evidence of oedema and necrosis of the medullary adipose tissue have been reported.
4. Desmitis or entheseopathy of the navicular suspensory ligaments or impar ligament can be a cause of navicular lameness, but desmitis and entheseopathy are less common than the osseous abnormalities.

Aetiology and pathogenesis

There are two basic theories regarding the cause of the various pathological changes reported in navicular disease: the vascular theory and the biomechanical theory.

1. The vascular theory suggests that the main nutrient arteries in the distal part of the navicular bone develop arteriosclerosis and thrombosis, which results in ischaemic necrosis and bone resorption following reperfusion. However, histologic and morphometric data indicate that increased bone turn-over occurs rather than ischaemia and necrosis.
2. The biomechanical theory is based on the presence of abnormal mechanical forces between the DDFT and the flexor surface of the navicular bones in some horses, resulting in progressive degenerative changes of the flexor surface and the spongiosa. Biomechanical data have indicated that in navicular disease cases, the force on the navicular bone is increased by contraction of the deep digital flexor tendon in the early phase of the stride. Histological evidence has revealed these changes to be similar to the subchondral bone and spongiosa changes of OA in low-motion joints. These changes include initial generalized sclerosis and thickening of the subchondral bone plate, followed by focal bone destruction with replacement by granulation tissue and fibrosis with venous hypertension in the spongiosa.

Clinical signs

- Typically in middle-aged horses 7 to 12 years old, but all ages are affected.
- Most common in Thoroughbred-cross horses and Warmbloods; rare in Arabian horses or ponies.

- The onset of lameness is most commonly associated with an irregular workload or a period of enforced rest.
- Lameness is mostly insidious in onset and intermittent initially. The horse is noticed to be stiff when leaving the stall or at the beginning of exercise. The horse frequently stumbles, is reluctant to work in circles, and is either reluctant to jump or rushes jumps. During the early stages of the condition, horses tend to warm out of the lameness.
- Horses may be seen to point one foot at rest or to heap up the bedding in order to stand with the heels elevated.
- The trot is bilaterally short-strided, with a shortened cranial phase, and the foot lands toe-first. Lameness is mostly bilateral, but one limb is generally more painful than the other, which results in a visible head-nod.
- Lameness may switch between limbs when a horse is exercised in different directions on a circle or following nerve blocks.
- Lameness appears worse on hard ground.
- Distal limb flexion test generally exacerbates the lameness. Hyperextension of the DIP joint with a wooden wedge (toe-elevation test) may also increase the severity of lameness.
- The digital arteries are often palpably enlarged without increased pulsation.
- Heat, pain and swelling are not present. The pain response to application of hoof testers is variable. Generally there is no remarkable response, except when the testers are applied across the middle third of the frog in some small-footed horses. Pain may also be elicited with hoof testers if navicular disease is accompanied by poor foot balance.
- Diagnostic anaesthesia: A clinical diagnosis of navicular disease can be made only if lameness is significantly improved by at least 2 of the following 3 local anaesthetic techniques:
  1. Palmar digital nerve block.
  2. Intra-articular anaesthesia of the DIP joint.
  3. Intrabursal anaesthesia of the navicular bursa.

Some horses (approximately 20% of those positive to a PDNB) improve significantly more to intrabursal anaesthesia than to intra-articular anaesthesia. This means that a negative response to intra-articular anaesthesia of the DIP joint does not always exclude a diagnosis of navicular disease.

Radiology

The radiographic criteria for diagnosis of navicular disease have historically been based on the appearance of the synovial invaginations along the distal border of the navicular bone (Figure 25.40), as seen on the upright pedal or high coronary projection. As there is a wide variation in the number, shape and size of these synovial invaginations in horses without lameness, this approach is unreliable. Alternative, more reliable radiographic signs can be used to support the diagnosis of the navicular disease.

On the lateromedial projection:

1. An ill-defined radiolucent area in the flexor cortex (classic navicular disease).
2. Changes in shape (distoproximal lengthening) of the navicular bone due to peripheral new bone formation.
3. Decreased radiopacity and thinning of the flexor cortex in working horses.
5. Medullary sclerosis.
6. Loss of corticomedullary distinction.
7. Calcification of the impar ligament, navicular suspensory ligaments or DDFT.

On the upright pedal projection:

1. A central radiolucent area (classic navicular disease) (Figure 25.38).
2. Ill-defined radiolucency of the distal border surrounded by sclerosis.
3. Proximal or distal elongation of the navicular borders with ill-defined deposits of new bone.

On the flexor view:

1. Cavitation of the flexor cortex (classic navicular disease) (Figure 25.41).
2. Alterations to the well-defined contour of the flexor cortex.
3. Decreased radiopacity of the flexor cortex.
4. Loss of a distinct interface between the cortex and spongiosa.
5. Medullary sclerosis.

It should be remembered that radiography is a relatively insensitive imaging technique and that the navicular bones may have a normal radiographic appearance in horses with navicular disease. Equally, diagnosis of navicular disease cannot be based solely on the presence of radiographic abnormalities. It is especially impossible to use the radiographic appearance of the distal border of a navicular bone to predict whether a horse will develop navicular disease. This practice should be strongly discouraged during interpretation of pre-purchase radiographs.

The identification of increased radionuclide uptake in the region of the navicular bone with nuclear scintigraphy may indicate the presence of abnormal bone turn-over in horses with navicular disease and normal radiographs.

A definitive imaging diagnosis of navicular disease often requires the use of MR imaging. MR imaging is able to show abnormalities of the navicular apparatus that cannot be identified radiographically, such as fibrocartilage erosions, oedema and trabecular remodelling in the medullary cavity, remodelling of the flexor cortex and osseous fragments of the distal border, as well as soft-tissue injuries.

Treatment and prognosis

The wide variation in response to different treatment regimens makes case selection for any of the available treatment options difficult.

1. Therapeutic foot care to encourage a more upright conformation of the foot, a straight hoof-pastern axis and downward growth of the heel.
   - Egg-bar shoes.
   - Wide-webbed, seated-out shoes.
   - Rolled toe, raised heel shoes.
2. Medicinal therapy.
   - Phenylbutazone, 1 to 2 g daily, allows the horse to be exercised during treatment. Enforced rest is generally contra-indicated for horses with navicular disease.
   - The β-adrenergic drug, isoxsuprine hydrochloride, increases peripheral circulation by relaxing the vascular smooth musculature. After the horse has been sound for at least 2 weeks, the drug is gradually withdrawn. Bioavailibility studies have questioned the oral absorption of this drug.
   - Intrabursal or intra-articular infiltration of corticosteroids is a palliative treatment aimed at temporarily controlling inflammation and lameness associated with navicular disease. Of treated horses, 60% are sound for 2 months or more, but less than 10% are sound 12 months after injection. Intrabursal injection is more effective but also more invasive and technically more difficult than intra-articular injection. Repeat injections are less effective, and horses eventually become unresponsive to corticosteroid infiltrations. Intrabursal corticosteroids may result in degeneration of the DDFT.
   - Tiludronate (Tildren®) is a bisphosphonate that reduces osteoclast activity. It has been administered to prevent progression of osteolysis and suppress osseous inflammation.

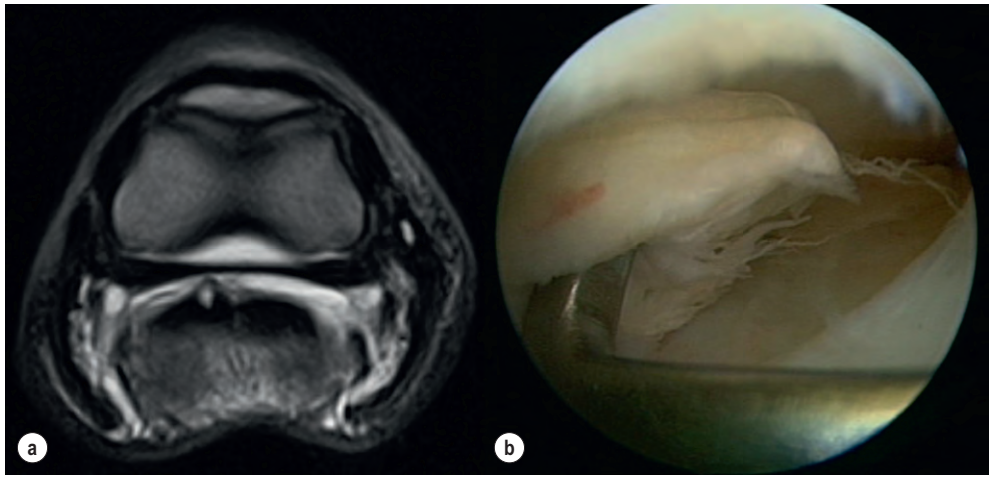


Figure 16.8 Standing transverse MR image (T2 weighted) (a) showing a small dorsal surface defect in the deep digital flexor tendon within the navicular bursa, immediately proximal to the navicular bone, which is amenable to bursoscopic examination and debridement (b).

3. Surgical treatment.
   - Desmotomy of the suspensory ligaments of the navicular bone is performed to reduce the abnormal mechanical load between the DDFT and the flexor surface of the navicular bone. Improvement in lameness is achieved in approximately 50% of horses.
   - Neurectomy of the palmar digital nerves is a palliative treatment of last resort to provide symptomatic relief in horses with navicular disease. Desensitization of the palmar structures of the foot allows the horse to exercise pain-free for approximately 2 years. Complications of this treatment result in recurrence of lameness and are often career-ending. They include re-innervation of the foot, painful neuroma formation, tearing of the DDFT, exungulation of the hoof as a sequel of unrecognized and untreated infection. Neurectomy of the palmar digital nerves is contra-indicated in horses with distal tendinopathy of the DDFT.
   - Some equestrian competition jurisdictions do not permit neurectomy procedures.

Distal tendinitis of the deep digital flexor tendon (DDFT)

Introduction

Since the advent of MRI, it has become clear that tendinitis of the DDFT is the most common soft-tissue injury causing lameness in the horse's foot. Tendinitis of the digital portion of the DDFT may occur alone, in conjunction with navicular bone disease or as one component of a complex of a multiple soft-tissue injuries in the foot. In a review of 199 referred cases of foot lameness without radiographic or ultrasonographic abnormalities, there was a prevalence of 33% for primary tendinitis and 59% for all abnormalities in the DDFT.

Pathology

There are four different types of lesion in the digital portion of the DDFT: core lesions, dorsal border lesions, sagittal plane splits, and insertional lesions (entheseopathy).

- Core lesions are characterized by fibre disruption in the centre of usually one lobe only and span the upper extent of the T-ligament into the proximal recess of the navicular bursa but can extend a variable distance proximodistally, occasionally causing a continuous core defect from the insertion up to the level of the distal aspect of the proximal phalanx.
- Sagittal plane splits occur mostly within the navicular bursal portion of the DDFT, at the level of the flexor surface of the navicular bone and can extend proximodistally (Figure 16.8).
- Insertional lesions are limited to the most distal 10–20 mm of the DDFT near its insertion on the distal phalanx. They consist of small core lesions, sagittal plane splits or osseous changes of the insertion site.
- Lesions of the dorsal border of the DDFT are mainly found in the navicular bursa and consist of superficial fibrillations or diffuse lesions communicating with the dorsal margin of the DDFT. Lesions of the dorsal border are commonly accompanied by degenerative changes of the palmar surface of the navicular bone.

Any of these types of lesion may occur alone or in combination with others.

Aetiopathogenesis

- Repetitive overload stress or acute traumatic tearing.
- Predisposition to injury may increase with degenerative ageing changes in the DDFT.
- Possible risk factors include hoof conformation, the angle of the distal phalanx, the horse's athletic discipline and previous palmar digital neurectomy. Horses jumping over fences have a significantly higher risk of injuring the digital portion of the DDFT than horses that are used for other sporting endeavours. Horses that have undergone a palmar digital neurectomy are at significant risk of deterioration of existing DDFT abnormalities.

Clinical signs

- Generally no visible or palpable abnormalities except for occasional distension of the DFTS or focal, painful, firm soft-tissue swelling on the palmar aspect of the DDFT in the distal aspect of the pastern.
- Elevation of the toe, and sometimes the heel, with a wooden wedge may exacerbate lameness.
- Approximately 50% of horses with distal tendinopathy are presented with an acute-onset, moderate to severe lameness while the other 50% experience a more insidious disease progression.
- Lameness is generally accentuated by hard work but improved by rest.

- Many horses are seen pointing the affected foot intermittently at rest.
- Some horses with DDFT tendinitis appear to be lamer on soft ground than on hard ground, especially when the affected limb is on the outside of the circle.
- Approximately 60% of all horses with digital tendinitis is only lame in one limb, while the remaining 40% are bilaterally lame either before or after diagnostic anaesthesia of the lamer limb.
- Lameness can be significantly improved in 70% of horses with either a PDNB, intra-articular anaesthesia of the DIP joint or intrabursal anaesthesia. An abaxial sesamoid nerve block is usually required to abolish lameness completely. It has been suggested that intrathecal anesthesia of the digital flexor tendon sheath may provide a way to desensitize the digital portion of the DDFT without anaesthetizing simultaneously other parts of the foot.

Imaging

- Radiography, ultrasonography and scintigraphy are generally normal. Ultrasonographic abnormalities are rarely present at the level of the pastern, when the DDFT lesion progresses proximally beyond the level of the navicular bone.
- Distal tendinitis of the DDFT in the foot is predominantly an MRI diagnosis (Figure 25.69). Tendon damage is seen as focal signal increase on both T1- and T2-weighted sequences (Figure 16.8), variably accompanied by swelling of the affected lobe in the acute stages of injury. There is a good correlation between the MRI appearance of DDFT lesions and their pathological classification into core lesions, sagittal plane splits, insertional lesions and dorsal surface erosions. In the chronic stages of healing by fibrosis, signal intensity in core lesions generally decreases in T2-weighted sequences but stays high in T1-weighted sequences.
- Distal tendinitis has also been diagnosed with CT. Contrast enhancement on CT images may differentiate between acute and chronic injuries.

Treatment

- The most important aspect of treating injuries of the DDFT in the foot is rest. Rest should minimally consist of stall confinement for 6 months, including low-grade maintenance exercise like daily in-hand walking for 10–15 minutes.
- Because of the poor outcome after rest alone, several additional or alternative therapies have been tried with variable results:
  1. Corrective trimming (shortening of the toe) and remedial farriery (application of graduated bar shoes) to ease break-over and reduce tension on the DDFT.
  2. Injection of a corticosteroid into the digital flexor tendon sheath or navicular bursa.
  3. Application of extracorporeal shockwaves to the DDFT.
  4. Intralesional injection, under CT or bursoscopic control, of acellular urinary bladder matrix, the vascular stromal fraction of fat, bone-marrow-derived mesenchymal stem cells or bone-marrow concentrate.
  5. Inferior check ligament desmotomy.
  6. Bursoscopic debridement of prolapsing torn fibres from the damaged dorsal surface of the DDFT.

Prognosis

- Horses with primary injury of the DDFT have a guarded prognosis for return to athletic function if treated conservatively (approximately 30%). If the injury is accompanied by navicular disease the outcome is considered poor.
- Following surgical debridement via bursoscopy, full return to athletic function has been reported in approximately 60% of horses with dorsal margin lesions of the DDFT.
- The effect of stem-cell therapy is currently unknown.

Collateral desmitis of the distal interphalangeal (DIP) joint

Introduction

Collateral desmitis of the DIP joint has long been ignored as a potential cause of foot lameness because of the difficulties associated with imaging the collateral ligament. MRI findings suggest that collateral desmitis is the second most important soft-tissue injury responsible for foot lameness.

Aetiology

- Acute traumatic or chronic repetitive strain injury in the front feet.
- The medial CL is injured almost twice as often as the lateral CL. In a small proportion of horses, both the lateral and medial CLs are affected.

Pathology

- Entheseopathy at the bone/ligament interface of the origin or insertion of the collateral ligament can result in an osteolytic or an osteoproliferative response.
- Desmitis/desmopathy of the mid portion of the collateral ligament consists of mild to moderate fibre damage (sprain) or severe fibre damage (partial or complete rupture with subluxation or luxation).
- OA as a sequel of collateral desmitis is uncommon unless the damage is so severe that it creates instability in the DIP joint.

Clinical signs

- Horses used for jumping are at increased risk.
- Acute injury is rarely reported in the history.
- Obvious localizing signs are rare.
- Mild to moderate foot lameness of more than 2 months' duration that has all the hallmarks of palmar foot pain without radiographic abnormalities. Lameness is worsened when trotting in a circle.
- Improvement to diagnostic anaesthesia is variable: 24% of horses improved following intra-articular anaesthesia of the DIP joint, 72% of horses to a palmar digital nerve block and 100% following an abaxial sesamoid nerve block.

Imaging

- Osteoproliferative or osteolytic changes in some cases of entheseopathy.
- Ultrasonography is difficult and limited to the proximal 25% of the collateral ligament, and artefacts are common.
- Scintigraphic hot spots may be present at the insertion site of collateral ligament in horses with entheseopathy. Scintigraphy has a low sensitivity but a high specificity.
- MRI shows increased cross-sectional area, irregular contour and increased signal intensity of affected collateral ligament. Damage at the origin or insertion of a collateral ligament causes signal alteration in the adjacent bone.

Treatment

- Correction of foot imbalance.
- Application of shoes with a rolled toe or 'Denoix' shoe with a double width web on the side of the affected collateral ligament (Figure 16.9).

Figure 16.9 The use of a wide medial branch of the shoe to treat a medial collateral ligament injury of the DIP joint. When the foot is placed on *soft ground* (e.g. a sand school) the wider medial branch does not sink in as far and so protects from excessive stretching of the medial collateral ligament.

- Focal shock wave therapy.
- Restricted exercise for 3–6 months.
- Intra-articular injection of the distal interphalangeal joint with a corticosteroid is generally contra-indicated because it slows soft-tissue healing although it may be beneficial for treating concurrent synovitis, as can Interleukin-1 receptor antagonist protein (IRAP) which avoids the adverse effects of coriticosteroids on ligament healing.

Prognosis

- Approximately 30% of horses make a full recovery with long-term rest.
- Outcome may be improved after shockwave therapy and intra-articular medication.

Traumatic and degenerative arthritis of the DIP joint

Aetiology

- OA secondary to osseous cyst-like lesions, septic arthritis, intra-articular fractures, sprains and luxations.
- Traumatic synovitis of the DIP joint is a significant cause of racing lameness, particularly in the American Quarterhorse.

Clinical signs

- Insidious onset, mild to moderate, bilateral forelimb lameness. Most obvious on hard surfaces, on circles, and after flexion tests.
- Acute unilateral lameness with localizing signs in case of sprain.
- Frequently palpable effusion of the DIP joint just proximal to the dorsal aspect of the coronary band.
- Arthrocentesis may reveal increased fluid pressure and decreased synovial viscosity.
- Lameness resolves after intra-articular anaesthesia.
- Radiographic abnormalities are uncommon in horses with synovitis. Signs of OA or chronic capsulitis are uncommon. Enlargement of synovial invaginations in the distal border of the navicular bone, traditionally interpreted as the cardinal radiographic sign of 'navicular disease' may be more indicative of synovitis of the DIP joint.

Treatment

- Treat primary condition (e.g. sprain, chip fracture, subchondral bone cyst)
- Horses with synovitis, capsulitis and OA can be medicated with intra-articularly administered drugs (PSGAG, sodium hyaluronate, corticosteroids).
- Alternatively repeated injections of IRAP obtained from the horse's own blood may be helpful, especially if previous intra-articular administration of corticosteroids has failed to improve lameness.
- Associated foot imbalances must be corrected with appropriate trimming and shoeing.

Prognosis

Horses with radiographic evidence of OA of the DIP joint have a poor prognosis for return to soundness.

Septic arthritis of the DIP joint

Aetiology

- Penetrating injuries to the joint.
- Intra-articular injection.
- Neurectomized horses may develop septic arthritis from expansion of an unnoticed subsolar abscess.

Clinical signs

- Severe, non-weight-bearing lameness.
- Hot, painful swelling proximal to the coronary band.
- Analysis of synovial fluid to support septic arthritis.

Treatment

- Treatment must consist of aggressive joint lavage in conjunction with intra-articular and systemic antimicrobial therapy. Lavage should be performed via both the dorsal and palmaro/plantaroproximal pouches of the joint.
- With chronic damage to cartilage or bone, or if treatment is unsuccessful in resolving infection, ankylosis of the DIP joint can be attempted to obtain pasture (breeding) soundness with permanent mechanical lameness.

Prognosis

Horses with infection of the DIP joint have a poor prognosis for return to function.

Flexural deformity of the DIP joint

Aetiology and pathogenesis

- Flexural deformities of the DIP joint are caused by relative shortening of the musculotendinous unit of the DDFT. These deformities may be congenital or acquired.
- Congenital flexural deformities of the DIP joint have been associated with intra-uterine malpositioning, genetic factors and teratogenic drugs. Congenital rupture of the common digital extensor tendon (CDET) may also lead to this deformity.

Figure 16.10 Flexural deformity of the DIP joint. This case would be classified as a Stage II as the dorsal wall is past the vertical such that weight-bearing tends to flex the distal interphalangeal joint rather than extend it. These cases therefore require surgical intervention, such as transection of the accessory ligament of the deep digital flexor tendon or the deep digital flexor tendon itself, and shoeing with toe extensions.

- Acquired flexural deformities of the DIP joint typically develop in foals of weaning age and may be a manifestation of developmental orthopaedic disease (DOD). Contributing factors are pain causing a flexion withdrawal reflex, lack of exercise, rapid growth and nutritional imbalance. Imbalance between longitudinal growth of the skeleton and elongation of the musculotendinous unit of the DDFT has been hypothesized.

Clinical signs

- Tight DDFT, detected by palpation.
- In the acute stage, the foal may only be able to walk on the tip of the toes, without heel-ground contact ('ballerina foal') (Figure 16.10).
- In the subacute stage, the foot adopts a boxy appearance ('club foot', 'buck hoof') because of the lack of heel wear.
- Flexural deformity of the DIP joint can be recognized in two stages:
  *Stage I, in which the dorsal wall of the foot does not pass beyond the vertical.
  *Stage II, in which the dorsal wall of the foot passes beyond the vertical (Figure 16.10).

Treatment

Congenital form:

- Most improve spontaneously within first week of life.
- Intravenous administration of oxytetracycline.

Acquired form, Stage I:

- Attempt to slow growth by dietary management.
- Exercise/physiotherapy with judicious use of analgesic drugs.
- Application of a toe extension and trimming of the heels. The toe needs to be protected against excessive wear and tearing of the dorsal hoof wall with a shoe.
- Paradoxically application of a wedge to elevate the heel may reduce pain in foals with acute 'ballerina syndrome' and induce relaxation of the DDF muscle.
- If the deformity is unresponsive to conservative management, desmotomy of the inferior check ligament is a valuable option.

Acquired form, Stage II:

- Desmotomy of the accessory ligament of the deep digital flexor tendon.
- DDFT tenotomy at the middle third of the metacarpus or at the palmar aspect of the pastern if desmotomy of the accessory ligament of the deep digital flexor tendon is unsuccessful.

Prognosis

Prognosis for exercise is favourable, as long as a DDFT tenotomy is not required to achieve normal posture. Most deformities, including stage 1 and 2 manifestations, can be corrected by desmotomy of the accessory ligament of the deep digital flexor tendon. Prognosis in foals with bilateral disease is guarded.

16.2 DISEASES OF THE PASTERN AND FETLOCK REGION

'Ringbone'

Definition

Ringbone is a lay term used to describe any bony enlargement on the dorsal surface of the pastern. This can be caused by periostitis of the proximal or middle phalanx or osteoarthritis (OA) of the PIP joint ('high ringbone') or DIP joint ('low ringbone'). Ringbone may be articular or, more rarely, non-articular.

Phalangeal periostitis

Aetiology

- Trauma from lacerations of brushing.
- Chronic microtrauma to ligamentous and tendinous attachments, especially with poor conformation (i.e. base narrow or base-wide; toed-in or toed-out).
- Extensor tendon strain.

Clinical signs

- Lameness only in acute stage of a soft-tissue strain.
- Sometimes persistent lameness if common digital extensor tendon is involved.

Treatment

Rest and anti-inflammatory drugs.

Osteoarthritis (OA) of the PIP joint

Aetiology

- Usually chronic and idiopathic, with an insidious onset.
- High-speed exercise involving sudden turns and stops that occur in events such as polo, calf roping and other rodeo events, may predispose by damaging collateral ligament support.
- Less frequently, secondary to penetrating wounds, fractures or OCD.

Clinical signs

- Non-specific, chronic lameness.
- Sometimes obvious external signs, i.e. heat and pain if acute, or firm dorsal enlargement of the pastern region.
- Lameness is exacerbated by flexion of the distal limb.
- Lameness is abolished by abaxial sesamoid nerve block.
- Radiographic signs of joint disease.

Treatment

- Exercise the horse, if possible, using anti-inflammatory/analgesic medication.
- Intra-articular injection of the PIP joint with a corticosteroid may provide temporary relief.
- Surgical arthrodesis is usually necessary to reach a pain-free stage of ankylosis. The proximal and middle phalanges are immobilized in compression most optimally provided by a plate in combination with two abaxial lag screws to promote fusion. Cast immobilization is required in the postoperative period.

Prognosis

Between 65 and 85% of surgically treated horses can return to full activity. Results are better for hind limbs than for forelimbs. Complete recovery can take up to 12 months.

'Subluxation' of the pastern joint

Aetiology

Due to the maintenance of joint congruity, a better term is instability of the pastern joint.

- Dorsal subluxation.
  - Sometimes congenital in Arabian horses.
  - Rupture or desmotomy of the suspensory ligament.
  - Reported in hind limbs following transection of the medial head of the DDFT.
- Palmar subluxation.
  - Injury to palmar support structures of the PIP joint (the 4 palmar ligaments, the SDFT branches and the straight distal sesamoidean ligament), e.g. in foals jumping from heights.

Clinical signs

- Intermittent dorsal 'clicking' of the pastern.
- Dorsal convexity or concavity of the pastern following trauma.

Treatment

For cases associated with substantial soft-tissue damage:

- Eight weeks in cast with limb in flexion followed by 6 months of box rest.
- The PIP joint should be stabilized in normal alignment with a surgical arthodesis before scar tissue permanently lengthens or shortens the supporting structures.

Prognosis

For those cases associated with significant lameness, the prognosis is guarded for return to full exercise unless timely arthrodesis can be performed.

Fracture of the middle phalanx

Aetiology and classification

- Fractures of the middle phalanx are most common in the hind limbs of horses used for polo and Western performance.
- Types include chip fractures, wing (palmar/plantar process) fractures and comminuted fractures.
- Fractures of the middle phalanx commonly result from sudden compression, in combination with a twisting force that occurs with sudden stops, starts or short turns.
- Comminuted fractures are more common than wing fractures. Chip fractures are rare.

Clinical signs

- Chip fractures result in acute lameness, but lameness usually improves with rest.
- Wing and comminuted fractures.
  - Acute, severe lameness.
  - Swelling, crepitus, pain.
  - Diagnosis is confirmed by radiography.

Treatment and prognosis

- Chip fractures.
  - Arthroscopic removal of the fragment.
  - Prognosis for return to soundness is good.
- Wing and comminuted fractures.
  - Reconstructable fractures must be accurately reduced and stabilized with a lag screw (wing fractures), or screws and plates (comminuted fractures), to give the horse a realistic chance.
  - For fractures involving the PIP joint that cannot be accurately reduced, or biaxial wing fractures, arthrodesis of the PIP joint is required.
- If both the PIP and DIP joint have been compromised, the prognosis for return to exercise is poor.
- Horses with severely comminuted fractures in which no continuous bony strut remains between the PIP and DIP joints, may be treated using lag screws to reduce the articular deficits followed by application of a transfixation pin cast. Provided the fracture involves a hind limb and the transfixation pin cast can be maintained for more than 40 days, survival is possible.

Fractures of the proximal phalanx

Fractures of the proximal phalanx are among the most common fractures encountered in racehorses. In UK flat racing on turf, fractures of the proximal phalanx are responsible for the highest proportion of fatal distal limb fractures. Various different fracture configurations exist. Treatment options typically involve surgery.

For the purpose of this chapter the following differentiation is used:

Sagittal and comminuted fractures ('split pastern').
Longitudinal frontal fractures.

Chip fractures.
Palmar/plantar process (eminence) fractures.

Sagittal and comminuted fractures ('split pastern')

Aetiology and classification

- These fractures propagate distally from the sagittal groove in the proximal articular surface of the proximal phalanx. They may be caused by the screwdriver effect of the sagittal ridge of the distal end of the metacarpus/metatarsus.
- Sagittal fractures of the proximal phalanx may be:
  - Incomplete: the fracture extends a variable distance distally from the sagittal groove towards the medullary cavity, but does not include the lateral or medial cortex. Incomplete fractures have been further classified as short (<30 mm) or long (>30 mm) depending on their propagating distance.
  - Complete, non-displaced: the fracture most commonly extends sideways from the medullary cavity to exit at the lateral or medial cortex, or less commonly extends into the pastern joint, bisecting the proximal phalanx.
  - Complete displaced: same configuration as above.
  - Comminuted, reconstructable: multiple fracture planes are present. A continuous vertical strut of bone remains between the fetlock joint and PIP joint.
  - Severely comminuted: no bone continuity remains between both joints. These fractures are generally not reconstructable.

Clinical signs

- Signs vary from acute, severe lameness that improves with box rest, to non-weight-bearing lameness, with obvious crepitus and instability.
- Pain, heat and swelling at the level of the pastern.
- Pain on flexion of the fetlock joint.
- Diagnosis is confirmed by radiography (Figure 25.3). Several oblique radiographs are necessary to show distal propagation of the fracture line. The fracture line may remain invisible for several weeks following injury.
- Do not use nerve blocks if a fracture of the proximal phalanx is suspected. Freedom from lameness may turn an incomplete fracture into a complete one.

Treatment and prognosis

- First aid for an incomplete fracture consists of a Robert Jones bandage with lateral and medial splints or a commercial distal limb splint. A complete fracture is best supported by a first aid cast, applied with the horse standing and the affected limb lifted off the ground by an assistant. The metacarpus and phalanges should be vertically aligned with a dorsal splint. A commercial (e.g. Kimzey) splint is an acceptable alternative.
- Incomplete fractures can be treated by cast immobilization and/or lag-screw fixation. Conservative treatment has been recommended for incomplete fractures that are less than 15 mm long. Up to 70% of Thoroughbred racehorses can be returned to racing with conservative treatment, but outcomes have been variable. This contrasts to a 100% success rate for conservative management in non-racehorses. Long incomplete fractures treated surgically result in 65% recovery for racehorses and 33% recovery of non-racehorses. According to one study conservative management of incomplete vertical fractures > 30 mm can result in non-union and delayed catastrophic fracture (even after a year).
- Complete fractures should always be treated by lag-screw fixation or plating.

Even a small amount of displacement of fragments compromises the final outcome, because it often results in permanent damage to the articular suface(s). Up to 71% of horses with fractures that exit laterally have been reported to return to racing. A similar success rate has been reported in non-racehorses. A variable outcome has been reported for return to racing with treatment of complete sagittal fractures that extend to the PIP joint (16–55%).

- Horses should be recovered from general anaesthesia in a cast. If the fracture is not comminuted, the cast can be removed after 7–14 days.
- Horses with comminuted fractures of the proximal phalanx can be salvaged for breeding, using a combination of external and internal fixation as well as cast immobilization. If no strut of bone remains between the fetlock and PIP joint, the horse's prognosis for survival is guarded although one recent study reports survival of 60% of horses with use of a specialized external skeletal fixator in support of the internal fixation.

Longitudinal frontal fractures

Treatment

- Horses with incomplete, non-displaced fractures can be managed with 6 months of box rest or cast immobilization for 6 weeks followed by 6 weeks of box rest.
- Complete fractures, with or without displacement must be accurately reduced and stabilized with lag screws or dynamic compression plates.
- For fractures into the distal articulation that cannot be accurately reduced, arthrodesis of the PIP joint is indicated.

Prognosis

The prognosis for athletic function depends on the degree of damage to the fetlock joint but is similar to sagittal fractures.

Chip fractures

Aetiology and location

- Overextension and concussion, especially in fast-gaited (race) horses.
- Chip fractures occur at the proximal, dorsal or proximal palmar/plantar articular margins of the proximal phalanx.
- Both dorsal and palmar/plantar osteochondral fragments (POFs) have been recognized as incidental radiographic findings, not always associated with lameness (OCD, possibly separate centre of ossification)

Clinical signs

- Acute or subacute onset, moderate to severe lameness.
- Sometimes incidental finding on radiographs without lameness.
- Joint effusion, pain on forced flexion.
- Obvious improvement with rest, but recurrence with exercise.

Diagnosis

- Radiography is important for exact localization of the fragments (Figure 25.17). Oblique proximolateral-distomedial and proximomedial–distolateral views may be necessary to view palmar/plantar fragments and determine if they originate from the proximal phalanx or proximal sesamoid bones.
- Intra-articular anaesthesia confirms the clinical significance of the radiographic finding.

Treatment

Treatment entails arthroscopic fragment removal before OA develops.

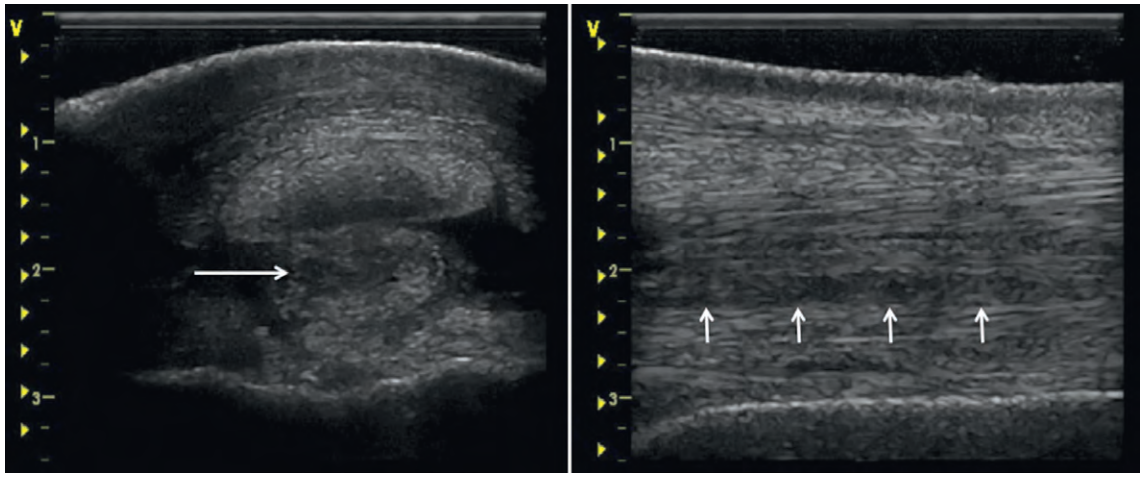


Figure 16.11 Transverse (left – lateral to the left)) and longitudinal (right – distal to the right) ultrasonographic images of desmitis of a straight distal sesamoidean ligament (arrows).

Prognosis

- The prognosis depends on the degree of damage to the articular surfaces of the fetlock joint and its supporting structures sustained during the traumatic accident that caused the chip fracture.
- Generally good for return to full exercise following arthroscopic removal, provided there is no radiographic evidence of OA.

Palmar/plantar process (eminence; wing) fractures

Location

- This type of fracture is located further abaxially along the palmar or plantar proximal articular margin of the proximal phalanx than chip fractures, and also tends to be larger. The lateral plantar tuberosity or eminence is more frequently involved than the medial.
- Fragmentation of the palmar or plantar eminence of the proximal phalanx may also be encountered as an incidental radiographic finding. This fracture-like appearance of the palmar or plantar eminence(s) may be related to an uncommon site of OCD, and there is generally no obvious association with lameness. This condition has been referred to as an ununited palmar or plantar eminence (UPE).
- Fractures may be articular or non-articular.
- Avulsion fractures involving the deep portion of the collateral ligament of the fetlock joint may appear similar to palmar process fractures. The joint should always be assessed for the presence of lateromedial instability that may lead to luxation.

Treatment

- Horses with non-articular fractures are managed with 3 months of box rest.
- Horses with articular fractures are candidates for fragment removal or lag-screw fixation of the fracture fragment. Fragment removal may require a small palmar arthrotomy as the articular component of the fracture may be too small for arthroscopic removal.

Desmitis of the distal sesamoidean ligaments

Aetiology

Overextension of the fetlock can result in damage to any component or combination of components of the suspensory apparatus. The distal sesamoidean ligaments extend distally from the distal border of the sesamoid bones and form the distal attachments of the suspensory apparatus.

Clinical signs

- In the acute stage of injury, oedema, heat, pain and swelling are present dorsal to the DDFT in the region of the pastern. Normally, the distal sesamoidean ligaments cannot be palpated deep to the DDFT, but, with injury, there may be palpable thickening of the lateral or medial oblique distal sesamoidean ligament.
- Lameness is usually abolished with an ASNB or anaesthesia of the digital flexor tendon sheath.
- Ultrasonography may show enlargement and/or hypoechogenicity of the oblique or distal sesamoidean ligament (Figure 16.11). When compared to MRI, ultrasonography has poor sensitivity and specificity and only predicts 20% of lesions accurately. However, the magic angle artefact can also result in false-positive diagnoses of oblique distal sesamoidean ligament desmitis when using standing MRI units.
- Entheseophytes may be present at the attachments of these ligaments to the proximal phalanx (Figure 25.5a) or sesamoid bones. Entheseophytes may sometimes be an incidental radiographic finding.
- Lesions can sometimes extend into the digital sheath (Figure 16.11)

Treatment

- Stall rest for 4–6 months and anti-inflammatory medication, followed by gradual return to exercise.
- Provide palmar/plantar foot support with egg-bar, or long, wide-webbed, seated-out shoes.
- Anti-inflammatory intrathecal medication of the digital flexor tendon sheath with a corticosteroid has been advocated although it may slow soft-tissue healing.
- Extracorporeal shock wave therapy and ligament splitting has been advocated for treatment of chronic cases.
  - Intralesional injection of core lesions with platelet-rich plasma or bone-marrow derived mesenchymal stem cells may be helpful.
  - Tenoscopic debridement of lesions extending into the digital sheath.

Prognosis

Approximately 50% of horses return to previous level of performance with 6 months of rest and intrathecal medication of the DFTS sheath.

Distal and proximal digital annular ligament desmitis

Aetiology

- The proximal digital annular ligament (PDAL) stabilizes the superficial (SDFT) and deep (DDFT) digital flexor tendons in the proximal aspect of the pastern. The ligament is intimately associated with the palmar/plantar aspect of the SDFT and digital flexor tendon sheath.
- The distal digital annular ligament (DDAL) stabilizes the DDFT at the level of the PIP joint.
- Injury of these ligaments is rare, and the clinical relevance is not well determined. DDAL desmitis may be seen concurrently with distal DDF tendonopathy.

Diagnosis

- Usually mild lameness.
- Lameness can be abolished with a palmar digital nerve block (DDAL) or an abaxial sesamoid nerve block (PDAL). Lameness may also be improved after intrathecal anaesthesia of the digital flexor tendon sheath.
- Ultrasonographic examination may reveal thickening (diffuse or focal) of the PDAL, but abnormalities of the DDAL can only be identified with MRI.

Treatment and prognosis

- Conservative treatment may consist of box rest, application of bar shoes and intrathecal medication of the DFTS.
- Surgical treatment is rarely needed and entails tenoscopic or open desmotomy of the PDAL or DDAL.
- Prognosis is usually good.

Villonodular synovitis

Definition

- Chronic, proliferative, hypertrophic synovitis and capsulitis in the proximal dorsal pouch of the fetlock joint. It is caused by trauma to a normal transverse fold of synovial tissue in this location of the joint.
- Chronic proliferative synovitis may also occur in the proximal aspect of the palmar/plantar pouch of the fetlock joint.

Pathogenesis

- Chronic, low-grade trauma caused by repeated overextension of the fetlock.
- Chronic synovial inflammation can result in synovial proliferation and hypertrophy. The resulting soft-tissue mass may cause bone remodelling due to sustained local pressure on adjacent cortical bone.

Clinical signs

- Mainly in racehorses.
- Obvious fetlock lameness with swelling and pain on flexion.

Radiography/ultrasound

- Presence of a depression in dorsodistal surface of the cortex of the cannon bone just proximal to the distal sagittal ridge.
- Chronic palmar synovitis may cause supracondylar lysis of the palmar metacarpal cortex proximal to the sagittal ridge.
- Positive-contrast arthrography (filling defect) or ultrasonography is often necessary to confirm the presence of a space-occupying soft-tissue mass in the proximal dorsal pouch of the joint. A villous fold of greater than 7 mm in thickness requires surgical removal.

Treatment

- Local infiltration of the mass with corticosteroids.
- Optimally, arthroscopic removal followed by 2–3 months of stall rest.

Prognosis

80% of horses that undergo arthroscopic removal have additional cartilage lesions. Approximately 70% of horses return to exercise regardless of additional abnormalities present.

Luxation of the fetlock joint

Aetiology and classification

Luxation is classified as (a) simple luxation, in which there is no tissue disruption other than the collateral ligament(s), or (b) compound luxation, in which the joint is open or the suspensory apparatus is disrupted.

Luxation of the fetlock is usually associated with an accident in which the digit is caught in a hole or cattle grid. It can also occur during fast turning with high rotational forces on the joint (Western riding, polo).

Clinical signs

- The lateral or medial collateral ligament is disrupted or avulsed, resulting in a valgus or varus deformity of the distal limb.
- Sometimes overriding of the distal end of the metacarpus/metatarsus and the proximal end of the proximal phalanx.
- If the luxation reduces spontaneously, minimal external signs are seen.
- Manual reluxation and reduction of the fetlock joint, causing relatively little pain, may be possible (unlike with a fracture). In one report joint laxity was only detected during palpation in approximately 50% of the cases.

Diagnosis

- Ultrasonographic examination shows disruption of the collateral ligament.
- Radiographic examination may show an avulsion fracture of the origin or insertion of the ligament. An avulsion fracture of the deep portion of the collateral ligament may resemble a fracture of the palmar/plantar eminence.
- To detect joint instability stressed radiographs of the fetlock joint should be obtained.

Treatment and prognosis

- Reduction and immobilization of the distal limb with a cast for 6 to 8 weeks allows most horses with simple luxation to regain soundness. Approximately 50% of horses with simple luxations are able to return to riding. Residual lameness may develop as a consequence of OA.
- Open luxations are treated in a similar manner following meticulous surgical debridement and irrigation. There is a considerable risk of secondary OA causing permanent lameness.
- Compound luxations accompanied by suspensory breakdown or complicated fracture may be salvaged with fetlock arthrodesis, with a fair prognosis for survival.

Collateral ligament desmitis of the metacarpo(tarso)phalangeal joint

Introduction

The collateral ligament of the fetlock joint consists of a superficial and a deep portion. The superficial part extends from the proximal extent of the epicondylar fossa of the distal metacarpus/metatarsus to the proximal aspect of the proximal phalanx. The deep portion courses in a slightly proximodorsal to distopalmar/plantar direction from the epicondylar fossa to the palmar/plantar eminence of the proximal phalanx and to a lesser extent also to the abaxial aspect of the sesamoid bone.

Aetiology

- Sprain injury to the fetlock joint. Hyperextension may result in excessive strain in the deep portion of the ligament.
- Front limbs are more commonly affected than hind limbs.

Diagnosis

- In the acute stage focal swelling and positive pain response to palpation over the affected ligament.
- Flexion test of the distal limb is usually markedly positive.
- Lameness may be improved by an abaxial sesamoid nerve block or by intra-articular anaesthesia of the fetlock joint. Lameness is usually only abolished by a low 4-point nerve block.
- Ultrasonographic examination should be used to determine if size difference exists between the affected collateral ligament and the same structure in the contralateral limb. Loss of echogenicity may also be present. Normal areas of hypoechogenicity occur in the collateral ligaments due the presence of off-incidence fibre bundles.
- Desmitis of the collateral ligament frequently represents part of combination-type injury with other abnormalities of the fetlock joint.
- MRI is most sensitive and helps identify other concurrent injuries.

Treatment

- Stall rest for a minimum of 8 weeks followed by 2 months of restricted exercise and small round pen turnout.
- Extracorporeal shock wave therapy.
- Anti-inflammatory intra-articular medication may be helpful.

Prognosis

Generally good for a return to soundness. Overall outcome depends on the presence of other abnormalities.

Desmitis of the intersesamoidean ligament

Aetiology

- Uncertain aetiology. Sepsis (haematogenous or iatrogenic) and trauma have been implicated.
- Hind limbs are more commonly affected.

Diagnosis

- Lameness is usually severe and of sudden onset.
- Distension of the fetlock joint or DFTS is possible.
- Radiographic evidence of irregularity of the axial margin of the sesamoid bones due to osteolysis or fragmentation.
- Ultrasonography may reveal echogenicity changes and/or irregularity to the axial margin of the sesamoid bones.
- Synoviocentesis of the fetlock joint and digital flexor tendon sheath should be performed to rule out sepsis of either synovial structure because the intersesamoidean ligament separates these two synovial cavities.

Treatment

- Conservative management with 6 months' stall rest and gradual rehabilitation can be successful in traumatic cases.
- Arthroscopic debridement of the axial margin of the sesamoid bones has been reported as the preferred treatment option.
- Aggressive antibiotic therapy if sepsis is diagnosed. Tissue samples should be taken during surgery for culture and sensitivity.

Prognosis

- Poor with conservative management.
- Guarded to poor with surgical treatment of septic lesions.
- Fair to good with surgical treatment if no infection is present.
- A postoperative increase in radiographic distance between the axial margins of the sesamoid bones (>6 mm) may indicate instability and abaxial displacement.

Subchondral bone injury in the fetlock region

Aetiology

- Acute post-traumatic subchondral bone damage following sprain injury.
- Stress-induced fatigue damage to bone occurs frequently in the immature equine athlete such as the Thoroughbred racehorse. The palmar/plantar metacarpal/metatarsal condyles are a predilection site, and bone injury has been referred to 'palmar osteochondrosis', 'hot condyles', 'palmar osteosclerosis and osteonecrosis complex' and 'palmar osteochondral disease'.
- The same process may occur in the mature sporthorse, but the location of bone injury of the fetlock is more varied and may include the proximal phalanx and the dorsal aspect of the metacarpal/metatarsal condyles.

Clinical signs

- Lameness varies from acute severe fetlock lameness following sprain injury to low-grade stiffness in both fetlocks or simply loss of performance in young Thoroughbreds.
- Usually no localizing signs present.

Diagnosis

Lameness is improved following intra-articular anaesthesia of the fetlock joint and abolished with a low 4- or 6-point nerve block. Pain associated with palmar osteochondral disease can be abolished by selective anaesthesia of the palmar metacarpal nerves only.

Radiographic abnormalities are minimal except in advanced cases with focal osteolysis in the palmar/plantar metacarpal/metatarsal condyle. This diagnosis can only be made on oblique dorsoproximal-palmarodistal oblique projections.

Nuclear scintigraphy shows increased radionuclide uptake at the site of osseous injury.

MRI is able to show localized bone oedema, sclerosis and focal osteonecrosis that can not be seen radiographically. It is further capable of providing more detailed information about the nature of bone injury than scintigraphy. Special fat suppressed image sequences (STIR) highlight the presence of fluid within bone (bright white signal) allowing the detection of bone oedema or bone bruising.

Treatment

- Acute or subacute bone bruises or bone oedema are best treated with stall rest and minimal in-hand walking for 2 to 3 months. Resolution of bone injury can be monitored scintigraphically.
- Administration of bisphosphonates like tiludronate may inhibit further bone resorption and reduce bone inflammation.
- Palmar or plantar osteochondral disease in young Thoroughbreds can also be managed conservatively but is often refractory to rest. Prevention is important by designing training programmes that include short periods/distances of high-speed work while mainly working at below maximum speed.
- In horses with irreversible changes such as cartilage erosions, bone cyst formation, subchondral bone lysis or periarticular osteophyte formation, palliative intra-articular medications may temporarily reduce lameness and slow progression to OA.
- Arthroscopic intervention may be useful in areas where bone injury is accessible to surgical debridement.

Prognosis

- Prognosis is favourable when bone injury is diagnosed early.
- Prognosis is unfavourable when secondary degenerative changes like OA and bone necrosis have become established.
- In one report of subchondral bone injury involving the fetlock joint of mature horses diagnosed with MRI, 75% returned to athletic soundness.

Osteochondritis dissecans (OCD) and osseous cyst-like lesions in the fetlock joint

Location

- Sites of occurrence for OCD lesions in the fetlock joint are:
  - the proximal end of the sagittal ridge of the metacarpus/metatarsus.
  - the distal end of the sagittal ridge of the metacarpus/metatarsus.
  - the proximal dorsal margin of the proximal phalanx (dorsal P1 fragments).
    These fragments may be differentiated from traumatic chips (see 'Chip fractures' in 'Fractures of the proximal phalanx', above) by their smooth, rounded appearance.
  - the proximal palmar/plantar margin of the proximal phalanx (POFs).
    Lesions involving the sagittal ridge have been classified as follows:
    Type 1: depression in the sagittal ridge; no fragment.
    Type 2: depression with corresponding osteochondral fragment.
    Type 3: loose osteochondral fragment.
    Palmar/plantar fragments of the proximal phalanx are classified as:
    Type 1: small articular margin fragments.
    Type 2: palmar process fragments (including ununited palmar/plantar eminence; discussed in 'Palmar/plantar process [eminence; wing] fractures' in 'Fractures of the proximal phalanx', above).
    Osseous cyst-like lesions in the fetlock joint are found at.
  - the proximal articular surface of the proximal phalanx.
  - the distal articular surface of the metacarpus/metatarsus.

Clinical signs

- Joint swelling, mild lameness.
- Radiography (Figure 25.10).
- Lameness is exacerbated by flexion and abolished by intra-articular anaesthesia.
- Seen in young horses, mostly at the time of first introduction to exercise.
- All four fetlock joints can be involved.
- Palmar/plantar Type 1 fragments may not cause lameness or only cause performance loss at high speed. These fragments are typically encountered in hind limbs on the medial aspect of the proximal phalanx.

Treatment and prognosis

- Arthroscopic fragment removal and lesion debridement into healthy subchondral bone. Prognosis is favourable unless additional cartilage damage is present.
- Osseous cyst-like lesions may respond favourably to 3 months' box rest. Administration of tiludronate has been advocated to prevent further enlargement of the cyst and suppress bone inflammation.
- Osseous cyst-like lesions that fail to respond to rest can be:
  - Injected with a corticosteroid using arthroscopic control.
  - Debrided with curettage under arthroscopic control if accessible. In 12 horses with osseous cyst like lesions 80% returned to exercise following arthroscopic debridement.
  - Debrided through an extra-articular transcortical drill tract with or without engraftment with cancellous bone or tricalcium phosphate.

Osteoarthritis of the fetlock joint

Introduction

OA of the fetlock joint is one of the 10 most common causes of lameness in all categories of sport horses surveyed. It is a more common cause of lameness in racehorses than in non-racing sport horses. The fetlock joint is the most frequently affected of all equine joints with degenerative and traumatic lesions.

Aetiology

1. Primary OA.
   - Probably due to chronic repetitive trauma in racing Thoroughbreds.
2. Secondary OA – the consequence of other joint diseases including:
   - Traumatic synovitis, capsulitis or sprain.
   - Articular fracture.
   - OCD.
   - Osseous cyst-like lesions.
   - Septic arthritis.

Clinical signs

- Mild to moderate, persistent lameness.
- Joint effusion.
- Positive distal flexion test.

Diagnosis

- Lameness abolished by intra-articular anaesthesia.
- Radiography (Figure 25.4):
  - Osteophytes at the dorsoproximal and palmaroproximal margins of the proximal phalanx, the apices and distal articular margins of the sesamoid bones, and the lateral and medial joint margins.
  - Joint space narrowing, often asymmetric.
  - Flattening of the palmar aspect of the metacarpal condyles.

- Supracondylar lysis proximal to the sagittal ridge of the metacarpus either at the palmar or dorsal aspect of the joint (Figure 25.49).

Treatment

- There is no effective treatment for OA.
- Lameness can be managed with palliative anti-inflammatory systemic medication (NSAIDs) or intra-articular therapies (hyaluronan, PSGAGs, IRAP, corticosteroids).
- Regular exercise may have a protective effect against the progression of OA.
- Surgical arthrodesis of the fetlock joint can be performed in horses with advanced crippling OA (see breakdown injury of the suspensory ligament).

Sesamoiditis

Definition

The term 'sesamoiditis' is used to describe proliferative or degenerative bony changes of the proximal sesamoid bones. Two forms of sesamoiditis have been described, a 'periostitis' form and an 'osteitis' form.

Aetiology and pathogenesis

Periostitis form. Acute or chronic injury to the attachments of the suspensory ligament branches, distal sesamoidean ligaments or palmar/plantar annular ligament of the fetlock, may result in damage to the bone–ligament interface with the sesamoid bones. Osseous metaplasia of scar tissue and periostitis may lead to the formation of entheseophytes and changes to the contour of the sesamoid bones. Periosteal sesamoiditis is therefore synonymous with entheseopathy of the soft-tissue structures that attach to the proximal sesamoid bones.

Osteitis form. Arteriosclerosis and thrombosis of the nutrient arteries, and local ischaemic necrosis have been implicated to explain the radiographic appearance of linear demineralizations along the vascular channels within the sesamoid bones, in analogy with the vascular theory of the navicular syndrome. This theory is no longer accepted. It is more likely that these linear radiolucencies either reflect a normal anatomical variation or are caused by entheseophyte formation between the nutrient foramina on the abaxial surface of the bone.

Clinical signs

- Acute injury to soft-tissue structures associated with the sesamoid bones causes moderate to severe lameness with swelling, heat and pain.
- The horse moves with a 'splinted' fetlock, avoiding dorsiflexion of the joint during weight-bearing.
- Chronic sesamoiditis produces non-specific distal limb lameness.
- Sometimes soft-tissue structures are palpably enlarged.
- Lameness is markedly exacerbated by distal limb flexion.

Diagnosis

- Low 4-point nerve block abolishes lameness while intra-articular anaesthesia does not.
- Radiographic signs (Figure 25.48):
  - Entheseophyte formation along the non-articular surface of the sesamoid bone results in shape and contour changes.
  - Calcification within the soft tissues of the suspensory apparatus.
  - When chronic lameness is present, changes may be accompanied by a degree of disuse osteoporosis of the proximal sesamoid bones.
  - Localized areas of decreased radiopacity along the vascular channels are of questionable significance. Vascular canals with parallel sides <2 mm in width are considered normal.

Treatment

- Horses with acute injury of the suspensory ligament are managed with prolonged rest and external support.
- Chronically affected horses can be managed with analgesic, anti-inflammatory drugs to control active periostitis or to allow the horse to exercise pain-free.
- Extracorporeal shock-wave therapy for chronic entheseopathy (insertional desmopathy) of the supensory ligament, palmar annular ligament or distal sesamoidean ligaments.
- Tiludronate to suppress ongoing osteolysis or bone inflammation.
- The use of the peripheral vasodilating drug, isoxuprine hydrochloride, has been advocated by some to treat horses with osteitis of the sesamoid bones.

Prognosis

Prognosis for athletic soundness is determined by the degree of damage to the suspensory ligament and distal sesamoidean ligaments.

Fracture of the proximal sesamoid bone

Classification and incidence

- Classification according to location as apical, midbody, basilar, abaxial, axial, sagittal or comminuted (Figure 16.12).
- In Thoroughbreds apical sesamoid bone fractures are more common in hind limbs and more frequently involve the medial sesamoid bone.
- Mid body fractures occur more frequently in the medial sesamoid bone of the fore limbs.
- Chip fractures of the axial margin of a sesamoid bone may be caused by avulsion or severe sprain of the intersesamoidean ligament. Axial fractures may also occur in conjunction with condylar fractures of the metacarpus in racing Thoroughbreds.
- Biaxial proximal sesamoid bone fractures represent the most common fatal distal limb fracture in all-weather flat racing in the UK.
- Sometimes occur bilaterally in young foals.

Aetiology

- Acute overextension of the suspensory apparatus. In racehorses, this may be associated with muscle fatigue at the end of a race.
- Foals tend to fatigue and fracture their proximal sesamoid bones while galloping alongside the mare in the field. Unilateral sesamoid bone fractures in foals that heal spontaneously result in megasesamoids (unusually large or misshapen sesamoids).
- Fracture of the proximal sesamoid bone should be differentiated from bipartite development (smaller proximal and larger distal separate centres of ossification).
- Degenerative changes (sesamoiditis) may be a consequence of chronic cyclic loading (bone fatigue) in young racehorses and predispose the sesamoid bones to fracture. Training may strengthen other components of the suspensory apparatus relatively more than the sesamoid bones, making the sesamoid bones more susceptible to failure.
- Direct impact trauma (e.g. brushing)
- Secondary to disuse osteopenia after removal of a distal limb cast.

Clinical signs

- Acute, moderate to severe lameness.
- Usually only one sesamoid affected. Fracture of both sesamoids results in breakdown of the suspensory apparatus.
- The horse walks with a 'splinted' fetlock, avoids dorsiflexion during weight-bearing.

Figure 16.12 Pre- and post-operative dorsopalmar radiographs of a chronic apicoabaxial proximal sesamoid bone fracture fixed with two 3.5-mm screws.

- Horses with small apical or basilar chip fractures may improve dramatically with rest, but lameness returns when exercise is resumed.
- Variable signs are swelling, effusion of the DFTS or fetlock joint, crepitus, pain on pressure over the sesamoid bones, enlargement of a suspensory branch.

Treatment and prognosis

- Conservative treatment of sesamoid bone fracture in mature horses is generally unsuccessful. As with fractures of the distal phalanx or navicular bone, these fractures tend to heal by fibrous union resulting in a permanent radiographic fracture gap.
- Arthroscopic fragment removal of apical fractures to allow return to athletic exercise and to avoid OA. Up to one third of the apical region of the sesamoid bone can be removed. The prognosis for return to soundness depends on the degree of damage to the suspensory ligament. Surgical outcome ranges from 64 to 88% for return to racing. This contrasts with 37% for horses that were treated conservatively. Medial sesamoid bone fractures of the forelimb carry a worse prognosis in Thoroughbred horses (approximately 45% return to racing).
- Basilar chip fracture fragments can be removed arthroscopically and carry a fair prognosis for return to performance (50–60% in racehorses). Larger basilar slab fractures that span the entire width of a sesamoid bone require lag-screw or cerclage wire fixation. Prognosis for return to athletic function is guarded because the insertion of the distal sesamoidean ligaments is lost.
- Midbody fractures are reconstructed with lag-screw fixation or cerclage-wiring. In a recent study, lag-screw fixation allowed 44% of the horses to race while none with wire fixation did.
- Abaxial surface fractures require surgical removal only if the fracture has an articular component. Prognosis for soundness has been reported to be approximately 70%.
- Chip fractures of the axial margin of a sesamoid bone involving the intersesamoidean ligament can be removed arthroscopically. Larger axial (sagittal) fractures associated with condylar fractures require lag-screw fixation, but horses with a condylar fracture complicated by an axial sagittal fracture of a proximal sesamoid bone have a very poor prognosis for return to soundness.

Breakdown of the suspensory apparatus

Aetiology

Acute overextension of the fetlock that disrupts any or all of the components of the suspensory apparatus:

- the suspensory ligament.
- the sesamoid bones.
- the distal sesamoidean ligaments.

These injuries usually occur in racehorses exercising at high speed. The use of toe grabs in racing shoes has been shown to increase the risk for suspensory apparatus failure significantly.

Clinical signs

The fetlock drops down to the ground during weight-bearing, and the pastern lies horizontal to the ground.

Diagnosis

- Radiography may be necessary to recognize concurrent bone damage (e.g. avulsion of a collateral ligament, fracture of a sesamoid bone, condylar fracture of the metacarpus/metatarsus, proximal phalanx fracture).
- Vascular supply of the distal limb must be assessed for permanent damage. Assessment is best performed after the horse has been treated for several days with a first-aid, distal limb cast, to give damaged arteries time to recover. Irreversible damage leads to ischaemic necrosis and sloughing of the limb distal to the fetlock.

Treatment and prognosis

- First aid consists of a distal limb cast or splint, applied with the distal limb vertically aligned. Alternatively a board splint can be used. After 5–7 days the cast is removed to examine the distal limb for ischaemic damage.
- If the vascular supply to the distal limb is not irreversibly damaged, the limb can be re-immobilized to allow formation of fibrous tissue of sufficient strength to support the fetlock, although this rarely results in pain-free weight-bearing.

- The treatment of choice to salvage valuable breeding animals is arthrodesis of the fetlock joint with a dorsal dynamic compression plate or locking plate. Two parasagittal lag screws or tension band wires are used to immobolize the proximal sesamoid bones.

Prognosis

Thirty-two of 54 horses treated with surgical arthrodesis survived and were eventually allowed unrestricted activity.

Annular ligament syndrome

Aetiology and classification

- Annular ligament syndrome refers to lameness caused by a restriction of the 'fetlock-canal' between the palmar/plantar annular ligament (PAL) of the fetlock and the intersesamoidean ligament.
- Indirect restriction occurs due to:
  - chronic primary tenosynovitis of the DFTS, chronic tenosynovitis due to tendinitis of the SDFT or DDFT; tendinitis can consist of a 'low bow' of the SDFT, tendinitis or vertical marginal tearing of the DDFT, or a tear of the manica flexoria.
- Direct restriction is caused by primary desmitis and thickening of the PAL, possibly as a consequence of hyperextension injury.
- Annular ligament syndrome is more commonly seen in ponies and cob- and draft-type horses and typically involves a hind limb.

Clinical signs

- Persistent lameness, even after long periods of rest.
- Swelling of the DFTS, proximal and distal to the borders of the annular ligament, gives a characteristically notched, hour-glass appearance to the palmar/plantar surface of the fetlock (Figure 16.13).
- Splinted fetlock gait: dorsiflexion of the fetlock is reduced during weight-bearing.
- Sometimes the horse is reluctant to place the heel of the foot fully to the ground.
- Lameness is exacerbated by a flexion test of the fetlock region.

Figure 16.13 The appearance of annular ligament syndrome. Note the 'notch' at the level of the plantar annular ligament (arrow). This notch is not pathognomonic for annular ligament syndrome as it occurs whenever there is digital sheath distension so an ultrasonographic examination is necessary to identify if the annular ligament is thickened. Thickening of the annular ligament may be due to primary desmitis or secondary to primary intra-thecal abnormalities.

Diagnosis

- Diagnostic anaesthesia:
  - Lameness is improved or abolished by intrasynovial anaesthesia of the DFTS.
  - Lameness is unaltered or improved by an ASNB and abolished following a low 4-point nerve block.
  - Sometimes a residual mechanical gait restriction persists in spite of diagnostic anaesthesia.
- Tenography following insufflation of air into the DFTS shows thickening of the PAL.
- Ultrasonography unequivocally establishes thickening of the PAL and/or the palmar/plantar soft tissues. A normal PAL is less than 2 mm thick, and the subcutaneous layer between the PAL and the palmar/plantar skin surface is <5 mm thick.

Treatment and prognosis

- Treatment consists of surgical decompression of the 'fetlock-canal' and the DFTS with desmotomy of the PAL if there is significant constriction. This is probably best assessed tenoscopically.
- Desmotomy of the PAL is best performed using a 'key-hole' approach under tenoscopic guidance to avoid complications with wound healing that may occur with an open surgical technique and which also allows assessment of the degree of constriction and the presence of primary disease. Alternatively a semi-open technique can be used to allow blind transection of the PAL with scissors through a small incision in the palmaroproximal pouch of the DFTS.
- The horse is rested in a stall, and its limb bandaged until sutures are removed at 2 weeks. Exercise is initiated at this time and gradually increased (2 weeks walking in hand, followed by 4 weeks lunging exercise) to avoid formation of restrictive adhesions within the DFTS.
- The overall prognosis for return to soundness following desmotomy is favourable to good (55–80%). However, outcome is significantly less favourable (22–42%), when additional tendon injury is present.
- According to one study, desmotomy under tenoscopic guidance results in less morbidity and higher return to previous exercise levels (78%) than open desmotomy (55%).

Tenosynovitis of the digital flexor tendon sheath and longitudinal tears of the superficial or deep digital flexor tendon

Aetiology and pathogenesis

- Longitudinal tears of the intrathecal part of the SDFT or DDFT result in protrusion of damaged collagen bundles from the tear into the synovial environment. Exposure of damaged collagen may cause chronic tenosynovitis with synovial distension.
- Longitudinal tears occur at the lateral or medial border of the intrathecal part of either flexor tendon. Lateral lesions are more common than medial ones. In addition tearing of the manica flexoria of the SDFT may also occur. Lesions of the DDFT are most common in the forelimbs while lesions of the manica flexoria are most common in the hind limbs.

Clinical signs

- Lameness may be subtle but is worse following exercise.
- Distension of the DFTS that waxes and wanes with exercise and rest.
- Positive lower limb flexion.
- Positive intrathecal anaesthesia (not always a complete response).

Diagnosis

- Ultrasonographic examination is often unable to visualize abnormalities of the margins of the intrathecal part of the flexor tendons. An irregular contour and/or the presence of echogenic material adjacent to the tendon may be the only subtle abnormal findings.
- Further diagnostic information may need to be obtained from MRI or tenoscopic examination (Figure 16.14).
- A failure to respond to intrathecal corticosteroid medication and rest suggests the presence of an underlying primary tendon abnormality in a chronically distended DFTS.

Treatment

- Tenoscopic debridement of the protruding collagenous tissue from vertical marginal tears in the SDFT or DDFT to remove the initiating cause of synovitis.
- Small tears of the manica flexoria are debrided. If the tear is so large that it causes instability of the manica flexoria, the manica should be completely removed.
- Intralesional or intrathecal administration of bone-marrow derived mesenchymal stem cells, may support connective tissue regeneration in the tear.
- Palmar/plantar annular ligament desmotomy is only indicated if the PAL is significantly enlarged and causing constriction.
- Slowly progressive rehabilitation of controlled walking exercise and stall rest over a period of 4 to 6 months.
- Intrathecal injection of hyaluronic acid during the rehabilitation period may reduce the incidence of restrictive adhesions.

Prognosis

- The prognosis is considered guarded to favourable following surgical debridement.
- Reportedly, 68% of horses were sound and 54% returned to preoperative levels of performance. Marginal tears of the DDFT, tears >3 cm and marked preoperative distension were associated with persistent lameness. Return to exercise required an average of 7 months' rehabilitation.

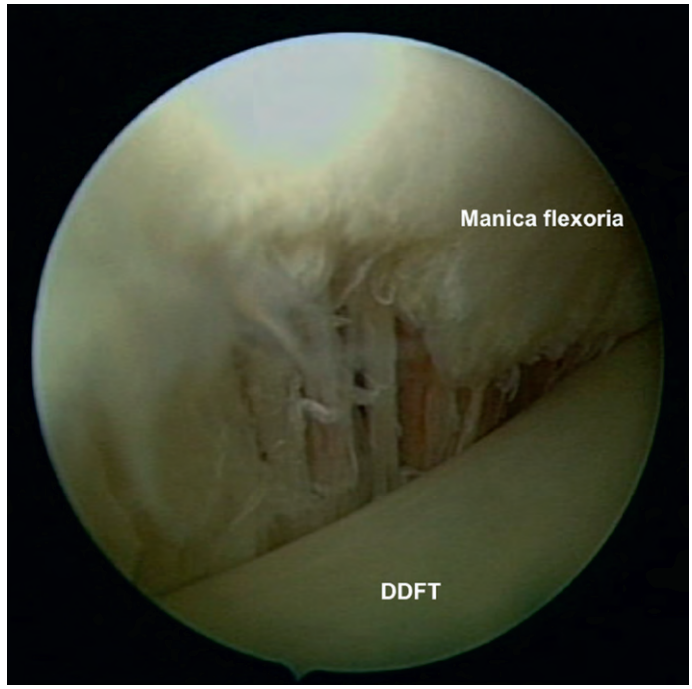


Figure 16.14 Tenoscopic view taken from between the manica flexoria and the dorsal surface of the deep digital flexor tendon in the proximal digital sheath, showing tearing of the attachment of the manica flexoria to the border of the superficial digital flexor tendon (not visible). Such cases are treated by tenoscopic removal of the entire manica flexoria with good post-operative outcome.

Septic tenosynovitis of the digital flexor tendon sheath (DFTS)

Aetiology

- Lacerations or puncture wounds of the DFTS. Small puncture wounds that readily seal may initially not be recognized.
- Iatrogenic following therapeutic injection.
- Occasionally septicaemia or bacteraemia may result in infection of the DFTS.

Clinical signs

- Severe distension of the DFTS, sometimes difficult to localize due to generalized cellulitis with painful swelling of the distal limb.
- Swelling, pain and lameness may not develop for some time after the initial injury if there is adequate drainage of fluid from the DFTS.
- Severe, non-weight-bearing lameness. Adequate drainage may result in less lameness than typically associated with septic tenosynovitis.
- Small lacerations may result in extensive tendon damage within the DFTS due to movement of the horse at the time of injury.

Diagnosis

- Synovial fluid analysis to support a diagnosis of septic tenosynovitis.
- Remote injection of sterile balanced electrolyte solution into the DFTS may confirm communication with a penetrating wound.
- Radiography may reveal osteolytic changes in the sesamoid bones.

- Ultrasonography to evaluate the integrity of the flexor tendons within the DFTS or the presence of foreign bodies. Tendon injury may have resulted from the original penetrating injury. Sepsis may also cause secondary 'melting tendinitis' of the flexor tendons in an infected DFTS.

Treatment

- Immediate primary closure of an open DFTS shortly after injury is usually contra-indicated. This may trap bacteria in the DFTS and promote the development of septic tenosynovitis. The wound should be thoroughly cleaned and is best maintained under a sterile dressing until further surgical treatment can be performed under general anaesthesia.
- Treatment of septic tenosynovitis must consist of emergency aggressive lavage and debridement of the DFTS under general anaesthesia, in conjunction with intrasynovial or regional and systemic intravenous antimicrobial therapy. Primary apposition of transected tendons may be attempted at the same time.
  - Recurrence of synovial infection following tenoscopic lavage and antimicrobial therapy is best managed by open drainage through incisional wounds. The limb should be kept under a sterile bandage, and the open incisions aseptically prepared on a daily basis.
  - Tendon lacerations must be immoblized using half-limb casts for 8 to 12 weeks. Regional antimicrobial therapy can be established in spite of cast immobilization with indwelling continuous antibiotic delivery devices or regional intravenous perfusion.
- Desmotomy of the PAL of the fetlock may cause significant pain relief in horses with chronic septic tenosynovitis of the DFTS.

Prognosis

- Complications of septic tenosynovitis are unresolved infection, adhesion formation, osteomyelitis of the sesamoid bones, septic 'melting' tendinitis and tendon rupture.
- Prognosis for survival and return to soundness is good if treatment of penetrating wounds is initiated before septic tenosynovitis develops. If there is partial or complete transection of flexor tendon(s) and/or infection has become established, prognosis is guarded. Horses treated within 36 hours of injury reportedly have a much better chance for survival than horses treated outside that time window (76% versus 33%).

Flexural deformity of the fetlock joint

Aetiology and pathogenesis

- Flexural deformity of the fetlock is caused by relative shortening of the musculotendinous units of the SDF, the DDF, the suspensory ligament or any combination of these structures.
- Flexural deformities may be congenital or acquired (see 'Traumatic and degenerative arthritis of the DIP joint' above).
- Congenital rupture of the common digital extensor tendon may result in flexural deformity.
- Acquired flexural deformities of the fetlock develop typically in yearlings and may be a manifestation of developmental orthopaedic disease (DOD).
- The combination of longitudinal growth of the distal radial physis, the position of the superior check ligament and loss of elasticity in the suspensory ligament, may be responsible for flexural deformities in yearlings (cf. DIP joint in foals).
- Flexural deformity can develop in older horses due to chronic superficial digital flexor tendinitis (Figure 16.15)

Figure 16.15 Flexural deformity of the metacarpophalangeal joint associated with chronic superficial digital flexor tendinopathy. This is a rare complication of chronic tendon scarring and is a difficult condition to treat.

Clinical signs

- Flexural deformity of the fetlock is characterized by a straight fetlock angle with a variable amount of dorsal knuckling of the joint.
- Palpation of the limb is required to assess which tendon or ligament is predominantly responsible.
- Two levels of severity:
  - Stage I, in which the fetlock is dorsiflexed during weight-bearing, with only occasional knuckling.
  - Stage II, in which the fetlock is permanently knuckled forward and no dorsiflexion occurs.

Treatment

- Congenitally affected foals may correct spontaneously, or they may require the application of bandages and splints and intravenous administration of oxytetracycline to induce musculotendinous relaxation.
- Limbs with flexural deformity associated with rupture of the common digital extensor tendon are best bandaged and supported with a PVC splint applied to the palmar aspect of the fetlock.

- Mildly to moderately affected horses (stage I) can often be corrected with dietary restrictions, controlled exercise, physiotherapy in combination with analgesic drugs, and application of a bandage with a splint applied to the palmar aspect of the fetlock. Therapeutic shoes should provide a toe extension and raised heels.
- Horses with stage I deformity unresponsive to conservative management can be treated surgically with inferior or superior check ligament desmotomy, depending on which musculotendinous unit is primarily involved.
- Horses with stage II deformity should be treated surgically. Both inferior and superior check ligament desmotomy followed by palmar application of splints may be successful, but some horses may require tenotomy of the SDFT or desmotomy of the suspensory ligament to achieve normal posture.

Prognosis

This deformity is usually more difficult to correct than flexural deformity of the DIP joint. The prognosis remains favourable as long as the deformity can be corrrected with desmotomy of one or both check ligaments, and if tenotomy or suspensory ligament desmotomy are not required.

Angular deformity of the fetlock

Aetiology and pathogenesis

- Angular deformity of the fetlock joint may be congenital or acquired.
- Congenital angular deformity is due to joint laxity.
- Acquired angular deformity is caused by asynchronous longitudinal growth at the distal metacarpal/metatarsal physis and is a manifestation of DOD (see 'Angular deformity of the carpus').
- Because most of the growth of the distal metacarpal/metatarsal physis occurs prior to 3 months of age, correction of angular deformity of the fetlock is urgent.

Clinical signs

- Fetlock deformity is mainly of the varus type and occurs predominantly in the hind limbs.
- Palpation to establish the presence of joint laxity.
- Radiography to assess involvement of the proximal physis of the proximal phalanx, the distal physis of the metacarpus/metatarsus or the presence of epiphyseal wedging.

Treatment

- Congenital form usually improves during first week of life. If joint laxity persists, the use of sleeve casts or braces may be appropriate.
- Conservative management consists of dietary restriction, stall rest and corrective trimming. If the angulation is <5° the prognosis is usually excellent.
- Foals unresponsive to conservative management or have >5° angulation are treated surgically by hemicircumferential periosteal transection or epiphyseal bridging with screws and wire, a transphyseal screw or a metal staple.
- Foals with permanent angular deformities need a corrective osteotomy. If left untreated lameness develops due to OA.

Prognosis

Favourable with early recognition and management.

16.3 DISEASES OF THE METACARPUS AND METATARSUS

Condylar, metaphyseal and diaphyseal fractures of the third metacarpal/metatarsal bone

Aetiology and classification

Condylar fractures

- Represent the most common cause of equine fatalities in Thoroughbreds on UK racecourses.
- These fractures occur more commonly in horses in their first year of racing and horses that started racing as 3–4 year olds.
- Other predisposing factors are firm or hard ground, absence of gallop training, long race distances, a large number of runners, shoes with toe grabs, and pre-existing suspensory ligament desmitis.
- Forelimbs are more frequently involved than hind limbs, and in the UK the right forelimb is affected twice as often as the left forelimb.
- Lateral condylar fractures are more common than medial condylar fractures.
- Displaced lateral condylar fractures may be associated with axial sesamoid bone fractures and palmar articular comminution.
- Medial condylar fractures are more common in the hind limbs and spiral further proximally into the metacarpus/metatarsus than lateral condylar fractures.
- Condylar fractures are stress fractures preceded by cyclical fatigue damage.

Metaphyseal fractures

- Salter-Harris types I and II are most common.
- Metaphyseal fractures are monotonic (traumatic) fractures.

Diaphyseal fractures

- Diaphyseal fractures are usually caused by high-energy impact trauma (monotonic).

Clinical signs

- From mild to non-weight-bearing lameness with obvious instability.
- Avoid nerve blocks if fracture is suspected.
- Heat, pain, and swelling depend on the type and age of the fracture.
- Metaphyseal and diaphyseal fractures are at high risk of becoming compound and in urgent need of adequate splinting.

Diagnosis

- Radiography (Figures 25.15 and 25.19)
  - Take multiple views to identify the fracture plane accurately.
  - Take flexed dorsopalmar, or dorsodistal-125° palmaroproximal oblique views of the fetlock to identify palmar cortical comminution in condylar fractures.
  - Look for overlapping vertical axial fracture lines in the corresponding sesamoid bone in horses with condylar fractures.
- Scintigraphy to identify stress fractures (Figure 25.18).

Treatment and prognosis

1. Condylar fractures:
   - Internal fixation with lag screws is the treatment of choice for incomplete, complete non-displaced and complete displaced lateral condylar fractures.

- Complete fractures may benefit from arthroscopic evaluation of reduction and removal of loose fragments. Palmar comminution can only rarely be debrided under arthroscopic visualization.
- The distal limb should be stabilized in a cast for recovery.
- Short incomplete lateral condylar fractures may be treated conservatively with casts only.
- Medial condylar fractures of the metatarsus must be carefully evaluated for proximal propagation. Incomplete fractures with proximal propagation are best treated using methods of internal fixation that incorporate the entire metatarsus in the repair.
- The prognosis for return to exercise for horses with incomplete condylar fractures is generally good (74–87% success). Prognosis is less favourable for complete condylar fractures but still fair. Horses with chronic or displaced fracture, concurrent palmar comminution or an axial proximal sesamoid bone fracture have a poor prognosis for return to their previous level of performance.

2. Diaphyseal fracture:
   - Reconstruction with one or typically two dynamic compression or locking plates.
   - Implants are removed in staggered fashion to increase the mechanical load on the healed metacarpus stepwise.
   - Bone healing requires minimally 3 to 4 months of immobilization/fixation. Gradual return to exercise can usually be considered from 6 months onwards.
   - Prognosis for survival is guarded. Prognosis is poor especially if the fracture is open which occurs frequently due to the poor local soft tissue cover. The prognosis for survival becomes hopeless if the nutrient artery is involved in the fracture.
   - In foals the prognosis is better due to less implant failure than in adult horses. One study reports a favourable outcome (86%) in spite of a high incidence of open fractures.
3. Metaphyseal fracture:
   - Can be managed with cast immobilization, with or without additional internal fixation.
   - Any implants used to treat the fracture should not engage or cross the distal metacarpal/metatarsal physis.
   - The prognosis for soundness is favourable, unless growth disturbances occur due to premature physeal closure. Complications of cast immobilization are also common in foals.

'Bucked shins'/'sore shins'/dorsal metacarpal disease

Definition

These terms imply a painful condition of the dorsal aspect of the third metacarpal bone of young racehorses, caused by stress or fatigue damage.

Aetiology and pathogenesis

- During fast-gaited exercise the dorsal cortex of the third metacarpal bone suffers greater damage from compression than the rest of the cortex. If damage continues to occur regularly, the osteoclastic phase of the remodelling response exceeds the repair phase, and the bone weakens until it fails. This may eventually result in microfracture in the dorsal cortex, subperiosteal haemorrhage and callus formation.
  - Although the majority of Thoroughbred horses have been reported to develop some degree of bucked shins, only about 10% develop a stress fracture.
- Two syndromes are recognized:
  - Type I syndrome affects the dorsomedial cortex in 2-year-old racehorses.
  - Type II syndrome affects the dorsolateral cortex in 3- to 5-year-old racehorses.
- Both syndromes are associated by fatigue damage to bone or stress fractures.
  - Stress fractures are located in the middle third of the dorsal cortex of the metacarpus and travel in a dorsodistal to palmaroproximal direction.

Clinical signs

- The acute form of Type I dorsometacarpal disease (DMD) causes a sudden onset of painful 'shins', but the horse does not show obvious lameness or radiographic changes.
- The subacute form of Type I is accompanied by mild lameness, variable pain on palpation, obvious dorsal swelling and radiographic evidence of subperiosteal callus formation mostly on the dorsomedial aspect of the metacarpal cortex.
- Horses with Type II DMD have an incomplete, oblique fracture line in the dorsal or dorsolateral aspect of the metacarpal cortex. Lameness is most obvious after strenuous exercise.

Treatment

- Prevention is by gradually increasing the work-load of immature racehorses prior to initiating fast exercise. Initiation of regular short-distance breezing together with a reduction in long-distance galloping exercise.
- Treatment of acute form Type I DMD consists of 3 months of stall rest followed by 3 months of controlled exercise. Anti-inflammatory drugs and physical therapy are used to reduce pain and swelling.
- Treatment of horses with the subacute form Type I and Type II DMD with rest and controlled exercise is often unsuccessful.
- Surgical alternatives in unresponsive horses are forage of the affected area of the cortex (osteostixis, nine 2-mm drill holes each approx 1 cm apart), and/or unicortical positional screw fixation with a 3.5-mm cortical screw.
- Current non-surgical alternatives include application of extracorporeal shock waves to the painful dorsal cortical region and freeze firing.

Prognosis

- Good to excellent.
- At the time of screw removal 2 months after surgery, 98% of horses with a single stress fracture show radiographic healing, and 85–95% of horses return to racing with no change in earnings or performance.
- Median time to return to training was 2.8 and 7.6 months for racing.

Proximal palmar/plantar metacarpal/metatarsal pain

Definition and classification

Proximal metacarpal/metatarsal pain results in lameness referable to the proximal, palmar/plantar aspect of the metacarpus/metatarsus. Lameness in this area can be due to:

1. Avulsion fracture of the proximal palmar/plantar cortex.
2. Stress fracture of the proximal palmar cortex.
3. Entheseopathy of the origin of the suspensory ligament.
4. Desmitis of the proximal part of the suspensory ligament.

Clinical signs

- Avulsion fracture of the proximal palmar cortex causes acute, severe lameness. Stress fracture of the proximal palmar cortex, entheseopathy and desmitis of the proximal part of the suspensory ligament cause chronic, intermittent lameness.
- Signs of focal pain can sometimes be elicited with pressure over the proximal palmar metacarpus/metatarsus.
- Lameness can be exacerbated by localized sustained pressure.
- Sometimes there is indistinct thickening of the soft tissues in region of the proximal plantar metatarsal region.
- Lameness usually improves with rest but returns with exercise.

Diagnosis

- Radiography.
  - changes to trabecular pattern of the proximal palmar cortex.
  - faint vertical or circular radiolucent line(s).
- Diagnostic anaesthesia of the proximal palmar/plantar metacarpal/metatarsal region is best performed with a nerve block of the deep branch of the lateral palmar/plantar nerve.
- Scintigraphy to identify stress fracture or entheseopathy.
- Ultrasonography or MRI to identify structural changes in the proximal part of the suspensory ligament. It was recently shown that the sensitivity and specificity of ultrasonography for the diagnosis of proximal suspensory desmitis are poor in comparison with MRI.

Treatment and prognosis

- Prolonged box rest (2 to 6 months) followed by graduated return to exercise; monitor healing with ultrasonography or scintigraphy.
- Prognosis for return to exercise is favourable for stress fractures, avulsion fractures and proximal suspensory desmitis in forelimbs.
- Prognosis is poor for proximal suspensory desmitis in hind limbs that is treated with rest alone.
  - Extracorporeal shock wave therapy to treat entheseopathy and proximal suspensory desmitis. Approximately 50% of horses make a full recovery.
  - Osteostixis of the proximoplantar aspect of the metacarpus/metatarsus has been described from both a dorsal and a palmar approach.
- Neurectomy of the deep branch of the lateral palmar or plantar nerve to remove sensation from the proximal part of the suspensory ligament. Approximately 75% of horses with proximal suspensory desmitis of the hind limb(s) make a complete recovery.
- Horses with osseous abnormalities (entheseopathy) do not appear to respond as well to this procedure as horses with proximal suspensory desmitis alone. Osteostixis can be combined with neurectomy.

'Splints'

Definition and classification

A splint is a bony enlargement involving the second or fourth metacarpal or metatarsal bones. Different types of splint are:

- An intermetacarpal/intermetatarsal splint is an exostosis between the metacarpus/metatarsus and a splint bone.
- A postmetacarpal/metatarsal splint is an exostosis on the palmar/plantar or axial surface of a splint bone.
- A 'blind splint' is an exostosis on the axial surface of a splint bone that is relatively undetectable to inspection or palpation.

Aetiology and pathogenesis

- The medial splint bone is fully loaded by the second carpal bone, but the lateral splint bone supports only half the axial load from the fourth carpal bone. Chronic tearing and inflammation of the medial interosseus ligament may occur when a horse is first introduced to exercise. Periostitis and osseous metaplasia of scar tissue may cause bony enlargement of the proximal third of the medial splint bone of young horses. As the horse ages, the medial splint may fuse to the metacarpus/metatarsus, eliminating further movement and irritation to the interosseus ligament.
- Conformational factors may exacerbate strain to the medial interosseus ligament (i.e. bench knees, carpal varus deformities), or lead to direct trauma by brushing (i.e. toed-out, base-narrow conformation).

Clinical signs

- Firm swelling along the splint bone. Heat and pain in acute phase, quiescent in the chronic phase.
- Lameness only in acute phase, usually mild, worse on hard ground and when the horse is exercised in a circle.
- Sometimes chronic lameness, due to encroachment of bony enlargement of the axial surface of a splint bone on the suspensory ligament although this scenario is extremely rare (Figure 16.16).

Treatment and prognosis

- Horses with acute periostitis respond favourably to rest and anti-inflammatory medication. Although an obvious firm swelling may remain, persistent lameness is not expected.
- Blemishes in show horses caused by chronic exostosis may need to be surgically removed. Recurrence of the exostosis can be expected in up to 50% of the operated horses!

Fractures of the splint bones

Aetiology and classification

- May be classified as proximal, mid or distal splint bone fractures.
- Fractures of the distal third of the splint bone may occur spontaneously or may be caused by external trauma, such as a kick to the lateral splint bone or interference trauma to the medial splint bone.
- Spontaneous distal fractures are avulsion fractures caused by the pull of the distal ligament of the splint bone during extreme dorsiflexion of the fetlock. Increased dorsiflexion of the fetlock during weight-bearing is associated with weakening of the suspensory ligament due to desmitis, and so these two injuries are frequently concurrent. These fractures occur commonly in the medial splint bone of the forelimbs of Thoroughbreds and the lateral splint bone of the hind limbs of Standardbreds.
- Proximal fractures are generally caused by kicks to the lateral aspect of a hind limb. Concurrent incomplete fractures of the third metacarpal/metatarsal bone are possible. This implies a significant risk for recovery from general anaesthesia when treatment options are considered for the fractured splint bone.

Clinical signs

- Acute fractures cause swelling, heat, pain and lameness.
- Often occurs concurrently with suspensory ligament desmitis.
- Lameness varies, but is generally due to suspensory ligament desmitis.
- Sometimes accompanied by generalized oedema of the distal limb.
- Traumatic fractures are often open.
- Bone sequestration is a common complication of open splint bone fractures, resulting in a chronic, discharging sinus.

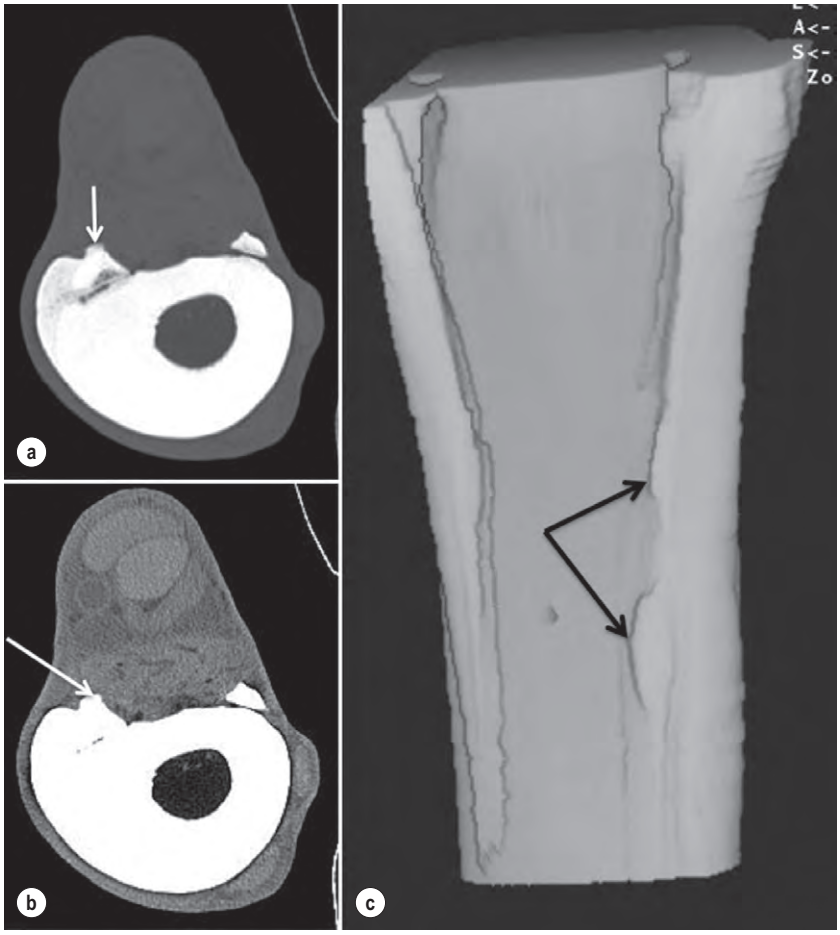


Figure 16.16 (a) CT (bone window) showing an axial exostosis of the second metacarpal bone (arrow). (b) CT (soft-tissue window) showing the capability of CT to demonstrate the relationship between bone and soft tissue, with the axial exostosis impinging onto the suspensory ligament (arrow). (c) 3D reconstruction of the same case showing the axial exostoses (black arrows).

Radiography

- Callus formation is commonly seen.
- Hypertrophic callus formation is associated with instability, excessive exercise or infection.
- Distal displacement of the distal fragment may lead to non-union or mal-union and can impinge on the suspensory ligament.
- Re-fracture through previous callus indicates persistent concurrent suspensory ligament desmitis and premature return to exercise.
- Radiographic complications like non-union, mal-union and hypertrophic callus are not usually associated with lameness.

Treatment and prognosis

- Distal fractures usually heal satisfactorily with conservative management. Return to the previous level of athletic activity depends frequently on healing of concurrent suspensory desmitis.
- Simple fractures of the middle or proximal third of a splint bone are usually best treated conservatively with rest.
- Comminuted and open fractures of the middle or proximal third of a splint bone:
  1. Should be treated initially as an open wound using wound debridement and lavage, systemically and regionally administered intravenous antibiotics, establishment of proper drainage and application of an absorbing, bulky dressing. This approach results in satisfactory healing in most horses.
  2. They can also be surgically removed by segmental ostectomy, leaving the unaffected proximal and distal portions of the splint bone *in situ*, or by subtotal ostectomy, leaving only the proximal portion of the splint bone behind. As much as the distal 2/3 to 3/4 of a splint bone can be safely removed, without causing instability of the remaining proximal fragment and the adjacent carpometacarpal or tarsometatarsal joint. If less than the proximal 1/3 to 1/4 of the splint bone remains, it should be stabilized with cortical screws or with a contoured small fragment plate. In hind limbs, the entire lateral splint bone can be safely removed in most horses, although in some horses this leads to persistent lameness, possibly due to instability of the fourth tarsal bone.
  3. Sequestration with osteomyelitis of a fractured splint bone can also be treated successfully with segmental or subtotal ostectomy. Occasionally sequestrum removal performed on the standing, sedated horse may lead to resolution of infection and satisfactory progression of healing.
  4. The choice between conservative and surgical management is often based on projected time to resolution, the cost of treatment or the presence of draining tracts. While surgical treatment may be more expensive, it can allow horses to resume normal activity more quickly, although the surgical treatment of splint fractures should be avoided initially because of possible concurrent incomplete fractures in the third metacarpal/metatarsal bone, which can become complete during recovery from general anaesthesia.

Proximal fractures, especially those with an intra-articular component and instability of the carpometacarpal or tarsometatarsal joint, require plate fixation with a small fragment plate.

Superficial digital flexor tendinitis

Definition and epidemiology

- Tendinitis refers to strain injury of a tendon. Strain is defined as damage to a tendon caused by overuse or overstress. Tendinitis can range from minor inflammation to disruption of the tendon.
- Tendinitis of the SDFT is one of the most frequent injuries affecting racing horses, especially those that compete over jumps.

Aetiology

- Tendinitis arises due to extrinsic (direct external trauma) or intrinsic factors (strain).
- Overstrain injuries can occur from sudden overloading of the SDFT or from progressive weakening of the tendon due to molecular degeneration which induces neither clinically evident inflammatory reaction nor any effective reparative response. This cumulative microdamage due to repeated mild overstrain injury predisposes to clinical injury during high-speed exercise.
- The incidence of tendinitis rises with an increase in age due to an ageing effect of the SDFT. Once skeletal maturity is reached, increasing age and exercise induce tendon matrix degeneration rather than adaptation in the horse.
- Since the SDFT operates close to its maximum strain capacity at high-speed exercise, any change in the tendon's elasticity and increase in strain may lead to injury. Above physiological strain levels are typically encountered at the end of a race, when muscle fatigue causes hyperextension of high motion joints and greater tendon deformation.

Pathophysiology

- The degree of damage varies from mild inflammation without fibrillar damage, to fibrillar stretching and slippage, to actual fibrillar rupture and, ultimately, to complete tendon rupture.
- Severe acute strain injuries typically are characterized by intra- and interfascicular haemorrhage (which separates and weakens remaining fibrils), and subsequent inflammation with oedema, hyperaemia, mononuclear infiltration, fibrin, local swelling, transudation and release of proteolytic enzymes.
- Healing typically represents the different stages of wound healing. The reparative phase starts within a few days, overlaps the inflammatory phase and lasts for several months.
- Following the initial stages of inflammation, healing is based on angiogenesis and fibroblastic infiltration. The reparative phase consists of the formation of granulation tissue and its organization into fibrous tissue. As fibroplasia progresses, ultrasonography reveals progressive increase in the tendon's echogenicity.
- During the remodelling phase, fibrous tissue matures, and there is a gradual transition from type III to type I collagen; collagen fibrils become thicker and cross-linked. As fibrous tissue matures and longitudinal collagen fibre organization improves, ultrasonographic evidence of a fibre pattern of long, parallel linear echoes returns.
- The healed, scarred tendon is stronger and stiffer but functionally inferior than the original tendon. It is therefore predisposed to re-injury, often at sites adjacent to the original injury.

Figure 16.17 Clinical appearance of superficial digital flexor tendinitis – note the palmar 'bowing' (arrows) in the metacarpal region.

- Healing occurs by intrinsic and extrinsic mechanisms. Severe injuries rely predominantly on extrinsic repair from the peritendinous vasculature and connective tissues. Extrinsic healing is very limited for injured tendons that lie within synovial sheaths, due to the absence of a paratenon and the presence of synovial fluid.

Clinical signs

- Minimal to marked increase in skin temperature over the palmar aspect of the limb.
- Immediate moderate to severe lameness.
- Bow-like palmar swelling develops immediately proximal to the level of the DFTS (Figure 16.17).
- Some injuries are not clinically apparent for 24–48 hours.
- Some injuries cause swelling but no lameness.
- Lameness usually improves quickly with box rest.
- Sinking of the MCP joint may occur with severe injury to the SDFT.
- Injury to the distal branches of the SDFT may occur at the level of the pastern. Swelling of the palmaroproximal aspect of the phalanges results and may be difficult to distinguish from desmitis of the distal sesamoidean ligaments.

Diagnosis

1. History and clinical signs as described above.
2. Careful palpation reveals warmth, thickening of the SDFT and pain on digital pressure.
3. Ultrasonography is best performed 7 days after the original injury, since the lesion may expand during the initial few days. Ultrasonographic findings include:
   - localized central or generalized diffuse hypoechogenicity.
   - enlargement of the tendon.
   - peritendinous oedema.
   - changes in shape, margin or position.

- A central core lesion is the most common finding (Figure 25.64).
- As the injury heals, the borders of the lesion become indistinct, and the lesion becomes more echogenic. Peritendinous oedema and inflammation disappear early in convalescence.
- Ultrasonographic findings allow lesion grading as mild, moderate, or severe depending on lesion size at the maximum injury zone (A) and the % of total tendon volume damaged (B). Several different grading systems have been used:
  - Mild tendinitis:
    A = <50% of cross-sectional area (CSA) and/or <100 mm in length
    B = 0–15%
  - Moderate tendinitis:
    A = 50–75% of CSA and/or 100–160 mm in length
    B = 16–25%
  - Severe tendinitis:
    A = >75% of CSA and/or >160 mm in length
    B = >25%

To determine when the horse is able to return to full exercise, the following criteria should be satisfied during sequential follow-up ultrasonographic examinations. Typically step-wise increase in exercise is documented ultrasonographically every 2 to 3 months.

- Good longitudinal fibre pattern and transverse echogenicity.
- Homogeneous appearance.
- No adhesions.
- Normal tendon size.

Prognosis

Prognosis for return to work depends on the location of the injury (within or without a tendon sheath), the severity of the injury and the level of exercise expected (racing vs. general riding). Recurrence is common.

Treatment

1. Acute (inflammatory) tendinitis.
   - Ice-packs, cold hosing.
   - Apply pressure bandage using several layers of padding.
   - Stall rest to limit further damage.
   - Intralesional PSGAG may help suppress acute inflammation.
   - Tendon splitting to decompress large-core lesions and promote extrinsic repair is optional.
   - Desmotomy of the accessory ligament of the superficial digital flexor tendon to lengthen the musculotendinous unit and prevent excessive strains in the repair tissue is optional. This may increase the risk for suspensory desmitis in the future.
   - Do not use heel elevation.
2. Subacute (fibroplastic) stage.
   - Stall rest with increasing periods of controlled hand-walking.
   - Intralesional injections: hyaluronic acid, polysulphated glycosaminoglycans.
   - Regenerative intralesional therapies: bone-marrow derived mesenchymal stem cells, platelet-rich plasma, bone-marrow concentrate, IGF-I, TGF-beta.
   - Extracorporeal shock wave therapy reportedly promotes growth factor release in healing tendons.
3. Chronic (remodelling) stage.
   - The aim of a controlled exercise regimen is to optimize scar tissue function without causing further injury. Exercise can be gradually increased to include periods of trotting from 3–4 months, cantering from 9 months and galloping exercise from 12 months. Typically step-wise increase in exercise is monitored ultrasonographically before and after every increase (or every 2 to 3 months). An increase in CSA greater than 10% between examinations suggests a degree of re-injury and consequently, that the exercise level should be reduced.
   - The horse should not be turned out to pasture until healing is complete.
   - Desmotomy of the accessory ligament of the superficial digital flexor tendon to lengthen the musculotendinous unit and prevent excessive strains in the repair tissue.

Spontaneous rupture of the superficial digital flexor tendon

Aetiology

- Affects forelimbs of aged horses (18–22 years).
- Rupture not associated with external trauma or previous injury.

Diagnosis

- Marked local swelling and hyperextension of the fetlock.
- Ultrasonography shows near complete anechogenicity at the level of the rupture in the SDFT.
- Lesions are seen in the metacarpal region.

Treatment

- Complete stall rest for 4 weeks, followed by stall rest and some in hand grazing for an additional 8 weeks. Gradual increase in walking exercise thereafter until 6 months post injury.
- Support bandages for at least 6 weeks.

Progression and prognosis

- 3 months post injury horses are typically sound at the walk and trot, and hyperextension of the fetlock is improved.
- Development of a mild carpal flexural deformity is not uncommon.
- Horses can return to previous exercise (pleasure riding), but re-injury, and involvement of the contra-lateral SDFT is possible.

Deep digital flexor tendinitis

Aetiopathogenesis

- The DDFT is most frequently injured in the digital flexor tendon sheath.
- The mechanism of injury is unknown, but a single excessive loading cycle may cause high pressure increase in the compressed portion over the palmar aspect of the fetlock resulting in bursting of the lateral (or medial) margin of the tendon.
- Small core lesions within the substance of the DDFT may be seen in the region of the pastern.

Clinical signs

- Effusion of the DFTS.
- Mild to severe lameness, exacerbated by distal limb flexion.
- Lameness improved or abolished by intrathecal anaesthesia of the DFTS or low 4-point nerve block.

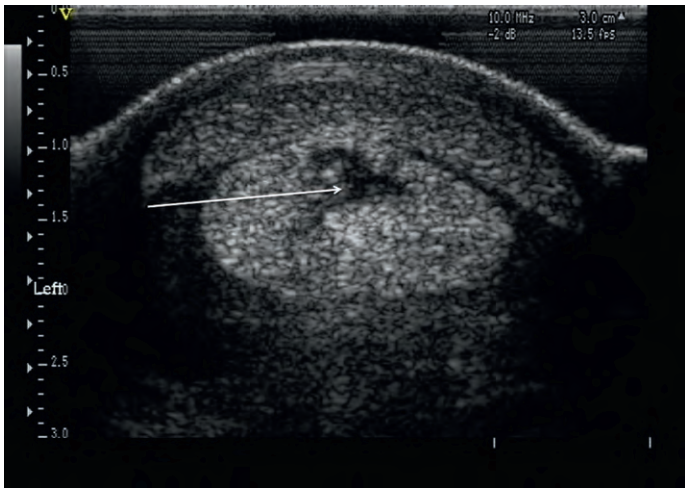


Figure 16.18 Transverse ultrasonograph taken just proximal to the metacarpophalangeal joint showing a large hypoechoic defect (arrow) in the deep digital flexor tendon representative of over-strain injury of the deep digital flexor tendon.

Diagnosis

- Ultrasonography reveals small, distinct, often circular, short, focal hypoechoic areas within the DDFT within the DFTS (Figure 16.18). Concurrent mineralization is common.
- Marginal tears of the DDFT are difficult to identify ultrasonographically.

Treatment and prognosis

- Marginal tears or defects should be debrided tenoscopically followed by a graduated controlled exercise programme. This may be combined with intralesional regenerative therapies (stem cell or PRP injection). The prognosis for return to previous performance is 20–40% depending on the extent of the tear.
- Intrasubstance small focal core lesions of the DDFT should be treated with rest and a graduated controlled exercise programme under ultrasonographic guidance. This may be combined with extracorporeal shock wave therapy, intralesional treatments, and/or intrathecal anti-inflammatory medication. Annular ligament desmotomy may be helpful if there is concurrent thickening of the palmar annular ligament. The prognosis for return to previous performance is guarded to poor.

Tendon lacerations

Aetiology

- Percutaneous tendon injuries arise from trauma to the distal limb mostly during weight-bearing (over-reaching, wire-cuts, kicks)
- Usually accompanied by extensive contusion and contamination.
- Entry wounds can be deceptively small and hide extensive internal damage.

Clinical signs

- Complete transection of the SDFT results in sinking of the fetlock.
- Transection of the DDFT causes the toe to lift off the ground.
- Severance of both flexor tendons as well as the suspensory ligament causes total breakdown of the stay apparatus and the fetlock rests on the ground while the toe lifts off the ground.
- Lacerations of the long or common digital extensor tendons results in dorsal 'knuckling' of the fetlock and tripping. The horse has difficulty placing the foot flat on the ground.
- Lacerations of the palmar/plantar aspect of the distal metacarpus/metatarsus usually involve the DFTS (see 'Septic tenosynovitis of the digital flexor tendon sheath (DFTS)').
- Horses suffering from flexor tendon laceration are usually in severe pain and need immediate attention.

Treatment

- General principles of wound care apply and include debridement, repair, wound closure and mechanical support.
- Even though lacerated extensor tendons usually heal without suturing, a recent study has suggested better outcome when surgical repair was attempted. A cast or a firm bandage with a toe-extension shoe may be necessary to help the horse place the foot and prevent 'knuckling'.
- Small transverse skin lacerations need to be extended proximally and distally with an elongated S-shaped incision to fully assess the effect of the laceration on the underlying tendons. It also allows the surgeons to gain sufficient access to mobilize, debride and suture the recoiled tendon ends.
- Suturing of lacerated flexor tendons is recommended if there is not a large defect after debridement. The locking-loop or three-loop pulley pattern is used.
- When the damaged tendon ends can not be apposed, an implant can be used to maintain alignment of the tendon ends during healing. Carbon fibre, terylene (polyester), autologous extensor tendon grafts and ply-L-lactic acid (PLLA) have been used successfully.
- The injured limb should always be supported post-operatively with a cast for 8 to 12 weeks. Following cast removal, the horse should be stall rested for a minimum of 3 more months. During this time the level of exercise should be slowly increased under ultrasonographic guidance. Return to full function takes 8 to 12 months.

Prognosis

- Guarded for flexor tendon laceration, particularly if more than one tendon is involved (45–59%). Horses can return to soundness if no complications occur.
- If the laceration occurs within the DFTS, the horse has an unfavourable prognosis for soundness.
- Unfavourable complications are infection, necrotizing (melting) tendinitis, flexural deformity, peritendinous adhesions and cast complications.
- Favourable for extensor tendon lacerations with approximately 75% of horses returning to soundness.
- Healing of an extensor tendon laceration may result in a stringhalt-like gait.

Desmitis of the suspensory ligament

Injuries of the suspensory ligament can occur at the level of:

- its proximal attachment to the proximal palmar/plantar cortex of the metacarpus/metatarsus (proximal suspensory desmitis).
- the main body (body desmitis).
- the branches that insert on the proximal sesamoid bones (branch desmitis).

Proximal suspensory desmitis

Aetiology

- Very common cause of hind limb lameness in all horses, especially sport horses.

- Exact aetiology is unknown, but preceding degeneration and/or excessive strain that occurs with hyperextension of the fetlock may be predisposing factors.
- Horses with a straight hock conformation and long, sloping pasterns are at an increased risk.
- Exercise in deep footing may also be a risk factor.
- Systemic proteoglycan storage disease affecting ligament matrix has been suggested as a possible cause of degenerative suspensory desmitis.
- Hereditary condition in Peruvian Paso and their cross breeds may lead to progressive degeneration of the suspensory ligament and hyperextension of the fetlock joint. A clinically similar condition can be seen affecting old brood mares.
- Transient proximal suspensory desmitis may result in degenerative neuropathy of the deep branch of the lateral plantar nerve, which may be responsible for persistent neurogenic pain, even after healing of the suspensory ligament.

Clinical signs

- Acutely and severely affected horses may have localized oedema, heat and swelling in the palmaro-/plantaro-proximal metacarpal/metatarsal region. The medial palmar/plantar vein may be distended.
- More frequently horses are presented with non-specific chronic hind limb lameness without localizing signs.
- Typically lameness is most visible when lunging the horse on a circle with the affected limb on the outside.
- Pain originating from the proximal part of the forelimb suspensory ligament is often seen in young horses in which a recent increase in training has been initiated.
- Hind limb lameness is often exacerbated by proximal and distal limb flexions.
- Diagnostic anaesthesia is required to localize the site of pain. Anaesthesia of the deep branch of the lateral palmar/plantar nerve abolishes lameness associated with the proximal part of the suspensory ligament. However, these nerve blocks may also improve lameness associated with the tarsal bones and intertarsal joints, as well as the distal limb.

Imaging

- Radiographic abnormalities include trabecular sclerosis and/or lysis, or entheseophyte formation. In the majority of horses no radiographic abnormalities are seen.
- Ultrasonographic examination of the origin of the suspensory ligament is difficult because of overlying structures and the presence of muscle tissue in the ligament.
- Ultrasonographic abnormalities are the presence of focal or diffuse hypoechogenicity (Figure 16.19), enlargement of the suspensory ligament and/or roughening of the palmar/plantar metacarpal/metatarsal cortex due to entheseophyte formation.
- High-field MRI is considered the gold standard for diagnosis. Abnormalities consist of signal hyperintensities within the substance of the ligament, enlargement of the CSA, irregular contour or focal lysis of the palmar/plantar cortex and/or endosteal remodelling changes like trabecular thickening, sclerosis and bone oedema.
- The sensitivity and specificity of ultrasonography have been found to be poor in comparison with MRI, especially in proximal suspensory desmitis of hind limbs.

Treatment

- Box rest followed by a regimen of increasing exercise (e.g. 30 days of box rest, 30 days of walking in hand, 30 days of walking and trotting). After this period, ultrasonographic appearance is re-assessed. A further 3 months of controlled exercise is usually necessary for hind limb proximal suspensory desmitis.
- Extracorporeal shock-wave therapy consisting of three bi-weekly applications.
- Anti-inflammatory infiltrations of the periligamentous region with a corticosteroid.
- Regenerative intra-lesional injections have also been used (PRP, bone-marrow derived mesenchymal stem cells, bone-marrow concentrate). However, these injections may help with healing of the suspensory ligament but may not directly target the pain causing the lameness.
- Neurectomy of the deep branch of the lateral plantar or lateral palmar nerve has a high incidence of success and a low incidence of complications. Horses are able to go back into work at 6 to 8 weeks after neurectomy. Progressive worsening of the suspensory desmitis may occur in exceptional cases.

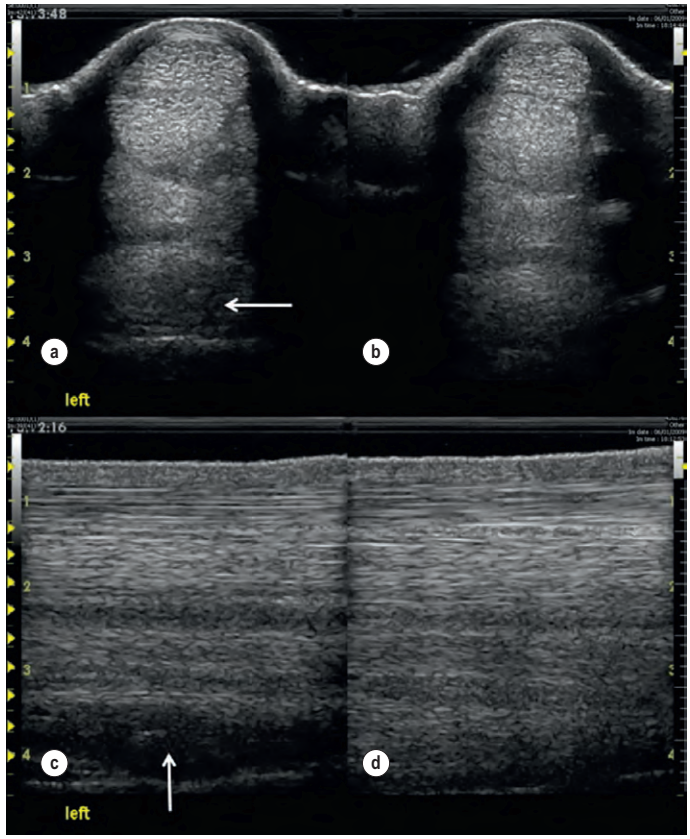


Figure 16.19 Transverse (a and b – lateral to the left) and longitudinal (c and d – distal to the left) ultrasonographs from a horse with proximal suspensory desmitis. Note the hypoechoic region in the proximal part of the suspensory ligament of the left forelimb (a and c – arrows), visible in both transverse and longitudinal images, which is not present on the right (b and d).

Prognosis

- The overall prognosis for return to exercise is 80% for forelimbs and 14% for hind limbs with conservative treatment.
- Using shock wave therapy the prognosis for hind limb proximal suspensory desmitis can be improved to approximately 50%.
- Neurectomy of the deep branch of the lateral plantar/palmar nerve enables approximately 75% of horses to return to their previous level of performance.

Desmitis of the body of the suspensory ligament

Aetiology

Associated with exercise at high speed in racehorses

Clinical signs and diagnosis

- Obvious localizing signs of heat, swelling and pain on palpation.
- Lameness may be variable, and some horses (Standardbreds) may tolerate desmitis and continue to work.
- Ultrasonography reveals the extent of structural damage. Extensive hypoechoic areas may persist for a long time despite resolution of clinical signs in many horses.
- Always take radiographs to check for distal splint bone fractures.

Treatment

- Rest, ice, and compression followed by a graduated, ultrasound-guided slow return to exercise.
- Standardbred racehorses may be able to tolerate the presence of desmitis and continue athletic function while being managed symptomatically.
- Extracorporeal shock wave therapy.
- Intralesional anti-inflammatory medication: HA, polysulphated glycosaminoglycans.
- Intralesional regenerative therapies: PRP, bone-marrow concentrate, bone-marrow derived mesenchymal stem cells, (IGF-I, TGF-beta).
- Percutaneous splitting to decompress core lesions and promote extrinsic repair.

Suspensory branch desmitis

Incidence

- May occur in the lateral or medial branch of forelimbs or hind limbs of all horse types.
- Most common injury of the suspensory ligament.

Clinical signs

- Commonly there is swelling of the injured branch. Always compare carefully with opposite branch and with the branches of the contralateral limb.
- Lameness may vary depending on the severity of the lesion.
- Periligamentous fibrosis is typical for chronic (re)injuries.
- Lesions involving the dorsodistal portion of the suspensory branch may be contained within the palmar/plantar pouch of the fetlock joint and cause marked synovitis and fetlock joint effusion.

Imaging

- Ultrasonography may reveal:
  - Small, focal to large areas of hypoechogenicity.
  - Enlargement of the affected branch (always measure and compare).
  - Loss of parallel fibre pattern near attachment to sesamoid bone.
  - Irregular osseous contour to abaxial surface of the proximal sesamoid bone.
  - Presence of periligamentous echogenic layer (fibrosis).

Extensive hypoechoic areas may persist for a long time despite resolution of clinical signs.

- Always take radiographs to check for distal splint bone fractures, apical sesamoid fractures and sesamoiditis.

Treatment

- Treatment is similar to that for tendinitis and body desmitis.
- Lesions involving the dorsal portion of the suspensory branch that may extend into the palmar/plantar pouch of the fetlock joint are amenable to arthroscopic debridement. In one report arthroscopic debridement of intrasynovial lesions enabled 13/18 horses to make a complete recovery.

Prognosis

- Guarded because of the high incidence of reoccurrence and the slow, unpredictable rate of healing.
- Marked periligamentous fibrosis appears to affect outcome negatively.
- Intra-articular lesions have a fair prognosis after arthroscopic debridement.

Desmitis of the accessory ligament of the deep digital flexor tendon

Anatomy

The inferior check ligament provides stability to the carpus in extension and it shares the tensile load of the DDFT in the midstance phase of the stride.

Clinical signs

- Swelling, heat and pain in the region of the proximal metacarpus.
- Horses are typically >10 years old.
- Injury of the accessory ligament of the deep digital flexor tendon in the hind limb is rare as it is a much smaller structure.
- Ponies or cob-type horses are at increased risk.
- Injury in hind limbs may be accompanied by flexural deformity of the distal limb with knuckling forward of the fetlock joint and elevation of the heel off the ground when standing.

Ultrasonography

- Ultrasonographic appearance varies from diffuse hypoechogenicity of the ligament to focal hypoechoic areas in the substance of the ligament. An injured ligament is always enlarged. Scar tissue and adhesions are typical in chronic re-injury cases.
- Echogenic extensions of the accessory ligament to the borders of the SDFT indicates adhesion formation.

Treatment

- Rest and a regime of gradually increasing exercise dependent on ultrasonographic appearance. Avoid free turn-out during convalescence. Convalescence may take from 3 to 9 months.
- Desmotomy or desmectomy of the accessory ligament of the deep digital flexor tendon is indicated:
  - in horses that fail to respond favourably to conservative management by 3 months.
  - in horses with adhesions.
  - in chronic cases.

Prognosis

- Acute desmitis in fore and hind limbs has a favourable prognosis.
- The prognosis is guarded to poor without surgery for:
  - chronic desmitis.
  - adhesions between the accessory ligament and the SDFT.
- The prognosis is poor for correction of postural abnormalities of the hind feet, even with surgery.

FURTHER READING

Auer JA, Stick JA (2012) Equine surgery. 4th edn. Saunders, St Louis

Baxter GM (2011) Adams and Stashak's lameness in horses. 6th edn. Wiley-Blackwell

Butler JA, Colles CM, Dyson SJ, Kold SE, Poulos PW (2000) Clinical radiology of the horse. Blackwell Scientific Publications, Oxford

Ross MW, Dyson SJ (2003) Diagnosis and management of lameness in the horse. Saunders, St Louis

APPENDIX

Differential diagnoses of lower-limb lameness

Acute, severe lower-limb lameness

1. Septic pododermatitis ('pus in the foot').
2. Laminitis.
3. Septic arthritis or synovitis.
4. Severe strain of DDFT.
5. Severe sprain of the DIP joint or fetlock joint.
6. Fracture of the proximal phalanx.
7. Intra-articular fractures of the fetlock joint.
8. Fracture of the distal phalanx.
9. Fracture of the navicular bone.
10. Fracture of the middle phalanx.

Insidious onset, low-grade, uni- or bilateral foot lameness

1. Chronic foot imbalance.
2. Chronic subsolar bruising ('pedal osteitis').
3. Navicular disease.
4. Tendinitis DDFT.
5. Collateral desmitis DIP joint.
6. Traumatic synovitis of the DIP joint.
7. OA of the DIP or PIP joint.
8. Keratoma.
9. Osseous cyst-like lesions of the distal or middle phalanx.

Lameness with swelling in the fetlock region

1. OA of the fetlock joint.
2. Intra-articular fractures of the fetlock joint.
3. Traumatic sprain of the fetlock joint.
4. Tenosynovitis of the DFTS.
5. Annular ligament syndrome.
6. Desmitis of the suspensory ligament branches (and sesamoiditis).
7. OCD and osseous cyst-like lesions in the fetlock joint.
8. 'Low bow' (tendinitis SDFT).
9. Septic arthritis of the fetlock joint.
10. Sesamoid bone fracture.
11. Distal sesamoidean ligament desmitis.
12. Distal splint bone fracture.
13. Villonodular synovitis.
14. Tendosynovitis of the DDFT and the DFTS.
15. Septic pododermatitis ('pus in the foot').
16. Lymphangitis.

Lameness referable to the proximal palmar metacarpus

1. Proximal suspensory desmitis.
2. Proximal metacarpal stress injury.
3. Incomplete fractures of the proximal palmar metacarpal cortex.
4. Inferior check ligament desmitis.
5. Proximal 'splints'.

Chapter 17

Orthopaedics 3. The proximal limbs

Marcus J. Head, Alistair Barr

CONTENTS

17.1 Carpus 370
Anatomy 370
Traumatic arthritis, osteoarthritis (degenerative joint disease) 370
Carpal fractures 370
Osteochondritis dissecans (OCD) 372
Subchondral cyst-like lesions (SCLs) 372
Septic arthritis 372
Angular limb deformities 372
Carpal canal syndrome 374
Osteochondroma of the distal radius 374
Soft-tissue injuries of the carpus 374
17.2 Elbow 375
Anatomy 375
Ulnar fractures 375
Subchondral cystic lesions (SCLs) 375
Septic arthritis 376
Capped elbow 376
17.3 Shoulder 376
Anatomy 376
Osteochondritis dissecans (OCD) 376
Subchondral cystic lesions 377
Fractures 377
Traumatic luxation 378
Bicipital bursitis 378
Diseases of the biceps tendon 378
Suprascapular neuropathy ('sweeney') 378
17.4 Hock 378
Anatomy 378
Osteoarthritis of the small hock joints ('bone spavin') 379
Osteochondritis dissecans 380
'Bog spavin' 380
Thoroughpin 380
Curb 381
Displacement of the superficial digital flexor tendon from the point of the hock/SDFT luxation 381
Gastrocnemius tendonitis 381
Rupture of the peroneus tertius 382
Collateral ligament injury 382
Tarsal bone collapse 382
Intra-articular fractures 383
Luxations 383
Capped hock 383
Calcaneal bursitis 383
Talocalcaneal osteoarthritis 384
17.5 Stifle 384
Anatomy 384
Osteochondritis dissecans 384
Subchondral bone cysts 384
Upward fixation of the patella 385
Patella luxation 386
Patellar fractures 386
'Gonitis' 386
Ligament injuries 386
Meniscal injury and associated meniscotibial ligament tears 386
Osteoarthritis 387
Calcinosis circumscripta 387
Fibrotic/ossifying myopathy 387
17.6 Hip 387
Anatomy 387
Osteoarthritis 387

© 2013 Elsevier Ltd
DOI: 10.1016/B978-0-7020-2801-4.00017-1

Luxation 387
Sepsis 387
Fractures 387
Further reading 388

17.1 CARPUS

Anatomy

Bones

The carpus normally contains seven small carpal bones in two rows (see Figures 25.51–25.54).

Proximal row:

- Radial (Cr).
- Intermediate (Ci).
- Ulnar (Cu).
- Accessory (ACB).

Distal row:

- 2nd (C2).
- 3rd (C3).
- 4th (C4).

A first carpal bone is found in about 50% of horses and may be unilateral. It is usually about the size and shape of a pea and is found embedded in the medial ligament of the carpus, behind the second carpal bone. A fifth carpal bone is also occasionally seen on the lateral aspect of the carpometacarpal joint.

The carpal bones articulate proximally with the distal radius, which has three articular facets relating to the proximal surfaces of the radial, intermediate and ulnar carpal bones. The lateral aspect of the distal radius is phylogenetically the distal ulnar epiphysis and is seen as a separate ossification centre in foals. The line of fusion to the rest of the distal radial epiphysis closes radiographically before 12 months of age but often persists into adulthood as a groove on the distal radial articular surface. The epiphysis fuses with the metaphysis at 20–24 months of age. A linear, mineralized density is sometimes seen on the caudolateral aspect of the distal antebrachium, representing the vestigial distal ulna. The distal row of carpal bones articulate distally with the second, third and fourth metacarpal bones.

Joints

The articulations between the carpal bones are complex, and some consist of two or three separate facets. Altogether, there are approximately 26 separate articulations. These are divisible into three principal joint levels:

1. Antebrachiocarpal.
2. Middle carpal (intercarpal).
3. Carpometacarpal.

There are usually two separate synovial cavities:

1. Antebrachiocarpal.
2. Middle carpal and carpometacarpal.

The antebrachiocarpal joint moves with a rotating and gliding action. The midcarpal joint has a hinge-like action while the carpometacarpal joint is capable of very little movement. During flexion, the articular surfaces of the antebrachiocarpal and midcarpal joints become widely separated dorsally but remain apposed at their palmar margins. The intermediate and ulnar carpal bones tend to move as one unit, but the radial carpal bone is not displaced as far from the distal row of carpal bones on flexion. Its dorsodistal margin is therefore separated from the intermediate carpal bone on a flexed lateromedial radiograph.

The fibrous palmar carpal joint capsule is dense and closely attached to the palmar aspect of the carpal bones. Palmarly, it forms the smooth dorsal wall of the carpal canal. It is continued distally to form the accessory head of the deep digital flexor tendon (inferior check ligament). The carpal fascia on the palmar aspect of the carpal region forms the flexor retinaculum (transverse carpal ligament) between the free palmar edge of the accessory carpal bone and the medial aspect of the carpus. This completes the carpal canal, which contains the superficial and deep digital flexor tendons, blood vessels, and a portion of the accessory ligament of the superficial digital flexor tendon (ALSDFT/superior check ligament).

Traumatic arthritis, osteoarthritis (degenerative joint disease)

Aetiology and pathogenesis

Equine carpal joint disease is seen most commonly as an 'occupational disease' of racehorses and occurs less commonly in horses used for other purposes. A form of traumatic arthritis/osteoarthritis (OA) appears to be the usual underlying disease entity. The vast majority of cases are a consequence of the chronic repetitive trauma suffered by the joint as a result of training and racing. Adaptive remodelling of bone in response to training may have deleterious consequences for the integrity of the joint as a whole. Initial osteoclastic resorption may weaken the bone, predisposing to microscopic or gross fracture. Subsequent densification increases the stiffness of the bone, increasing the peak load on the overlying articular cartilage. Chronically accumulating damage of OA may give rise to clinical signs of uni- or bilateral forelimb lameness in its own right, as well as acting as a precursor to more acute injuries such as carpal chip and slab fractures.

Carpal OA affects the middle carpal joint more commonly than the antebrachiocarpal joint in Thoroughbred racehorses. The articular surface damage is concentrated on the medial aspect of the joint and particularly involves the radial and third carpal bones. The articular margins and surfaces on the lateral aspect of the joint often appear relatively normal both radiologically and arthroscopically. This distribution of lesions is hypothesized to be a consequence of biomechanical influences; the ability of carpal bones to dissipate energy through lateral movement is thought to be more limited on the medial aspect, predisposing to fracture on this side of the joints.

Carpal OA is seen in horses used for other disciplines, although it is an uncommon cause of lameness in non-racehorses. Diagnosis and management is the same for other joints.

Carpal fractures

Carpal 'chip' fractures are frequently seen in association with OA and have been commonly considered as a potential cause of 'secondary' OA. Although this sequence of events undoubtedly occurs, the alternative scenario – that the arthrosis causes or predisposes to the fractures – may well be more common and more important, particularly with respect to distal radial carpal chip fractures. A similar role of carpal OA/subchondral disease in predisposing to third carpal bone slab fractures has also been suggested, and clinical evidence supports this as almost all third carpal slab fractures are pathological and occur through obviously diseased bone. Such a viewpoint puts the acute injury into perspective as part of a chronic disease process and thereby establishes the limitations on the success that might be expected with surgical treatment (e.g. fracture fragment stabilization or removal). The prodromal changes may be present in a large number of racehorses, not all of which go on to sustain a fracture. These changes can

Figure 17.1 Dorsolateral-palmaromedial oblique radiograph of the carpus. There is a large, slightly displaced fragment from the dorsodistal aspect of the radial carpal bone (Cr 'chip fracture'). Note also the soft tissue swelling on the dorsal carpus (caused by synovial effusion); the roughening and modelling of the dorsal aspect of Cr, above the fragment (indicating entheseous reaction) and the overall increase in density of Cr in the region of the fracture when compared to the nearby third carpal bone density. The last two observations confirm that these injuries occur as a result of prodromal bone changes, and ARE NOT a one-off loading event (so-called 'bad step').

be present with minimal lameness or mild bilateral lameness, confounding detection clinically. Additionally, these changes often cause no synovial effusion, giving the impression that the fracture occurs instantaneously.

- Chip fractures occur at the dorsal margins of the bones forming the antebrachiocarpal and middle carpal joints. Each individual chip fracture affects only one articular surface of the parent bone, although there may be multiple chips affecting different bones, different joints and different limbs in the same horse. The most common sites are the distal margin of the radial carpal bone (Figure 17.1) and the proximal margin of the third carpal bone, i.e. on the dorsomedial margin of the middle carpal joint. The distolateral radius, within the antebrachiocarpal joint is another predilection site.
- Slab fractures (see Figure 25.16) extend right through a carpal bone from the proximal to the distal articular surface. They occur most commonly in the frontal plane and primarily affect the dorsomedial aspect (radial facet) of the third carpal bone. Because they involve larger bone fragments and two articular surfaces they are a more serious injury than a chip fracture, and affected horses have a correspondingly more guarded prognosis for return to athletic activity. Slab fractures in the sagittal plane are occasionally seen.
- Incomplete fissure fractures of the third carpal bone have been described and may cause lameness with few localizing clinical signs.
- Accessory carpal bone fractures are seen most commonly in jumping horses and often there is a history of a fall. The fracture usually occurs in the frontal plane in approximately the middle of the bone.
- Comminuted fractures of one or more carpal bones cause severe disruption of the articular surfaces and often result in instability of the carpus. They are usually due to severe trauma such as falls or road traffic accidents.

Clinical signs

- Lameness localized to the carpus. There may be obvious unilateral lameness or a more insidious bilateral lameness manifested as shortening of the stride and abduction of the distal limb during protraction to reduce the necessity for carpal flexion.
- Swelling, especially carpal joint effusion in the early stages, although – as mentioned previously – synovial effusion may not be present in the early stages of subchondral bone disease. Fibrous thickening on the dorsal aspect of the joint occurs with time and obscures the clear contours of the dorsal carpal bone margins.
- Pain on joint flexion and sometimes on palpation of the joint margins (especially dorsomedial aspect of Cr and dorsolateral aspect of the distal radius).
- Positive response to carpal flexion test.
- Restricted range of carpal flexion.
- Severe fractures may cause crepitus and instability.

Radiology

The four standard weight-bearing projections of the carpus should be supplemented with flexed lateromedial views (to separate the dorsal margins of the radial and intermediate carpal bones) and flexed dorsoproximal–dorsodistal oblique views (to evaluate sclerosis, lysis and fracture of the individual carpal bones) (see Figures 17.1 and 25.55).

Soft-tissue swelling

Soft-tissue swelling may be a result of synovial effusion or thickening of the synovial membrane/joint capsule or periarticular tissues. It is useful to note on radiographs the level of maximal swelling in relation to the joints in the carpus (i.e. antebrachiocarpal joint, middle carpal joint or carpometacarpal joint) because this can help in identifying the specific carpal joint principally involved.

Entheseophytes

The carpal bones are arranged to allow for their slight displacement in a horizontal plane when the carpus is loaded. The carpal bones are linked together by ligaments which constrain this movement and absorb some of the imposed load. Overloading of this system may result in damage to the origins and insertions of the intercarpal ligaments on the dorsal aspect of the carpal bones, which results in non-articular periosteal new bone formation at the sites of these attachments, i.e. entheseophytes. Once formed, this bone tends to persist throughout the horse's life, although it may remodel and become smoother in outline. Irregular, non-articular, periosteal new bone on the dorsal surface of the carpal bones in young racehorses in training may, therefore, be an indication that overloading of the carpal joints is occurring and that more serious articular surface damage may coexist. Such entheseophytes may, however, be an incidental observation without a great deal of clinical significance when seen later in life.

Osteophytes

Periarticular new bone is one of the cardinal radiological features of OA. Osteophytes may develop as a modelling response to joint

overload and damage. They may reflect an attempt to increase joint surface area and thereby reduce loading per unit surface area, and also to increase joint stability. They may thus be viewed as part of the joint's adaptive response to disease. Periarticular osteophytes tend to be seen primarily on the dorsomedial aspect of the carpal joints, and modelling of the dorsodistal margin of the radial carpal bone is particularly common.

Paramarginal subchondral bone lysis

Loss of subchondral cortex in a narrow strip approximately 5 mm palmar to the dorsal margin of the distal surface of the radial carpal bone is a common radiological finding in horses with OA of the midcarpal joint. It is seen as a small concave area of radiolucency in the subchondral cortical bone of the distal articular surface just palmar to the dorsodistal margin and is generally most apparent on the DLPMO projection. On the flexed dorsoproximal–dorsodistal oblique projection of the distal row (third carpal skyline) fissuring of the dorsal cortex of C3 may also be appreciated.

Subchondral bone densification

Densification (sclerosis) of the subchondral bone in the radial facet of the third carpal bone is recognized in young racehorses. This feature is best demonstrated on the dorsoproximal–dorsodistal oblique view of the third carpal bone and is seen as an increase in bone density and loss of the normal trabecular pattern. It is probably an adaptive change in response to increased loading with training, but, by stiffening the subchondral bone, it may increase the load on the overlying articular cartilage and thereby predispose it to damage. Densification and the resulting increase in stiffness, with transfer of loading, can in itself be painful and cause lameness: physiological imaging techniques such as scintigraphy and MRI often demonstrate increased uptake/high signal even with comparatively minor radiological change.

OA of the carpometacarpal joint

The equine carpometacarpal joint is rarely overtly affected by OA, although its synovial cavity communicates with that of the frequently affected midcarpal joint. Old ponies are sometimes affected with OA of the carpometacarpal joint. The associated radiological changes bear similarities to 'bone spavin' in the hock with prominent periosteal new bone proliferation and subchondral bone lysis. Like 'bone spavin', these changes may rarely progress to fusion of the affected joints.

Treatment

1. Traumatic arthritis – see Chapter 15.
2. Osteoarthritis – see Chapter 15.
3. Fractures: most carpal fractures can, in theory, be managed either surgically or conservatively. Surgical intervention is indicated particularly for horses with chip or slab fractures that are intended for athletic use. Surgery aims to restore articular congruency and stability so that the development of secondary OA is minimized. The limitations on this where the fractures are actually a product of existing OA have already been alluded to.
   - Chip fractures are generally treated surgically by removing the fragments under arthroscopic visualization. Abnormal articular tissues – particularly soft, crumbly subchondral bone – may be debrided at the same time.
   - Slab fractures are usually treated by lag-screw fixation. Initial debridement and reduction may be accomplished using arthroscopic visualization.
   - Accessory carpal bone fractures commonly form fibrous rather than bony unions. Techniques for lag-screw repair have been described but are difficult due to the curved shape of the bone and the comminuted nature of many of the fractures.
   - Comminuted fractures of one or more carpal bones usually end a horse's athletic career. Salvage for breeding or as a pet may be feasible by combining internal fixation and cast support, with or without combined partial or pancarpal arthrodesis. Comminution of multiple carpal bones is usually an indication for euthanasia.

Osteochondritis dissecans (OCD) (see Chapter 15)

OCD is rare in the carpus but is occasionally seen in foals. It may be associated with disease in other joints.

Subchondral cystic lesions (SCLs) (see Chapter 15)

These have been reported in the carpus but may not be of clinical significance; early reports probably overstated their importance as a cause of lameness. They are seen frequently in the ulnar carpal bone and in association with the presence of first and fifth carpal bones; they are invariably of no significance in these situations. Occasionally, a large 'cyst' is identified in the radial carpal bone or distal radius, sometimes with obvious articular communication, which should be considered potentially more important and evaluated comprehensively to determine its significance.

Septic arthritis

See Chapter 15.

Angular limb deformities

The term 'angular limb deformity' refers to lateral (valgus) or medial (varus) deviation of the distal limb from the sagittal plane. Thus 'carpal valgus' refers to outward deviation of the distal limb originating at the level of the carpus (Figure 17.2). These deformities are usually seen in young foals and can be congenital or acquired.

Aetiology

- Congenital deformities: cause often unknown, but suggestions include malpositioning of the foetus in utero, prematurity/dysmaturity, genetic factors, nutritional influences on the mare, and teratogens.
- Acquired: most commonly due to physeal injury or asymmetric loading due to excessive weight-bearing, e.g. as a result of overnutrition or contralateral limb injury, excessive exercise or improper foot trimming. In the case of carpal deformities, the distal radial physis is usually the site affected.

Pathogenesis

- Congenital laxity of the ligamentous support of the carpus and/or hypoplasia (inadequate ossification) of the small carpal bones may result in angular deformity. If left unsupported, the inadequately ossified bones may become crushed and deformed as the foal loads the limbs after birth.
- Acquired deformities usually develop as a result of asymmetric growth from the distal radial physis, which may be due to excessive loading of one side of the physis.

Figure 17.2 Example of carpal valgus. The distal limb is deviated laterally from the level of the carpus.

History

Important facts to establish include:

- The age of the foal.
- If the foal was premature or smaller than normal at birth.
- If the deformity was present at birth, and if not, when it first appeared.
- How the deformity has developed since it was first seen (i.e. improving, deteriorating or static).
- If there has been any associated lameness or other signs.

Physical examination

The following points in particular should be established:

- The site or sites of deformity.
- The direction of the deformity: valgus or varus.
- The degree of deviation: mild, moderate or severe.
- Evidence of instability: can the deformity be manually reduced?
- Evidence of lameness.
- Signs of inflammation: pain, heat or swelling.

Radiography

It is important to obtain dorsopalmar views centred on the carpus but including as much as possible of the distal radius and proximal metacarpus in order to be able to evaluate the degree of deformity present.

Radiology

1. Morphological changes: various combinations of the following features may be seen:
   - Irregularity of the line of the physis; often appears particularly disturbed on the convex side of the deformity.
   - Metaphyseal and epiphyseal flaring.
   - Wedge-shaped epiphysis.
   - Diaphyseal remodelling with asymmetry in cortical thickness.
   - Hypoplastic, rounded, collapsed, or subluxated carpal bones.
2. The radiographs may also be used to gain an estimate of the degree of angulation present. Traditionally, a piece of clear X-ray film is placed over the dorsopalmar radiograph and lines drawn down the centre of the radius and the centre of the third metacarpal bone. For single-site angulations, the intersection of these lines gives some indication of the level within the carpus at which the deformity is occurring and the degree of angulation (see Figure 25.56). The use of digital radiography and computer software now enables these measurements to be made more easily.

Treatment

Some degree of angular limb deformity is not uncommon in neonatal foals and often improves dramatically during the first 2 weeks of life without specific treatment.

Certain management principles are helpful in dealing with angular limb deformities regardless of what other techniques are employed.

- Foals should generally be confined to a large box or small yard while the deformity is present, as excessive exercise on an angulated limb tends to exacerbate asymmetric overloading of the physis, carpal bones and other joint structures.
- Limb angulation leads to asymmetric wear of the hoof. Foals with carpal valgus tend to wear the medial aspect of the hoof wall excessively. The hoof should be rasped regularly to bring it back into balance. This may be combined with the use of glue-on plastic shoes with medial (in the case of carpal valgus) or lateral (for carpal varus) extensions.
- Affected foals should not be allowed to become overweight. This may require restricting the mare's diet to control milk production, or it may require early weaning.
- Neonatal foals with deformity due to ligamentous laxity or carpal bone hypoplasia benefit from external support of the limb with either a well-padded splint or tube cast for 2–4 weeks. Splints should be reset daily and casts changed after 2 weeks to reduce the chance of development of pressure sores. In foals, there is usually little to be gained by external support where there is no evidence of instability, the deformity cannot be reduced manually, and the carpal bones do not appear hypoplastic radiographically.
- Foals with persistent deformities due to asymmetric growth from the distal radial physis are candidates for surgical intervention if they fail to respond to the measures outlined above, or if the age of the foal indicates that conservative measures will not have time to be effective. Surgery aims to manipulate growth at the

distal radial physis to cause limb straightening. This may involve either:

- temporary transphyseal bridging with either staples, screws and wire, or a transphyseal screw, or
- hemicircumferential periosteal transection.

Both techniques depend on continued growth of the bone to straighten the limb, and there are therefore time limits after which they are likely to be much less effective. In the case of angular limb deformities of the carpus, the surgery should be undertaken before the foal is 12 weeks of age. The earlier surgery is performed, however, the more rapid and complete will be the response.

Temporary transphyseal bridging aims to slow the rate of growth on the more rapidly growing (i.e. convex) side of the physis. It is therefore performed medially in the case of carpal valgus deformity. The use of two screws (usually fully threaded, 6.5-mm diameter AO cancellous screws) with one or more figure-of-eight wires (18G) joining them, has certain advantages over the use of staples. The two screws can be placed independently, unlike the tines of a staple, and immediate compression across the physis is achieved as the wire(s) is tightened. There is also less risk of extrusion of the implants as the foal continues to grow. Alternatively, a single 4.4-mm cortical screw can be inserted diagonally across the physis on the medial side. The implants must be removed as soon as the limb is straight, or overcorrection and deviation in the opposite direction may occur.

Hemicircumferential periosteal transection: the periosteum is postulated to exert a restraining influence on longitudinal growth of long bones. By releasing this restraint on one side of the bone, it is possible to induce asymmetric growth and thereby correct existing deformities. The transection is thus done on the side of the bone that is growing less quickly, i.e. the concave side (lateral side in the case of carpal valgus). The periosteal incision is made about 2.5 cm proximal to the physis. A simple horizontal incision is probably sufficient though many surgeons create an inverted 'T' incision and then elevate the flaps of periosteum created from the bone. In the case of carpal valgus deformities the rudimentary distal ulna should also be transected at the caudal end of the horizontal periosteal incision.

Hemicircumferential periosteal transection has certain advantages over temporary transphyseal bridging:

- There is no need for a second surgery to remove implants.
- There is less risk of postoperative infection because there are no implants.
- There appears to be no risk of overcorrection.

Hemicircumferential periosteal transection and temporary transphyseal bridging may be employed simultaneously in severely affected foals. However, more recently, the effectiveness of periosteal transection has been challenged.

Extracorporeal shockwave therapy has been used for the treatment of ALD. However, although subjectively the results may appear impressive, no controlled studies have been undertaken, to date. Whenever therapy for ALD is being considered, it must be remembered that many cases resolve spontaneously, without the need for treatment.

Carpal canal syndrome

Aetiology and pathogenesis

This syndrome is caused by trauma or a space-occupying lesion within the carpal canal leading to compression of the soft tissues within the canal (e.g. accessory carpal bone fracture, osteochondroma of the distal radius, tendinitis of the flexor tendons). It is questionable that this syndrome ever exists as a primary condition and, in the vast majority of cases, is NOT analogous to the human condition, carpal tunnel syndrome, which is associated with nerve compression without a primary lesion. Therefore it is strongly recommended to comprehensively evaluate the horse (using diagnostic imaging and, if necessary, tenoscopy) for an inciting cause.

Clinical signs

- Distension of the carpal sheath with synovial fluid.
- Reduced range of carpal flexion.
- Pain on carpal flexion.
- Reduced pulse volume in the digital vessels distal to the carpus.

Treatment

- Treat the primary lesion if possible (frequently requires tenoscopic examination of the carpal sheath).
- Resection of the carpal flexor retinaculum (rarely indicated).

Osteochondroma of the distal radius

This is a bony exostosis that develops on the caudal aspect of the distal radius. Its cortex is continuous with that of the distal radius and it has a cartilage 'cap'.

Aetiology and pathogenesis

There is localized abnormal growth of cartilage from an area of metaphyseal periosteum that subsequently undergoes endochondral ossification. It appears to be distinct from the condition of 'multiple exostoses', which is thought to be inherited.

Clinical signs

Signs are as for carpal canal syndrome plus the bony protruberance on the caudomedial aspect of the distal radius. Osteochondromas are not necessarily a cause of lameness and may thus be an incidental radiological finding. When they do cause lameness, it is often because they impinge on the flexor tendons within the sheath, initially often only at maximal exercise, and hence signs can be intermittent, and therefore diagnosis difficult.

Radiology

Radiology shows an irregular exostosis on the caudomedial aspect of the distal radius proximal to the physis; this should not be confused with the normal distal radial physis, which can, in some horses, appear pronounced.

Ultrasonography

Inspection of the flexor tendons and the surface of the caudal radius can provide further diagnostically useful information.

Treatment

Treatment comprises excision of the lesion: a tenoscopic approach minimizes postoperative morbidity. The prognosis is usually very good for simple cases with minimal soft-tissue damage.

Soft-tissue injuries of the carpus

Collateral ligament desmitis is invariably the result of significant trauma and may be seen in association with bone damage. Ultrasonography is the technique of choice for imaging the collateral ligaments. Prognosis depends on the extent of damage and instability, plus other lesions that have been caused by the trauma.

Tendonitis can affect the tendon of the extensor carpi radialis (ECR), the common digital extensor, or lateral digital extensor muscle. Injuries can be traumatic (common) or strains (rare). There may be associated synovial sepsis if the injury involves the enclosing sheaths. If present, infection requires aggressive therapy, although the

prognosis is considerably better than for infection of joints or flexor tendon sheaths. Radical 'stripping' of the sheath of ECR has been described to treat chronic infection, often secondary to foreign-body penetration, with success. Extensor tendons are considerably more forgiving than flexor tendons when injured.

The accessory ligament of the SDFT (superior check ligament) arises from the caudodistal radius and joins the SDFT within the carpal sheath. Injury is rare but an important differential diagnosis when considering lameness related to the carpal sheath. Diagnosis is by careful ultrasonographic assessment. Rest is the recommended treatment, although surgical transection can be performed tenoscopically in refractory cases.

17.2 ELBOW

Anatomy

The elbow is a ginglymus or hinge joint formed between the distal humerus and the proximal end of the radius and ulna and is restrained by collateral ligaments medially and laterally.

In the foal, the distal humerus comprises separate ossification centres for the distal epiphysis and the medial epicondyle. The distal humeral epiphysis fuses to the metaphysis at 11–24 months. Both the radius and ulna each have a single proximal epiphysis. The proximal radial epiphysis fuses to the metaphysis at 11–24 months. The proximal epiphysis of the ulna initially appears quite small radiographically and well separated from the rest of the ulna. It fuses to the rest of the ulna at 24–36 months.

Ulnar fractures

These usually involve the olecranon process (see Figure 25.8) and commonly the articular surface of the semilunar notch. They compromise the ability of the horse to maintain the elbow in extension during weight-bearing because the fracture interferes with the triceps mechanism of which the olecranon is the insertion. They are subdivided into types according to configuration:

Type 1 – physeal fractures occurring in the young horse.
  Type 1a – non-articular fracture involving the growth plate only. Note that if displaced, the epiphysis can move a considerable distance proximally and may even be outside the field of a standard mediolateral radiographic projection of the elbow.
  Type 1b – Salter-Harris type II fracture involving the anconeus and proximal semilunar notch.
Type 2 – Articular fractures involving the semilunar notch.
Type 3 – Non-articular fractures involving the metaphyseal region of the olecranon.
Type 4 – Comminuted fractures which generally involve the semilunar notch and body of the olecranon.
Type 5 – Fractures of the distal olecranon/ulnar shaft that extend proximally to involve the distal semilunar notch.

Aetiology and pathogenesis

Ulnar fractures are usually due to a single episode of traumatic overloading, e.g. a kick from another horse or a fall.

Clinical signs

- Severe lameness on the affected limb, although with undisplaced fractures this may improve quite rapidly.
- Localized soft-tissue swelling around the elbow.
- Horse may stand with the elbow 'dropped' mimicking radial paralysis.
- Pain on manipulation of the elbow joint.
- Crepitus may be present with severe fractures but is often minimal.

Treatment

The majority of ulnar fractures are probably best treated by open reduction and internal fixation with a narrow 4.5-mm ASIF dynamic compression plate(s) and screws. Type 1a fractures in foals may be best fixed with a pin or screw and a wire tension band, as the proximal fragment is usually very small. Some non- or minimally displaced fractures may be successfully managed conservatively. If plates are used on skeletally immature horses, the implants should, if possible, be placed so as to avoid impeding growth at the proximal radial physis.

Distal humerus stress fractures

These are unique to racehorses and, like other stress fractures, result from cyclical loading outstripping the modelling process of the bone. This results in microfractures which propagate to form cortical fissures and will, eventually, lead to complete failure of the bone if not detected. In the elbow region, the predilection site is the craniodistal humerus. Diagnosis is by radiography, scintigraphy or ultrasonography, depending on the stage of disease or equipment available. If detected early, these fractures have an excellent prognosis with a short period of rest and controlled exercise.

Subchondral cystic lesions (SCLs)

(see also Chapter 15)

In the elbow, cysts are occasionally seen in both young and adult horses in the proximal radius, usually on the medial aspect of the elbow joint (Figure 17.3). They are best demonstrated on a craniocaudal radiograph. They are sometimes associated with periosteal reaction at the site of attachment of the collateral ligament. Clinical and radiographic resolution may occur with conservative treatment, particularly in young horses where they can sometimes be observed to

Figure 17.3 Detail of a craniocaudal radiograph of the elbow. In the medial condyle of the radius there is a poorly defined region of reduced bone density that seems to communicate with the joint. This is a subchondral cyst-like lesion, seen here in the site most commonly affected in this joint.

migrate or 'grow away' from the joint surface. Surgical curettage/intralesional medication via a medial extra-articular approach has been described.

Septic arthritis (see also Chapter 15)

The elbow has been described as a predilection site for septic arthritis secondary to traumatic injuries, especially wounds caused by kicks from other horses, which may be due to the relative lack of soft-tissue covering on the lateral aspect of the joint.

Capped elbow

This is an acquired subcutaneous bursa on the point of the elbow.

Aetiology and pathogenesis

It is usually due to repetitive trauma, e.g. the branch of the shoe on the affected limb hitting the elbow as the horse lies down ('shoe boil'). The small true bursa that underlies the insertion of the triceps is rarely involved.

Clinical signs

In the acute stage the condition appears as a prominent fluid swelling over the point of the elbow (Figure 17.4) which may later subside to a fibrous thickening. It is not usually a cause of lameness unless the skin is broken and it becomes infected, in which case the condition can be severe enough to mimic olecranon fracture.

Figure 17.4 Acute capped elbow.

Treatment

Prevent further trauma: check shoeing; over-reach or square-section ('shoe boil boot') boots placed around the pastern can be very useful. Surgical intervention is usually unnecessary and best avoided.

17.3 SHOULDER

Anatomy

The shoulder joint is an enarthrosis (ball and socket joint) though its chief movement is as a ginglymus (flexion and extension). It is also a synsarcosis: its stability relies on the integrity of the surrounding musculature rather than intrinsic supporting ligaments.

The scapula has four centres of ossification:

1. Body.
2. Scapular cartilage.
3. Tuber scapulae (supraglenoid tubercle) – fuses to body by 12–24 months.
4. Cranial part of glenoid cavity – fuses to body by 5 months.

The proximal humerus has three centres of ossification:

1. The diaphysis.
2. The humeral head.
3. The greater (lateral) tubercle.

These centres fuse to each other from about 3–5 months. The proximal humeral physis closes at 24–36 months.

Important diseases affecting the shoulder include osteoarthritis (Figure 17.5), osteochondritis dissecans, subchondral cystic lesions, scapulohumeral dysplasia, fractures, luxation, bicipital bursitis, ossification of the biceps tendon, and suprascapular neuropathy.

Osteochondritis dissecans (OCD)

(see also Chapter 15)

Lesions may be unilateral or bilateral and may involve only the glenoid or the humerus or both, although the most commonly affected site is the humeral head. The affected articular cartilage is variable in thickness, soft, and less firmly attached to the underlying subchondral bone. There are often areas of full-thickness defects in the articular cartilage. The lesions are often extensive and irregular.

Clinical signs

OCD is seen most commonly in young horses. There is mild to moderate intermittent lameness, characterized – as for most lameness arising in the shoulder region – by a shortened cranial phase to the stride. There is also usually mild atrophy of the shoulder muscles. Occasionally, older horses can present with lameness referable to this condition, often when they are required to work for the first time.

Radiology

- Flattening of the contour of the humeral head.
- Irregular areas of subchondral radiolucency in humeral head or scapular glenoid.
- Remodelling of the joint margins, particularly the caudal margin of the glenoid.

Treatment

Conservative management is directed at rest, and reduction in growth rate may be successful in mildly affected horses. Some clinicians advocate the use of systemic disease-modifying drugs (e.g. PSGAGs), although the evidence of their effectiveness is only anecdotal.

Figure 17.5 Shoulder OA. Mediolateral projection of the shoulder. This is an advanced case of osteoarthritis and represents the extreme of what might be seen – most cases are more subtle than this. There is caudal extension of the glenoid with osteophytes on the opposite humeral head. The humeral head is flattened and far from the normal round shape of this structure, which creates the incongruity between humerus and glenoid – note that the joint space seems to widen and narrow (although this view is taken in a non-weightbearing position, the joint space should still be even). There is a suggestion of focal loss of density in the middle of the humeral head in the region of most extreme flattening, most likely associated with subchondral bone erosion.

Surgical debridement under arthroscopic visualization may be undertaken in more severely affected cases. Unfortunately, because lesions tend to be extensive and difficult to debride fully, the prognosis is always guarded.

Subchondral cystic lesions (see also Chapter 15)

These usually affect the scapular glenoid rather than the humeral head. A small lucent area in the middle of the scapular glenoid is a normal variation and should not be mistaken for a lesion. 'Cysts' vary in size and location with respect to the articular surface. They may be small and difficult to visualize radiographically, so should not be ruled out if lameness has been localized to the shoulder but radiographs are unremarkable. They can be treated by arthroscopic debridement.

Cystic lesions can also be found in the intermediate tubercle of the cranial humerus associated with lameness. Their cause is unclear, but they can be identified scintigraphically and radiographically, and occasionally ultrasonographically if an opening can be identified into the bicipital bursa. These can be debrided bursoscopically, but limited cases series have also reported resolution of lameness with conservative management with intrabursal instillation of a corticosteroid.

Clinical signs

- Usually seen in young horses: yearlings and 2-year-olds.
- Older horses can present with these lesions; it is not clear why these late-onset cases develop.
- Lameness, which can be improved by intra-articular analgesia of the shoulder.

Radiology (see Figure 25.12)

- Seen at the junction of the middle and caudal thirds of the scapular glenoid.
- Single or multiple, with sclerotic margin.
- Vary in size and proximity to the joint; may have a visible communication.
- May move proximally (i.e. away from the articular surface) with time in association with improvement in lameness.

Scapulohumeral dysplasia

Seen only in miniature breeds, this condition occurs because of a mismatch between the size of the glenoid and humeral head: this causes instability and secondary OA. Although undoubtedly a developmental disease, cases may not present until the animal is mature and sometimes well into adulthood.

Clinical signs

Lameness is usually easily localized to the shoulder; there is often pain on manipulation and proximal limb muscle atrophy.

Radiology (see Figure 25.58)

Mediolateral views demonstrate variable degrees of subluxation and OA. Oblique views may demonstrate significant erosion of the humeral head

Treatment

There is little that can be done for advanced cases, which most are by the time they are recognized. Scapulohumeral arthrodesis has been described but is rarely performed.

Fractures

Fractures involving the shoulder joint are usually fractures of the scapula rather than of the proximal humerus. There is usually a history of specific external trauma such as a fall or collision. Various configurations are seen, but the commonest is fracture of the *tuber scapulae* (supraglenoid tuberosity). Such fractures may involve separation of a single discrete fragment or the fracture may be comminuted. Large fragments can involve the articular surface of the glenoid. The *tuber scapulae* forms the origin of the biceps brachii muscle, and as a result, when fractured, it becomes displaced craniodistally.

The scapula may also be fractured further proximally in association with trauma or, in racehorses, as stress fractures. These injuries may be complete or incomplete. Displacement is usually minimal as the surrounding muscles splint the injury effectively. Occasionally comminuted fractures are seen and may result in proximal limb instability.

Stress fractures of the caudoproximal or craniodistal humerus are seen in racehorses and are probably best diagnosed by scintigraphy in the early stages. Radiographs demonstrate periosteal and endosteal

new bone formation once the fracture has been present for a short while.

Treatment

Horses with comminuted articular fractures are generally subjected to euthanasia on humane grounds. Horses with non-articular fractures of the body of the scapula may recover with conservative treatment comprising box rest for several months. Simple fractures of the tuber scapulae have been treated surgically, with varying degrees of success, by reduction and internal fixation in young horses and by fragment excision in older animals.

Traumatic luxation

Luxation ('dislocation') of the shoulder joint as a primary disease is rare in horses. The humeral head is reported to displace either cranially or medially. Immediate reduction under general anaesthesia is suggested to be the treatment of choice, but if trauma has been sufficient to result in this injury there will be significant soft-tissue or bony injury around the joint which is likely to make the prognosis unfavourable.

Bicipital bursitis

The bicipital bursa surrounds the origin of the biceps brachii muscle and lies between it and the bicipital groove of the humerus. Inflammation of the bicipital bursa is an uncommon cause of lameness. The inflammation may be due to trauma, infection (bacterial and fungal), or an underlying disease process in the nearby humerus or the biceps tendon itself; it is important to recognize the inciting cause before embarking on therapy. Occasionally, bicipital bursitis occurs without any evidence of an inciting cause (idiopathic); these cases often respond favourably to intrasynovial medication with a corticosteroid.

Diagnosis can be difficult. Localization is aided by injection of local anaesthetic solution into the bursa. Although bicipital bursitis is not a common cause of lameness, the possibility of pain originating from this structure should be borne in mind for forelimb lameness the cause of which remains elusive after other investigations. Radiography and ultrasonography allow inspection of the bone and soft tissues. Scintigraphy may identify bony lesions – such as humeral osteitis or cystic lesions – not observable on radiographs.

Treatment is aimed at the primary cause but may also consist of empirical therapy such as intrasynovial medication. Treatment of sepsis is as for other synovial structures.

Rupture of the biceps tendon may occur, usually following a fall. In these cases, the biceps mechanism is disrupted and the proximal limb appears 'disjointed' when manipulated. Diagnosis is made with ultrasonography. Bone damage may occur concurrently and may require surgery. Prognosis is guarded, but cases may recover function if given enough time.

Ossification of the biceps tendon

This has been reported as a rare cause of mild to moderate lameness. The ossification may be a result of trauma, or it may be associated with osteoarthritis of the shoulder. Treatment is usually empirical, and the prognosis is generally poor as dystrophic mineralization usually represents the end stage of a chronic disease process.

Suprascapular neuropathy ('sweeney')

This usually occurs because of damage to the suprascapular nerve, which innervates the supraspinatus and infraspinatus muscles. The nerve courses across the cranial aspect of the scapular neck where it is vulnerable to pressure from collars (draught horses) or direct trauma. Suprascapular neuropathy results in atrophy of the supraspinatus and infraspinatus muscles only. It is important to realize that 'sweeney' is a clinical sign typical of a syndrome, not a diagnosis in itself. Other causes of sweeney include brachial plexus injury (which usually increases the number of muscles showing atrophy, such as the triceps) and caudal cervical arthropathy (radiculopathy), although this causes atrophy of other muscle groups as well.

Clinical signs

The following signs may appear independently or together:

- Marked atrophy of the supraspinatus and infraspinatus muscles: the scapular spine becomes prominent on the affected side.
- Lateral subluxation of the joint during weight-bearing; the proximal humerus appears to bulge laterally as the affected limb takes weight (the shoulder joint is a synsarcosis requiring the support of the extrinsic musculature for congruency).
- Difficulty in advancing the limb.

Treatment

Conservative treatment often produces good results. Surgical decompression of the suprascapular nerve where it crosses the cranial border of the scapula has been suggested to be useful, particularly if progress with conservative treatment is poor. However, there is still debate on the optimum time for performing such surgery, and it is contraindicated if the damage is at a different site (e.g. brachial plexus). There is little that can be done to aid nerve regeneration, but careful electrical stimulation can maintain muscle mass during repair. Exercise can be helpful but must be limited to avoid excessive damage to the unstable shoulder joint. Careful assessment of bony and soft-tissue structures should be carried out to demonstrate any injury (e.g. scapula fracture) that may reduce the prognosis or render physiotherapy unsuitable.

17.4 HOCK

Anatomy

Bones

The hock is composed of six tarsal bones (see Figures 25.59–25.62):

- Tibial (talus).
- Fibular (calcaneus).
- Central.
- 1st and 2nd (fused).
- 3rd.
- 4th.

1. The 1st and 2nd tarsal bones of young foals may not be fused.
2. The epiphysis of the fibular tarsal bone is usually present at birth but may develop in the first few weeks of life in some foals.
3. The distal epiphysis of the tibia fuses to the metaphysis at 17–24 months.
4. The lateral malleolus of the tibia has a separate ossification centre which phylogenetically represents the distal epiphysis of the fibula; fuses to rest of epiphysis by 3 months.
5. Trochlear ridges of the tibial tarsal bone:
   - Lateral ridge has a longer notch at its distal end.
   - Medial ridge has a variably sized and shaped prominence at its distal end which may appear radiographically as one or occasionally several distinct fragments; these should not be mistaken for fracture fragments.

6. The central and 3rd tarsal bones have dorsomedial ridge-like prominences.

Joints

There are four principal levels of articulation in the hock:

1. Tarsocrural: main hock joint where most movement occurs.
2. Proximal intertarsal (talocalcaneal–centroquartal): communicates with tarsocrural joint.
3. Distal intertarsal (centrodistal): communicates with tarsometatarsal in a minority of horses.
4. Tarsometatarsal.

Osteoarthritis of the small hock joints ('bone spavin')

General

'Bone spavin' is a colloquial term used to refer to osteoarthritis most commonly of the distal intertarsal and tarsometatarsal joints. It is a common cause of persistent hind limb lameness in the horse. Osteoarthritis of the proximal intertarsal joint is less common but carries a more guarded prognosis for return to soundness as it communicates with the high-motion tarsocrural joint.

Clinical signs

There is unilateral or bilateral hind limb lameness, usually with an insidious onset. Some affected horses may be presented because of non-specific hind limb gait abnormalities, poor performance, or suspected back problems, particularly if the condition is bilateral. There are no pathognomonic gait changes to observe, but in many cases the cranial phase of the stride is shortened, causing a 'toe stabbing' gait. The limb may be carried medially during protraction and land more on the lateral aspect of the foot, leading to increased wear of the toe and lateral branch of the shoe (this has led some clinicians to use lateral extensions on the hind shoes to manage the condition). Bilateral involvement may lead to a short, choppy hind limb gait. There is usually a positive response to proximal hind limb flexion – the so-called 'spavin test' – although horses with other hind limb problems may also show increased lameness in response to this manoeuvre, and thus it is by no means specific to spavin. Bony enlargement on the medial aspect of the hock occurs in advanced disease but may not be apparent in many horses. The horse usually shows a positive response to intra-articular analgesia of the distal hock joints

Radiology (see Figure 25.7)

Typical radiological changes are:

- Marginal irregularity: a roughened hazy appearance to the joint margin.
- Marginal radiolucency: demineralization of the subchondral bone at the joint margin.
- Joint space may appear irregular in width and poorly defined.
- Subchondral bone erosion: seen as cyst-like radiolucent areas.
- Periarticular new bone formation on the central and third tarsal bone and third metatarsal bone.
- Progressive joint fusion may occur in association with destructive and proliferative changes.

Because of the clinical appearance of swelling on the dorsomedial aspect of the hock, the dorsolateral–plantaromedial projection is often said to be the most important view and may be taken in isolation during 'screening' radiographs. This trap should be avoided as assessment of all four projections may uncover disease only evident on one.

Some horses have pain associated with the small hock joints without accompanying radiological change (so-called 'occult spavin'). However, in recent years it has become known that many horses diagnosed with distal hock joint disease are, in fact, suffering from lameness arising from the attachment of the suspensory ligament (proximal suspensory desmitis). The suspensory ligament arises from the plantaroproximal aspect of the third metatarsal bone and is situated close to the tarsometatarsal joint (TMTJ). It is now recognized that diagnostic anaesthesia aimed at the TMTJ may influence this region and even that medication of the joint can affect the suspensory ligament. This should be borne in mind if lameness blocks to the hock without radiographic abnormalities, if repeated treatment of the hock has a reduced effect, or if certain gait characteristics (e.g. lameness is worsened by lunging on a soft surface, particularly with the limb on the outside of the circle) are evident. In all cases, it is good practice to consider proximal suspensory desmitis as the primary differential diagnosis in all distal hock lameness.

Treatment

1. Continued controlled exercise (± NSAIDs) in the hope of achieving spontaneous ankylosis of the affected joints. Some authorities have questioned whether in fact spontaneous fusion ever actually occurs; nevertheless, while spontaneous complete ankylosis is probably rare, controlled exercise can result in significant clinical improvement.
2. Intra-articular medication.
   - Corticosteroids.
   - (Hyaluronic acid).
   - (Polysulphated glycosaminoglycans).

This is simple and effective, but many horses require repeat medication which can become expensive and time-consuming, and can increase the risk of infection.

3. Shoeing: hind foot conformation and shoeing are often overlooked, while much attention is focused on the front feet. Some authorities recommend rolling the toes (sensible); elevating the heels (of doubtful value and may cause secondary ill effects), and creating a lateral extension (also of dubious value but unlikely to be of harm). Simple critical appraisal of dorsopalmar and mediolateral balance may be more worthwhile.
4. Cunean tenectomy: removal of a 2-cm section of cunean tendon on the medial aspect of hock. This is postulated to relieve pressure on the site of new bone formation, but is of debatable efficacy. The Wamberg modification, of equally debatable efficacy, cuts through all the soft tissues on the craniomedial aspect of the hock, thereby possibly destroying the nerve supply to the area. Cunean tenectomy is not widely performed in the UK.
5. Surgical arthrodesis is performed by destroying the articular cartilage by drilling tracks across the joints. At least 60% of the cartilage should be removed. The number of tracks advocated ranges from 3 to 6, although excessive drilling can also result in greater lameness due to bone collapse postoperatively. Joints to be drilled are first located with needle markers and radiographs. Alternatively, further destruction of cartilage has been proposed using a laser, although this has not been shown to be any more effective than drilling alone. Postoperatively horses are box-rested for 10 to 30 days until comfortable, and then started on a programme of controlled exercise which is thought to be important in encouraging ankylosis.
6. Chemical arthrodesis by intra-articular injection of sodium monoiodoacetate or ethyl alcohol has been described and may allow earlier return to work. Initial results with sodium monoiodoacetate were encouraging, but it has become less popular, partly because of the pain levels often seen post

injection. Trials using ethyl alcohol have shown that horses are comfortable after injection, perhaps because ethyl alcohol has neurolytic properties.

Osteochondritis dissecans

Sites

- The intermediate ridge of the distal tibia.
- The lateral trochlear ridge of the tibial tarsal bone.

Less commonly:

- The medial trochlear ridge of the tibial tarsal bone.
- The dorsal aspect of the medial malleolus of the distal tibia.

Clinical signs

- Usually in young horses: Standardbreds and European Warmbloods are said to be affected more commonly than Thoroughbreds. The disease is also common in Shire horses.
- Affected horses usually present with distension of the tarsocrural joint ('bog spavin').
- Lameness is variable and may be absent. Tarsocrural distension may be an incidental finding without lameness or radiological abnormalities.

Radiology

- Subchondral bone deficits and separate mineralized fragments arising from the sites outlined above.
- Lesions may be present without radiological changes; lameness referable to a distended tarsocrural joint may therefore warrant further investigation using ultrasonography or arthroscopy, even if radiographs are normal.

Treatment

- Surgical removal of the osteochondral fragment and curettage of the defect.
- Usually performed under arthroscopic visualization.
- Approximately 75% of affected horses can return to their intended use following arthroscopic surgery.
- There is an increasing tendency to 'treat' these lesions if they are discovered on survey – often pre-purchase – radiographs. The wisdom of such intervention should be considered on a case-by-case basis as the potential effect on a horse's athletic career probably varies significantly with the intended use of the horse and the type/location/size of the fragment, etc.

'Bog spavin'

This is a colloquialism used to describe a clinical sign and is NOT a diagnosis.

1. Fluid distension of the tarsocrural joint.
2. Up to three swellings may be detectable although they all reflect distension of the same structure.
   - Dorsomedially – between the peroneus tertius and the medial malleolus, usually the largest and most obvious.
   - Caudomedially: between the medial malleolus and the deep digital flexor.
   - Caudolaterally: between the lateral malleolus and the tuber calcis.
   - Lameness may or may not be present.

Aetiology

- Idiopathic.
- OCD.
- Trauma ± intra-articular fractures, e.g. lateral malleolus or trochlear ridge of tibial tarsal bone.
- 'Sprain' type injury with or without observable evidence of ligamentous damage.
- Infection – usually very painful and lame.

Treatment

- Treat underlying cause if apparent.
- Empirical local therapy, e.g. drainage and injection of a corticosteroid/sodium hyaluronate followed by bandaging (of questionable efficacy and necessity if not lame).

Thoroughpin

Colloquial term to describe distension of the tarsal sheath (Figure 17.6). Various qualifications exist (e.g. false or true thoroughpin), all of which are best avoided as they can be more accurately defined. The tendon of the deep digital flexor muscle traverses the tarsal groove formed at the back of the tarsus by the plantar surface of the sustentaculum tali and the medial surface of the tuber calcis. The 'tarsal canal' is lined by a synovial membrane, the tarsal sheath. This begins 5–7 cm proximal to the level of the medial malleolus and extends distally to the proximal third of the metatarsus.

Clinical signs

- Distinct swellings on either side of and cranial to the calcanean (Achilles') tendon (Figure 17.6).
- The swellings vary in size: the medial one is generally the larger, but they are part of the same structure.

Figure 17.6 Thoroughpin. Swelling of the tarsal sheath.

- There is sometimes swelling in the proximal metatarsus.
- Usually unilateral.
- Swellings are cold and painless, particularly in idiopathic tenosynovitis without lameness.
- Lameness may be associated with the distension, in which case further evaluation is necessary to determine the inciting cause.

Aetiology

In many cases the horse exhibits no lameness and has no underlying abnormality, and so, the condition does not require further investigation, although it may be considered a cosmetic blemish by some. In other cases, any injury to the structures that form the tarsal canal can cause distension and lameness: e.g. DDF tendonitis, damage to the sustentaculum tali, penetrating injury resulting in sepsis.

Radiology

Deposition of new bone or lysis can sometimes be seen along the medial border of the sustentaculum tali. A flexed plantaroproximal–plantarodistal projection is very useful to highlight this region, in addition to the usual projections (particularly the dorsomedial–plantarolateral oblique).

Treatment

- Synoviocentesis and medication may improve idiopathic cases, although the effect is usually only transient.
- Sepsis should be treated aggressively as for other synovial infections.
- Lameness referable to this structure can be evaluated tenoscopically so that damage to associated structures, such as the sustentaculum tali and DDFT, can be debrided.

Curb

A curb is the description given to the external appearance of the plantar hock, usually when a convex outline appears on the plantarolateral aspect, level with just above the head of metatarsal bone 4. Such a swelling was, in the past, thought to be associated with desmitis of the plantar ligament, but this is VERY rare. More commonly these swellings can be subcutaneous fibrosis, paratendonitis or tendonitis of the superficial digital flexor tendon, desmitis of the accessory ligament of the DDFT, proximal suspensory desmitis, or a prominent head of Mt4. The confusion is probably due to advances in imaging that allow us to rule out these injuries in many cases, and perhaps due to the changing use of horses over the years; historical inaccuracies have persisted in the literature. Therefore, as it means different things to different people, it is a term best avoided, except as a loose description for the external appearance of the hock. It is NOT a diagnosis.

Clinical signs

- Some horses have a prominent head of Mt4 and are described as 'curby' although this is a normal anatomical variation.
- Others have firm, non-painful soft-tissue swelling without lameness that may represent previous injury but which is rarely significant.
- If associated with lameness, there may be soft, sometimes slightly fluctuant swelling. These are the cases often erroneously diagnosed as having injury to the plantar ligament or SDFT. In most cases, radiographs and ultrasound scans demonstrate nothing more than subcutaneous oedema. The true aetiology remains obscure but is presumably a minor soft-tissue 'strain' without significant damage to any of the important structures visualized.
- Very rarely, damage to Mt4, the plantar ligament or SDFT will be seen.
- Horses with tarsal bone collapse, secondary to congenital incomplete ossification, often display abnormal hock conformation ('sickle hocks') and are described as being 'curby'. In this instance, the primary abnormality is not the curb.

Treatment

- As described above, most cases require no treatment.
- If either tendinitis or desmitis is present, a prolonged period of rest may be necessary along with treatment aimed at the particular lesion.
- In acute cases where there is lameness without evidence of damage to important structures, a short (2-week) period of rest with or without the use of topical/systemic anti-inflammatories is all that is required.

Prognosis

This depends on the underlying abnormality, if any. In general, it is very rare for a 'curb' to impact significantly on a horse's athletic career, although the swelling will persist to a greater or lesser degree.

Displacement of the superficial digital flexor tendon from the point of the hock/SDFT luxation

The superficial digital flexor tendon (SDFT) passes over the point of the hock and is secured by strong retinacula. Occasionally, the retinacula may be damaged, and the SDFT becomes unstable. It may displace permanently to one side (usually lateral) or it may move on and off the point of the hock. Less commonly, the SDFT sustains a longitudinal tear which results in it dividing over the calcaneus

Affected horses can be extremely distressed in the acute phase and require sedation as well as analgesia. The problem can usually be seen readily, but if intermittent it can be surprisingly difficult to detect. There are also some horses that suffer only partial subluxation under particular circumstances and these can be a diagnostic challenge, although there are usually other signs – such as calcaneal bursitis – that alert the clinician to this possibility. In acute, complete displacement, peritarsal swelling develops rapidly, making definitive diagnosis more difficult on clinical grounds alone.

Treatment

- The tendon usually eventually stabilizes in the displaced position, at which time the horse usually becomes pain-free, though there may still be slight mechanical interference with the gait.
- Intermittent luxations can present a challenge to treat as the horse may resent the movement of the tendon on and off the hock.
- Surgical replacement and repair of the supporting retinacular tissues are described and advocated by some. However, it is major surgery and is rarely successful. Owners should be aware of all possible complications before it is undertaken.
- It may be preferable with unstable displacements to cut the damaged attachments on the point of the hock, thereby creating a stable, permanent luxation. A bursoscopic approach has recently been described.

Gastrocnemius tendonitis

Anatomy

The gastrocnemius muscle has two heads (medial and lateral) that terminate in the mid-tibial region in a common tendon. The

superficial digital flexor tendon (SDFT) crosses around the medial side of the gastrocnemius tendon to become superficial just proximal to the hock. The SDFT and gastrocnemius tendons are separated by a bursa (calcaneal bursa) that extends to the mid-tarsal level. A small bursa also lies cranial to the insertion of the gastrocnemius on the tuber calcis (gastrocnemius bursa); there is usually a communication between these bursae.

Clinical signs

- Gait:
  - mild to moderate lameness with variable response to upper-limb flexion.
  - reduced hock flexion.
- Physical examination:
  - subtle to moderate enlargement of the gastrocnemius tendon.
  - local heat ± pain on palpation.
  - enlargement of the calcaneal bursa.
- Regional analgesia:
  - positive response to tibial and fibular block or tibial block alone.

Ultrasound

The gastrocnemius tendon normally has a heterogeneous echogenicity proximally, but becomes more uniform distally as it comes to lie cranial to the SDF tendon. Horses with gastrocnemius tendonitis have enlargement of the tendon, poor marginal definition, and focal or diffuse areas of reduced echogenicity.

Treatment

Treatment comprises box rest and controlled exercise.

Prognosis

The prognosis is generally poor; most affected horses remain lame.

Rupture of the peroneus tertius

The peroneus tertius originates from the lateral aspect of the distal femur (extensor fossa) and inserts on the dorsolateral aspect of the third metatarsal bone and fibular and fourth tarsal bones. It lies on the craniolateral aspect of the crus buried between the long digital extensor and the *tibialis cranialis*. It mechanically flexes the hock when the stifle is flexed (part of the reciprocal apparatus).

Aetiology

- Rupture is usually a result of overextension of the hock.
- May occur if the limb becomes trapped and the horse struggles violently to free the limb.
- As a complication following the application of a full-hindlimb cast.
- Occasionally spontaneous at exercise.

Clinical signs

- Inability to flex the hock.
- When the limb is advanced the hock remains extended; that part of the limb below the hock hangs limp as if fractured.
- The calcanean tendon loses its usual tension.
- The hind limb is readily extended caudally so that tibia and metatarsus both lie horizontal and the calcanean tendon develops a flaccid curve.
- The rupture cannot be detected on palpation as it usually occurs deep within the soft tissues of the crus.
- Disruption of the peroneus tertius may be demonstrated ultrasonographically.
- Occasionally, the peroneus tertius may be avulsed of its origin from the distal femur; these cases may have signs relating to the stifle as well as the evidence of reciprocal apparatus disruption.

Treatment

- Box rest for 6 weeks.
- Good prognosis for return to soundness.
- Repair can be monitored ultrasonographically.

Collateral ligament injury

The collateral ligaments of the hock consist of long and short groups medially and laterally that run at almost ninety degrees to each other, providing significant stability in a wide range of positions. Collateral desmitis may be seen associated with distension of the tarsocrural joint and variable lameness. It is diagnosed ultrasonographically, although cases frequently present with entheseous new bone on radiographs which may prompt ultrasound examination. It is usually thought of as a traumatic condition but is reported in Standardbreds as an athletic strain-type injury. Rest plus appropriate local therapy, depending on the stage and severity of the disease, are indicated (cold therapy, external coaptation, intra-articular medication etc.). Occasionally torn fibres of the ligament can prolapse into the tarsocrural joint, prompting arthroscopic evaluation and debridement

Tarsal bone collapse

Collapse of the dorsal aspect of the central or third tarsal bones results from incomplete ossification of the tarsal bones at birth and may be associated with other signs of dysmaturity/prematurity (it is an important but often overlooked aspect of care when dealing with such patients). In many cases, the condition goes unrecognized until the horse begins training (when lameness may occur) or is radiographed prior to sale. In rare cases, deformities may arise secondary to osteomyelitis of the cuboidal bones of the tarsus in foals.

Aetiology

- Incomplete ossification of the tarsal bones at birth.
- Tarsal osteomyelitis.

Clinical signs

Mature horses:

- In many cases the only outward sign is deviation of the normal conformation of the hock ('sickle hocks'). Some horses have palpable abnormalities to the outline of the distal tarsal joints detectable dorsomedially.
- Lameness may or may not be a feature, depending on severity of disease, age of the horse, level of exercise etc.

Foals:

- Signs of dysmaturity/prematurity.
- Abnormal hock conformation or progressive deterioration in hock conformation.
- Pyrexia, joint swelling, abnormal haematological and biochemical values, and other signs if associated with sepsis.

Radiology

Mature horses:

- Deformity of the dorsal aspect of the central and/or third tarsal bones (wedging) with possible fragmentation. There is a wide range of abnormality, varying from mild trabecular changes and slight deformity of the bones through to severe wedging/crushing resulting in apparent separation of the dorsal and plantar components of the bones.

- Some cases (usually racehorses) present with third tarsal slab fracture (or, less commonly, central tarsal slab fracture) when it can be appreciated that there was a pre-existing, clinically silent, tarsal bone deformity.
- Foals:
- Incomplete ossification is managed by external coaptation until the bones have mineralized. Severe cases can be very difficult to manage.

Prognosis

- In mature horses the prognosis depends on the severity of changes and the degree of lameness. Some cases come to light during pre-purchase examinations when no problems had been suspected. It is therefore likely that many horses exist with the disease but suffer no ill-effects. Even those that suffer slab fractures as a consequence of deformity usually recover (although the opposite hock should be inspected as some injure the contralateral limb subsequently).
- Intra-articular medication, as for OA, can be used (with care, so as to avoid fracture).
- In foals diagnosed with tarsal bone collapse the consequences can be career-limiting.

Intra-articular fractures

The incidence of tarsal fractures depends on the use of the horse. All horses can sustain traumatic injuries (lateral malleolar and trochlear ridge fractures are among the most common), but it is rare for horses other than racehorses to suffer slab fractures of the third and central tarsal bones.

1. Lateral malleolar fractures: if the fracture is comminuted and/or the fragments are displaced they are probably best removed surgically. Horses with minimally displaced single-fragment fractures can be successfully managed conservatively. Ultrasonographic evaluation can be very useful to determine involvement of the collateral ligaments and monitor repair.
2. Fracture of a trochlear ridge can be treated with arthroscopic debridement; the prognosis depends on how much and in what position the ridge is damaged.
3. Slab fractures (see Figures 17.7 and 25.16) of the third and central tarsal bones can almost always be managed conservatively with success. Rarely, internal fixation can be considered for uncomplicated and recent slab fractures if significantly displaced.

Luxations

Luxation of the tarsocrural joint is usually an indication for euthanasia.

- Very difficult to reduce and subsequently stabilize.
- Usually causes severe damage to articular surfaces.
- Luxation of the more distal joints (see Figure 25.16) may be treatable with elective surgical arthrodesis and/or cast support but is still a very severe injury. Success is more likely in smaller patients (foals or ponies).

Capped hock

- Fluid accumulation or oedematous thickening under the skin, over the point of the hock.
- Usually due to direct trauma: one-off or repetitive.
- Not usually a cause of lameness, just a cosmetic blemish.
- Best left alone; recurrence of trauma should be prevented.
- Avoid surgical intervention: prone to wound breakdown.

Figure 17.7 T3 slab fracture. Dorsomedial-plantarolateral oblique radiograph of the hock. There is a near vertical line about a centimetre from the dorsal surface of the third tarsal bone (T3), running from the centrodistal to the tarsometatarsal joints. This is a slab fracture. Note also the entheseous new bone on the front of T3 and around the joint margins which are part of the same disease process.

- Drainage ± corticosteroid or irritant: of debatable efficacy, and carries a risk of infection.

Calcaneal bursitis

This is sometimes colloquially known as 'deep-capped hock', but the aetiology and prognosis are very different from the above.

- Fluid accumulates within the bursa between the SDFT and the gastrocnemius tendon.
- It is VERY important to identify this and differentiate it from other types of fluid swelling in the hock.
- It may be associated with infection: usually severe lameness; often present with a wound nearby. Together with the four standard views of the area, a plantaroproximal–plantarodistal oblique radiograph may reveal damage to the calcaneus if the horse will tolerate a fully flexed hock. Requires URGENT surgical treatment. Extension of the infection into the adjacent bone of the calcaneus (producing radiographically evident osteolysis) is associated with a poor prognosis.
- Trauma: may be self-limiting, but damage to the calcaneus and/or SDFT and/or gastrocnemius should be ruled out.
- SDF tendonitis or intermittent luxation of the SDFT from the point of the hock. Careful clinical assessment (both at rest and when walking) and imaging helps to rule these conditions out.
- Idiopathic: if all other possibilities have been ruled out then consideration can be given to intrasynovial medication with a corticosteroid.

Talocalcaneal osteoarthritis

OA of the talocalcaneal joint (between the tibial and fibular tarsal bones) is a rare but important differential diagnosis for lameness localized to the hock. It causes variable but frequently severe lameness, and radiographic signs can be difficult to identify, consisting usually of a combination of sclerotic and lytic cystic lesions; these are best visualized on a good lateromedial projection. Strangely, intra-articular analgesia of the tarsocrural joint (with which it communicates) is rarely completely positive, and the most effective (but least specific) diagnostic analgesic technique is tibial and peroneal analgesia. Scintigraphy is invariably positive. Response to conservative therapy is usually ineffective, and, while surgical therapy can restore function, the disease remains a therapeutic challenge.

17.5 STIFLE

Anatomy

Bones

1. Femur.
2. Tibia.
3. Fibula.
4. Patella.

1. The medial ridge of the femoral trochlear is much larger than the lateral. This difference in size is not apparent radiographically in the neonatal foal but develops from 2 months of age.
2. The proximal end of the medial trochlear ridge acts as a catch for the patellar fibrocartilage and the medial patellar ligament, enabling the joint to be locked in extension.
3. There is a small notch at the junction between the medial trochlear ridge and the medial condyle.
4. At this site on the lateral side there is a flattened and slightly roughened area at the transition.
5. Closure of the distal femoral and proximal tibial physes occurs at 24–30 months of age.
6. Separate ossification centre for tibial tuberosity (apophysis) fuses to epiphysis at 9–12 months and to the metaphysis at 30–36 months.
7. The fibula ossifies from two or more ossification centres which are visible radiographically by 2 months. One or more residual cartilage lines separating these centres may be seen throughout life and should not be confused with fractures.

Joints

1. Femoropatellar.
2. Femorotibial.
   - medial, compartment.
   - lateral compartment.
3. The femoropatellar joint often communicates with the medial compartment of the femorotibial joint.
4. The femoropatellar joint communicates with the lateral compartment of the femorotibial joint in 20–25% of horses.
5. The two compartments of the femorotibial joints do not communicate with each other in normal horses.

Ligaments

1. Medial and lateral collateral (femorotibial) ligaments.
2. Meniscal ligaments anchor menisci to tibia (and femur).
3. Cranial and caudal cruciate ligaments.
4. Femoropatellar ligaments (thin).
5. Patellar ligaments.
   - medial.
   - middle.
   - lateral.

A large hooked plate of fibrocartilage attaches to the medial aspect of the patella proximally and forms the origin of the medial patellar ligament. In the ordinary standing position, in which the angle of the stifle is about 135° and both hind limbs bear weight, neither stifle joint is locked. When the horse rests one limb the angle of the stifle joint of the supporting limb is increased to about 145° and the fibrocartilage is hooked over the top of the medial ridge. The ridge then protrudes through the loop formed by the medial and middle patellar ligaments and the patellar fibrocartilage. This mechanism is an important part of the stay apparatus which enables the limb to support the weight of the body with the minimum of muscular effort.

Osteochondritis dissecans (see also Chapter 15)

In this disorder there is osteochondral fragmentation, usually from the lateral trochlear ridge of the femur.

Clinical signs

- Young horses (usually <2 years old).
- Present with synovial effusion of the femoropatellar joint ± lameness.
- Commonly bilateral.
- Lesion severity can vary considerably, but affected young horses (<12 months old) often have severe clinical problems and extensive radiographic changes, while older horses may have less severe lesions.

Radiology (see Figure 25.11)

- Defect in the lateral trochlear ridge ± free mineralized fragment.
- Usually involves the middle to upper thirds of the lateral trochlear ridge.
- Always radiograph the contralateral limb.
- Radiologically silent defects can be detected ultrasonographically; this technique should be considered in young horses with effusion but normal radiographs.

Treatment

- Surgical removal of the cartilage flap and curettage of the underlying defect.
- Usually performed under arthroscopic visualization.

The prognosis is variable depending on the extent of the lesion; in one large case series 64% of the horses returned to their intended use after arthroscopic surgery.

If the lesion is detected in horses less than 1 year of age, it is now considered best practice to treat conservatively and monitor the lesions; significant improvement (though rarely resolution) can occur which may dramatically reduce the extent of surgical debridement necessary.

Subchondral bone cysts (see also Chapter 15)

Clinical signs

- Generally found in young horses but can present in middle-aged to old horses. It is unclear whether old horses have had the lesions since they were young without showing signs, or whether a different aetiology exists in these patients.

Figure 17.8 Stifle cyst. In this caudocranial radiograph of the stifle, a large elliptical region of reduced bone density is clearly visible in the medial femoral condyle. This subchondral cyst-like lesion (SCL) is surrounded by a rim of dense bone (most are) and appears to communicate with the joint space. Note the concavity in the condyle that creates the 'neck' of the cyst. Other views (particularly flexed lateromedial) and ultrasonography can help to characterize the lesion further.

- Usually presents clinically as a persistent, mild to moderate lameness, although it may be intermittent in nature (and sometimes severe depending on concurrent abnormalities).

Radiology

- Seen best on the caudocranial projection (see Figures 17.8 and 25.12).
- Large circular or dome-shaped cysts in the medial femoral condyle, usually with a distinct communication with the medial compartment of the femorotibial joint.

Treatment

- Conservative management (i.e. 6 months rest) may restore soundness in 20–50% of cases.
- Surgical treatment by debriding the cyst under arthroscopic control has also achieved good success with up to 80% of horses returning to normal work.
- Direct injection of a corticosteroid – either under arthroscopic guidance or via ultrasound guidance in the standing, sedated horse – can be very beneficial in some cases. The latter technique has the advantage of being minimally invasive but does not enable the identification of concurrent disease.
- There has been recent interest in the use of cartilage resurfacing techniques.

Upward fixation of the patella

With upward fixation of the patella, the medial patellar fibrocartilage and medial patellar ligament become locked up over the proximal end of the medial trochlear ridge of the femur and do not spontaneously unlock when the horse moves forwards. In some cases (delayed release) unlocking is delayed.

Aetiology

- Straight-legged conformation has been suggested to predispose.
- Poor general condition or lack of fitness. Horses may start to show clinical signs when stabled.

Clinical signs

- Permanent or temporary locking of the limb in extension. Limb is held caudally.
- Intermittent catching of the patellar during motion; stifle suddenly unlocks with a jerk and sometimes an audible 'clunk'.
- Intermittent cases may be more obvious when the horse is reversed or turned toward the affected side.
- It may be possible to temporarily fix the patella by manually forcing it proximally and laterally.
- The problem may be bilateral.

Treatment

1. Mildly affected horses that are in poor condition or unfit often cease to have problems when their general condition and fitness are improved. Young horses may similarly grow out of the problem.
2. Some cases of delayed patellar release appear to worry the rider/owner/trainer more than the horse. However, some subtle cases have been shown to be suffering from lameness emanating from other sites, presumably resulting in less than optimal muscle development and leading to the secondary effect seen. This should be borne in mind when dealing with these cases.
3. Elevating the heels of the hind feet with wedges or graduated shoes can aid in the period while the horse's exercise level is being increased or in refractory cases. Raising the heels should always be used with caution as it may become difficult to revert to normal shoes (the condition recurs) or they may have an adverse effect on the foot conformation (exacerbating collapsed heels).
4. A fixed patella may sometimes be temporarily released by:
   - Suddenly startling the horse.
   - Backing the horse while pulling forward on a rope attached around the fetlock to hyperextend the stifle.
   - Manually forcing the patella from its fixed position.
5. Permanent fixation necessitates surgery – medial patellar desmotomy – the treatment of choice for severe or persistent cases. Can be done under general anaesthesia or, more easily, under sedation and local analgesia where the ligament can be felt more easily and transected with a bistoury knife. It is said that horses should have 4–5 months rest after surgery before returning to full work to enable the patella to adopt a new tracking orientation through the trochlear groove and so minimize apical patella fragmentation. However, this has been disputed. Percutaneous ligament splitting has also been advocated as an alternative to desmotomy.
6. Some horses develop persistent lameness following medial patella desmotomy associated with new bone formation or fragmentation at the distal pole of the patella. This condition has been treated successfully by debriding the lesions under arthroscopic visualization. Fragmentation may be more common than lameness associated with it, but owners should

be aware of possible long-term implications before proceeding with surgery.
Some clinicians advocate the use of so-called internal blisters to 'tighten up' the ligaments. An internal blister may even be used as a general performance-enhancing technique by some, but their use is discouraged. Intra-articular medication of the stifle does not resolve the problem UNLESS there is another primary disease within the stifle causing discomfort.

Patella luxation

Aetiology

1. Congenital/developmental.
   - May occur in foals, especially Shetland ponies.
   - May be associated with a hypoplastic lateral femoral trochlear ridge (more common in dysplastic breeds).
   - Surgical intervention in the form of lateral retinacular release and medial imbrication. Trochleoplasty may help in some cases.
2. Acquired.
   - Intermittent lateral subluxation may occur especially in young ponies.
   - Can be the consequence of extensive lateral trochlear ridge OCD and/or after excessive debridement for this condition. Causes stifle clicking and abnormal hind limb gait. Leads to secondary osteoarthritis and joint effusion.
   - Traumatic lateral luxation may occur. Patella palpable laterally. Limb held semiflexed – no stability. Stifle and hock collapse when weight taken.

Patellar fractures

Patellar fractures are usually a consequence of direct external trauma. Affected horses usually have local swelling over the patella and femoropatellar joint effusion. Lateromedial and cranioproximal–craniodistal oblique radiographs are generally the most useful to demonstrate the injuries.

Non-articular fractures can be managed conservatively. Articular fractures that do not impair the quadriceps mechanism (e.g. parasagittal fractures) may be managed by removal of separated fragments. Transverse articular fractures that prevent stifle extension require internal fixation and carry a guarded prognosis.

'Gonitis'

Gonitis is a non-specific term indicating inflammation of the stifle joint(s); it has little meaning and should be avoided as more accurate terms exist to describe specific clinical conditions.

Ligament injuries

Injuries to the cruciate ligaments (particularly the cranial) are very common in other species, and this prevalence has been transposed to the horse with little evidence. In fact, cranial cruciate ligament injury is not as common as previously thought in the horse. Of significantly more importance are injuries to the cranial meniscal ligaments (especially medial) and the menisci themselves (again, particularly medial). Medial collateral ligament desmitis is, again, common in other species, including dogs and humans but not seen frequently in horses unless associated with significant trauma.

Cranial cruciate (CrCL)

Tests are described in the literature to evaluate the integrity of the CrCL, along the lines of the cranial drawer test used in dogs. In practice, these are difficult to perform, potentially dangerous to the operator, and of very limited usefulness. There are better ways to diagnose these injuries.

Localization

Cases invariably have a degree of synovial effusion, usually of the medial compartment of the femorotibial joint. Intra-articular anaesthesia improves but may not abolish lameness.

Radiography

New bone formation or avulsion fragment at the ligament insertion; signs of OA.

Ultrasonography

A technique is described to evaluate the CrCL ultrasonographically, but it is technically demanding, and interpretation requires experience

Definitive diagnosis and treatment

Arthroscopic evaluation of the stifle is required for diagnosis and the assessment of concurrent disease. Therapy includes debridement of the injured ligament and postoperative rest. No CrCL reconstruction technique has yet proved viable in the horse. Significant injures carry a poor prognosis for future soundness.

Collateral ligament injuries

1. The medial collateral ligament is the more frequently affected. Significant trauma is required; a degenerative desmitis and injury in association with damage to the cranial cruciate and medial meniscus along the lines of the so-called 'terrible triad' in human orthopaedics has not been demonstrated in the horse.
2. Radiology: entheseous new bone may be seen at ligament attachments; assuming the injury has been present long enough. OA is an inevitable consequence.
3. Stress radiographs may help to demonstrate instability.
4. Ultrasonography is the technique of choice to assess the integrity of the medial collateral ligament.
5. Attempts at stabilization with a prosthesis have often been unsuccessful, and the prognosis is generally poor.

Meniscal injury and associated meniscotibial ligament tears

Injury to the medial meniscus is a common cause of stifle-related lameness, with or without damage to the medial meniscotibial ligament (medial meniscal ligament). Localization is as for CrCL injury, and the diseases are inseparable on the basis of clinical signs alone.

Radiography

New bone may be evident at the insertion of the ligament, on the cranial aspect of the intercondylar eminence (this is often mistakenly identified as associated with the CrCL but the insertions are separate and distinct). A flexed lateromedial projection of the stifle highlights this region and is the view of choice in such cases; it can be taken in addition to, or as a substitute for, the weight-bearing lateromedial. Signs of OA may be present (especially at the medial tibial plateau, medial distal femur).

Ultrasonography

Unlike CrCL injuries, medial meniscal and meniscotibial ligament injuries are comparatively easy to document ultrasonographically. The medial (and lateral, although this is affected much less commonly) meniscus can be evaluated via a simple transcutaneous approach in the weight-bearing horse. This allows visualization of the middle third of the meniscus. The cranial third and the ligament can be appreciated by flexing the stifle and examining the region via cranial and craniomedial approaches.

Definitive diagnosis and treatment

The meniscus and associated ligaments can best be evaluated by a combination of imaging and surgical visualization; the techniques complement each other as neither can observe the meniscus in its entirety. However, surgical debridement requires general anaesthesia and arthroscopy. The prognosis varies according to use of the horse, the extent of the meniscal tear, time from injury to treatment, etc.

The use of biological treatments, such as stem-cell therapy in the treatment of meniscal injuries is being investigated.

Osteoarthritis (see also Chapter 15)

Comparatively uncommon as a primary disease in the stifle; the inciting cause (meniscus; ligament etc.) should be sought.

Calcinosis circumscripta

This is a disease of unknown aetiology but, fortunately, rarely of significance. The lateral aspect of the stifle is the site most commonly involved. Affected horses present with a mineralized plaque under the skin over the stifle/lateral thigh. It rarely communicates with the stifle joints and does not cause lameness. Radiographs demonstrate a granular, mineralized mass. No treatment is required. Its relationship to human tumoral calcinosis/calcinosis circumscripta is unclear, and it may require reclassification to distinguish it from these conditions.

Fibrotic/ossifying myopathy

Aetiology

Traumatic injury to the semitendinosus muscle results in scarring (and in some cases subsequent mineralization) with adhesions between the semitendinosus, semimembranosus and biceps femoris muscles.

Clinical signs

The affected hind limb is suddenly pulled distally and caudally at the cranial limit of the stride; this results in the foot 'slapping' the floor.

The scarring or mineralization of the affected muscle may be palpable as a hard area on the caudal aspect of the thigh.

Treatment

Mildly affected horses may not require treatment and may be able to cope with the gait abnormality. It is unclear how horses with this condition are affected at speed.

Surgical treatment involves tenectomy or myectomy of the semitendinosus muscle. The tenectomy procedure has fewer complications and may be preferable.

17.6 HIP

Anatomy

The hip is a ball and socket joint composed of the head of the femur and the acetabulum of the pelvis. In addition to the round ligament running from the fovea on the femoral head to the acetabular fossa as in other species, the horse also has an accessory femoral ligament that runs from the fovea medially and cranially through the acetabular notch and along the ventral surface of the pubis to insert on the prepubic tendon.

Osteoarthritis

Osteoarthritis of the hip in the horse is rare, but it is also difficult to diagnose so its prevalence may be underestimated. It may occur secondary to trauma which may cause rupture of the round ligament and consequent instability (subluxation). Clinically, the horse may show outward rotation of the affected limb at rest. Crepitus may be palpable externally or on rectal examination. Scintigraphy and/or radiography may help confirm the diagnosis, and ultrasonography can also be useful either pecutaneously or *per rectum*. Localization can be aided with intra-articular anaesthesia. The prognosis is usually poor.

Luxation

Luxation of the hip is also rare in the horse. It usually occurs as a result of trauma. The femoral head is usually displaced craniodorsally. The limb may be rotated outwards and appears shortened. The greater trochanter may appear particularly prominent, and crepitus may be palpable externally or *per rectum* on manipulation. The diagnosis may be confirmed radiographically or ultrasonographically. The radiographs should be carefully scrutinized for accompanying fractures of the dorsal rim of the acetabulum, although highly detailed images are difficult to obtain.

Closed reduction under general anaesthesia may be possible in recent injury, although recurrence is common. Both open reduction and salvage by femoral head excision have also been reported and are recommended for luxations that have been present for more than a few days.

Sepsis

Infection of the hip joint is rare in foals (usually haematogenous) and almost unheard of in mature horses unless associated with significant trauma or rare haematogenous spread/local invasion from abscesses. It can be difficult to diagnose because synoviocentesis of this joint, which provides fluid for definitive diagnosis, is difficult and uncommonly performed by most clinicians. Waiting for radiographs (again sometimes difficult to obtain) to show changes and confirm the diagnosis means missing the treatment opportunity. If suspected, for example in a foal with lameness and other signs of sepsis with no obvious cause, consider prompt referral. Synoviocentesis of the hip joint can be aided by ultrasound guidance.

Fractures

Fractures involving the hip region are usually the result of trauma from falls or collisions but may occur as a result of stress fractures

in racehorses. Theoretically any configuration is possible. If articular, the prognosis for future soundness is very poor, but rare cases return to training. Diagnosis can be frustratingly difficult, but scintigraphy is usually helpful (although transport to a suitable centre may not be possible). Ultrasound examination, especially *per rectum*, can be a very useful technique as the dorsomedial aspect of the acetabulum, the ischium, and part of the ilial shaft can all be assessed; however, false negatives are possible. Displaced ilial shaft fractures can sever the iliac or gluteal arteries and result in fatal haemorrhage.

FURTHER READING

Auer JA, Stick JA (2012) Equine surgery, 4th edn. Elsevier, St Louis

Baxter GM (2011) Adams and Stashak's lameness in horses. 6th edn. Wiley-Blackwell, Oxford

Butler JA, Colles CM, Dyson SJ, Kold SE, Poulos PW (2008) Clinical radiology of the horse, 3rd edn. Wiley-Blackwell, Oxford

Mcllwraith CW, Turner SA (1987) Equine surgery: advanced techniques. Lea & Febiger, Philadelphia

Ross MW, Dyson SJ (2003) Diagnosis and management of lameness in the horse. Elsevier, St Louis

Turner SA, Mcllwraith CW (1989) Techniques in large animal surgery. Lea & Febiger, Philadelphia

Wyn-Jones G (1988) Equine lameness. Blackwell Scientific Publications, Oxford

Chapter 18

Orthopaedics 4. The back and pelvis

Martin P. Weaver

CONTENTS

18.1 Anatomy of the horse's back 389
18.2 Diagnostic approach to diseases of the horse's back 389
History 389
Examination at rest 390
Examination at exercise 391
Aids to diagnosis 391
18.3 Disorders of the horse's back – conditions that may present as a back problem 392
Hind limb lameness 392
Temperamental or managerial difficulties 392
Hind limb incoordination – neurological disease 392
18.4 Vertebral column deformities 392
18.5 Soft-tissue injury 392
18.6 Fractures 393
18.7 Impingement and overriding of the dorsal spinous processes ('kissing spines') 393
18.8 Other conditions of the spine 393
Spondylosis deformans 393
Osteoarthritis 394
Osteomyelitis 394
Neoplasia 394
18.9 Sacroiliac joint disease 394
Acute sacroiliac injury 394
Chronic sacroiliac disease 394
18.10 Treatment of back conditions 395
18.11 Pelvic fractures 395
Hip luxation 397
18.12 Caudal aortic or iliac artery thrombosis 397
Further reading 397

18.1 ANATOMY OF THE HORSE'S BACK

The equine thoracolumbar spine is composed of a complex arrangement of soft-tissue structures supporting a comparatively rigid vertebral column. The equine back has been likened to a 'bow and string' arrangement, where the 'bow' is the almost rigid vertebral column; the supporting muscles and ligaments act as the 'string', maintaining the spine under tension.

There are usually 29 vertebrae in the back (T18, L6, S5); variations in the vertebral back formula are not unusual in clinically normal horses (e.g. 17 thoracic and 7 lumbar vertebrae). The vertebrae are held in place by a series of ligaments (Figure 18.1). The supraspinous ligament is the caudal continuation of the nuchal ligament and inserts at the tip of every thoracolumbar dorsal spinous process. The main muscles of the back include the largest muscle in the body, the longissimus dorsi, and the powerful gluteal muscles (Figure 18.2).

The neck, consisting of seven vertebrae, is most frequently associated with neurological disease and is covered in Chapter 11.

18.2 DIAGNOSTIC APPROACH TO DISEASES OF THE HORSE'S BACK

The back of the horse is defined here as the thoracolumbar (T1 to L6) vertebrae, the sacrococcygeal (S1 to Cy2) vertebrae and their associated structures, including the soft tissues. The most common presenting sign of back pain is loss of athletic performance.

History

The history is often of great help in determining whether the clinician is dealing with a genuine case of back pain, or whether factors such as poor behaviour or schooling may be the underlying problem. The history is frequently long and involved; a questionnaire helps to ensure that all relevant details are obtained. Specific aspects include:

- The type of work: the horse may not be suitable for the work expected of it; certain forms of exercise may prove more problematic, e.g. jumping.

© 2013 Elsevier Ltd
DOI: 10.1016/B978-0-7020-2801-4.00018-3

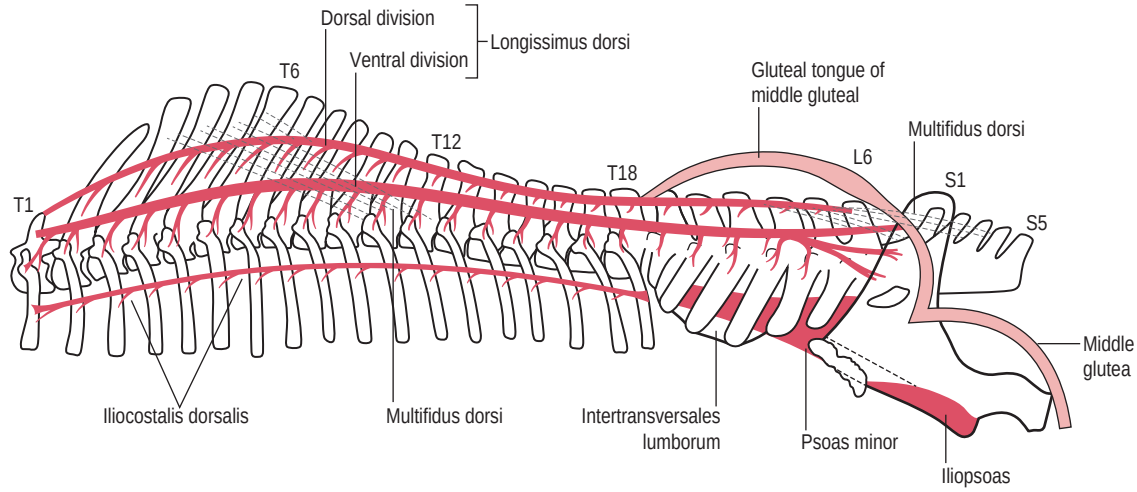


Figure 18.1 Epaxial muscles of the equine thoracolumbar spine (excluding the superficial layer of muscles). The multifidus dorsi forms a series of segmental muscles that lie alongside the spinous processes from T1 to S5.
Reprinted from Jeffcott LB, Dalin G (1980) Equine Veterinary Journal 12:101–108, with kind permission from Professor Jeffcott and Wiley-Blackwell.

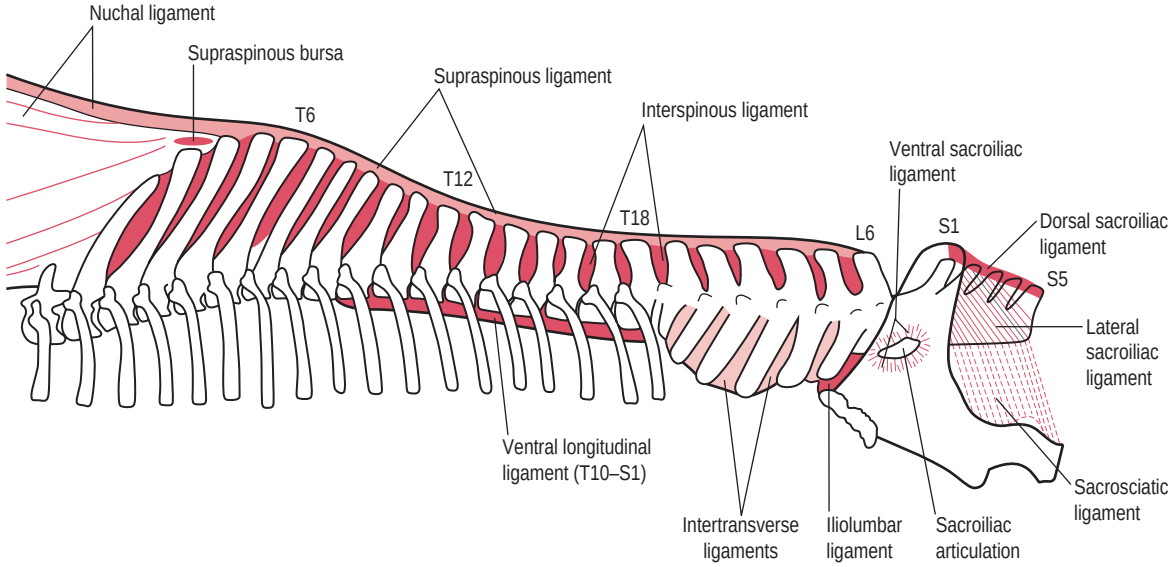


Figure 18.2 Ligaments of the equine thoracolumbar and sacral spine.
Reprinted from Jeffcott LB, Dalin G (1980) Equine Veterinary Journal 12:101–108, with kind permission from Professor Jeffcott and Wiley-Blackwell.

- Temperament: owners of horses with a genuine back problem frequently report that the horse has become ill-tempered, fractious or reluctant to work.
- Acute or chronic problem: if the problem has arisen following recent trauma, details of the accident may help to localize the site of pain, although more typically no such incident will have been observed.
- Position of limbs: the horse may no longer straddle when urinating or defecating; there may be reluctance to bear more weight on one hind limb (e.g. during shoeing).
- Bucking: if the history includes episodes of bucking, the safety of any riders should be paramount, regardless of whether the signs are due to pain in the horse's back or are behavioural; this may limit the clinical investigation, and this should be explained to the owner at the outset.

Examination at rest

Stocks are useful for this part of the investigation, and the horse must be relaxed and bearing weight evenly on all four limbs.

Conformation

General condition should be noted. Abnormal curvature of the back is assessed: i.e. lordosis (ventral deviation), kyphosis (dorsal deviation) or scoliosis (lateral deviation of spine due to spinal malformation or asymmetric muscle spasm). Conformational defects of the limbs (e.g. straight hocks characteristic of proximal suspensory disease) may suggest bilateral hind limb lameness.

Muscle wastage

- Should be differentiated from poor condition.
- Wastage may be unilateral or bilateral or focal.

- Gluteal, longissimus dorsi and thigh muscles are most commonly affected.

Swellings and marks

- Assess significance of any saddle marks.
- Note any scars or localized soft-tissue swellings.
- 'Hunter's bump' ('jumper's bump') is a prominence of the lumbar or sacral dorsal spinous processes due to poor muscling, and is usually of no clinical significance in old horses.

Palpation/manipulation

- Palpate soft tissues and summits of dorsal spinous processes (DSPs) for pain, heat or swelling.
- Assess flexibility of the back by stimulation reflex extension (dipping), flexion (arching) and lateral bending. This is done by running a blunt probe along the midline of the back from the withers to the sacroiliac region, and laterally from midline. It is important to note that a high range of reflex back movements indicates a normal, pain-free horse, although these movements are often interpreted as a sign of pain. A horse with back pain braces its back rigidly and shows clear signs of resentment.
- Similarly, pressure applied upwards onto the ventral aspect of the sternum should induce a normal horse to arch its back without signs of discomfort.
- Apply pressure to both tubera coxae and tubera sacrale; pain here may indicate fracture of the ilium or a problem in the pelvic or sacroiliac region.
- Poor tail tone may indicate early cauda equina neuritis.

Examination at exercise

In hand

- The horse is walked and then trotted in-hand for lameness evaluation.
- Overt lameness or gait abnormalities should be identified; many horses with suspected back pain suffer from hind limb lameness. If present, this should be investigated first as described in other chapters.
- Positive hind limb flexion test(s) suggest lameness rather than back pain.
- Back pain often results in a reduced length of stride of the hind limbs and less hock flexion, but the same signs are seen in chronic hind limb lameness.
- Turning the horse tightly in both directions may provoke longissimus dorsi spasm due to pain caused by lateral spinal flexion.
- There may be reluctance to move backwards, with the head being raised and back muscle spasm because this maneuver may provoke spasms of the back muscles.

Lunging

- The horse should be exercised on both reins at trot and canter.
- Dragging and/or plaiting of hind toes, exaggerated longissimus dorsi contractions, high head carriage, tail swishing, and poor hind limb impulsion may be seen. These signs may also be seen in some unfit or untrained horses.
- Signs are often more apparent on one rein.

Riding

This should be performed – preferably by the horse's usual rider – unless the horse is considered dangerous to ride.

Observe the process of tacking-up and mounting; some horses may dip ventrally when the girth is tightened or when they are mounted. This is, however, often an acquired habit (i.e. 'cold back') and usually not a sign of back pain, although it may reflect a previous episode of discomfort: for example from an ill-fitting saddle.

Assessment of gait is repeated at walk, trot and canter, on both reins.

Following a period of rest, the horse should be trotted up again; a lameness may now be apparent, or in cases of low-grade exertional rhabdomyolysis ('tying up') there may be stiffness and reluctance to move.

Aids to diagnosis

Clinical biochemistry

The blood concentrations of the muscle-derived enzymes creatinine kinase (CK) and aspartate transaminase (AST) are measured before, immediately following, and about 12 hours after 10–20 minutes of exercise (trotting and cantering). Significant rises (by more than 100–200%) suggest an exertional rhabdomyolysis.

Radiography (see Chapter 25)

Powerful equipment (150 kVp/300 mAs) is required for a comprehensive radiographic examination of the thoracolumbar spine. Smaller portable units are capable of imaging the tips of the dorsal spinous processes (DSPs) from T6 to the cranial lumbar region, but not the vertebral bodies or dorsal intervertebral articulations, a common site of painful osteoarthritis.

Sedation (e.g. with detomidine or xylazine) should be used to reduce movement blur and reduce the restraint required to a minimum; this is important in optimizing radiation safety for the handlers. Large cassettes are used and should never be hand-held.

Typical exposure values are 80 kVp/30 mAs for the DSPs of the mid-back region.

Scintigraphy (see Chapter 25)

Bone scanning using radioactive technetium labelled with methylene diphosphonate indicate areas of abnormal skeletal metabolic rate due to trauma (e.g. fracture), inflammation or infection (Figure 18.3). A gamma camera is used, and the examination can be carried out on standing, sedated horses, but images of better quality are obtained under general anaesthesia.

Ultrasonography (see Chapter 25)

This technique can be used to evaluate soft-tissue structures such as the supraspinous, interspinous and dorsal sacroiliac ligaments, and is essental when directing a needle towards deep structures such as the

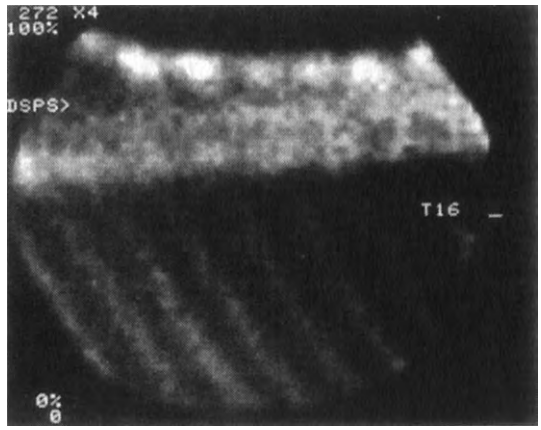


Figure 18.3 Image from a bone scan of the thoracic spine of an adult horse with 'kissing spines'. The bright zones at the top of the image represent areas of increased bone metabolic rate where the tips of the processes are overriding. See also Figure 18.5.

dorsal intervertebral articulations. Its value as a routine screening tool in cases of suspected back pain, for example to detect muscle abnormalities, remains unproven.

Local analgesia

This is most useful to confirm the suspected site of pain due to DSP impingement ('kissing spines').

The site(s) is/are localized by radiography, prepared aseptically, and 5–10 mL of 2% lignocaine or mepivicaine injected into the interspinous space(s) using a 3.75-cm 18-gauge needle. The dorsal intervertebral (facet) joints can also be injected, although this is technically demanding due to their deeper location; ultrasound guidance is recommended.

Systemic analgesia

If there is doubt about the presence of pain, particularly in the chronic case, the horse may be re-evaluated after receiving a short course of an orally administered non-steroidal anti-inflammatory agent (e.g. phenylbutazone) or shortly after intravenous injection of similar medication. This can be a most useful test to distinguish behavioural or equitation problems from those caused by back pain.

Other diagnostic tests

Thermography has been used in the diagnosis of back problems in horses but remains unproven. Several papers have described using the horse's response to measured focal pressure as a diagnostic tool.

18.3 DISORDERS OF THE HORSE'S BACK – CONDITIONS THAT MAY PRESENT AS A BACK PROBLEM

A wide range of conditions can be mistaken for back pain by owners. It is important to differentiate these from genuine cases of back disorders.

Hind limb lameness

The location of the lesion should be identified by routine lameness work-up.

Temperamental or managerial difficulties

These include a wide range of conditions:

- hypersensitivity of back ('cold back').
- ill-fitting tack.
- poor schooling and/or equitation.
- temperamental problems.
- lack of ability of horse to perform to owner's expectations.

Many of these cases challenge the veterinary surgeon's communication skills to the utmost, as owners frequently do not take kindly to criticism of their style of riding, however carefully this is expressed.

Hind limb incoordination – neurological disease

Horses with mild spinal cord compression/disease in the cervical (see Chapter 11) or thoracolumbar spine may present with stiffness or weakness behind, resembling a back problem.

18.4 VERTEBRAL COLUMN DEFORMITIES

Lesions

These include:

- Scoliosis (lateral curvature).
- Lordosis (ventral curvature).
- Kyphosis (dorsal curvature).
- Synostosis (vertebral fusion).

Aetiology and pathogenesis

- May be congenital or acquired.
- Often becomes more pronounced with age.
- Lordosis may be due to hypoplasia of the articular facets T7 to T10 and may be complicated by crowding of the DSPs in the mid-back.
- Instability and abnormal loading may predispose to injury (e.g. vertebral fractures).
- These horses are not necessarily in pain, but the deformity may preclude ridden exercise.

Diagnosis

- Examination, including visual inspection and palpation. Scoliosis can only be detected when observing from dorsal.
- Radiographic examination.

18.5 SOFT-TISSUE INJURY

Lesions

- Strain of individual muscle groups (e.g. longissimus dorsi, middle gluteal and sublumbar musculature).
- Damage to ligaments of the back (e.g. supraspinous ligament, dorsal sacroiliac ligament).
- Generalized post-exercise myopathy (myositis; rhabdomyolysis) affecting back musculature (see Chapter 21).

Aetiology and pathogenesis

Exertional rhabdomyolysis follows glycogen breakdown in muscles during exercise without sufficient oxygen supply to the tissues, resulting in lactic acid accumulation and pain during or after exercise. Factors such as poor conditioning, familial predisposition, high-energy feed during a rest period, and dietary mineral imbalances have been implicated. Other soft-tissue lesions are frequently due to trauma, such as a fall. Unless sufficient rest is given, these lesions may become chronic.

Clinical signs

- Poor performance.
- Stiff gait.
- Back muscle spasm on palpation or exercise results in a stiff gait.
- Soft-tissue swelling of affected muscle groups.
- Many affected horses show no specific signs.

Diagnosis

- A significant (at least two-fold) post-exercise rise in muscle enzymes (CK and/or AST) signifies myopathy.
- Palpation may localize sites of swelling and pain.
- Ultrasonography can be useful to demonstrate enlarged and hypoechoic ligaments but requires familiarization with the normal appearance of the soft tissue structures of the back because of the presence of insignificant hypoechoic areas.

18.6 FRACTURES

Sites of fractures (Figure 18.4)

- Dorsal spinous processes: one or several processes may be affected.
- Vertebral body and neural arch: the first three thoracic, first three lumbar vertebrae and T12 (i.e. mid-point of the back) appear to be more susceptible to fractures.
- Sacral fractures with sacral nerve damage may result in local neurogenic muscle atrophy and tail paralysis.
- Coccygeal fractures may heal with permanent kinking of the tail, or tail flaccidity without perineal paralysis.

Aetiology and pathogenesis

There may be predisposing factors (e.g. lordosis; osteomyelitis, abnormal muscle spasm such as occurs in tetanus).

Multiple fractures of the DSPs at the withers (T4–T10) are usually the result of a backwards fall or somersault. Usually young horses (6 months to 3 years) are affected.

Other vertebral fractures are also usually due to trauma: e.g. a fall at speed.

Diagnosis

Clinical signs. Fractures of DSPs are associated with localized pain, gross swelling, displacement of the affected summits, and possibly mild fore limb lameness. Palpation is usually only useful if there is DSP displacement. With other fractures one can expect the horse to brace its back when palpated in an effort to avoid reflex flexion or extension.

Signs of vertebral body fractures vary with the degree of spinal cord damage, from mild hind limb ataxia to acute paraplegia. Young foals are especially prone to crush-type fractures of the lumbar region.

Radiography. Radiography of regions caudal to the thoracolumbar junction is difficult in adults due to tissue thickness and superimposition of abdominal organs, although definition of fractures in foals is usually good. Radiographic signs of vertebral fractures include:

- malalignment of DSP or vertebral body.
- shortening of vertebral body due to 'crushing'-type fracture.
- sclerosis and new bone formation in chronic cases.

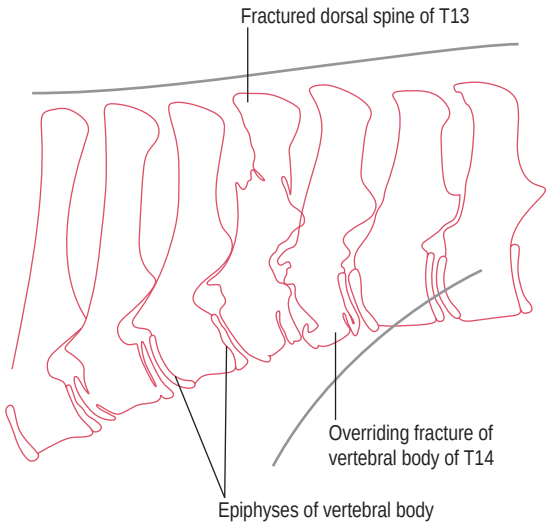


Figure 18.4 Diagram of a vertebral (T14) crush fracture, associated with a fracture of the T13 vertebral body and fractured dorsal spine of T13.

Scintigraphy. In those horses where the above examinations have not confirmed the presence of a fracture, scintigraphy can identify its presence. This is particularly true for stress fractures of the vertebrae seen in racehorses.

18.7 IMPINGEMENT AND OVERRIDING OF THE DORSAL SPINOUS PROCESSES ('KISSING SPINES')

Aetiology and pathogenesis

This condition is seen most frequently in young adult thoroughbred or TB-cross horses; it is rarely seen in ponies and draught horses.

Crowding of processes occurs most commonly from T12 to T17 (i.e. beneath-saddle region).

Pressure at site(s) of crowding produces bony changes, such as pseudarthroses (i.e. false joints) at the tips. These bony changes are sometimes accompanied by pain.

Some normal horses may show marked 'kissing spines' with no apparent ill effects. This may reflect individual variation in pain thresholds or that impingement is not always associated with pain. Consequently, confident diagnosis of significance is challenging.

Diagnosis

Clinical signs

- Usually insidious in onset (i.e. change in temperament, poor performance, reluctance to jump).
- Less commonly, acute onset seen following exacerbation by trauma.
- Pain is usually low-grade or not detectable by palpation, but spinal flexion in response to manipulation is restricted.
- Deep palpation reveals tips of DSPs close together in affected region.

Radiography

- Mild cases: narrowing of interspinous spaces; sclerosis of caudal and/or cranial edges of processes.
- Advanced cases: overriding of processes, especially at tips, with sclerosis, bony remodelling and cyst-like bone lucencies (Figure 18.5 and Figure 25.9).

Scintigraphy. Increased radionuclide uptake in regions of dorsal spinous impingement may increase the likelihood that the impingement is significant (Figure 18.3).

Local analgesia. The site of pain can be confirmed by local infiltration, followed by exercise, as already described.

18.8 OTHER CONDITIONS OF THE SPINE

Spondylosis deformans

- Formation of new bone on the ventral surface of the spine at one or several intervertebral joints (synonym: ventral spondylosis).
- Predilection sites: T9–T15.
- Can occur as an incidental finding. Clinical signs involve low-grade to severe pain or discomfort, stiffness of the back, reluctance to work, and impaired performance. There may be partial improvement with NSAID treatment.
- Diagnosis: radiography.

Figure 18.5 Post-mortem radiograph of thoracic 'kissing spines', showing marked overriding, sclerosis and bone 'cysts' at the tips of the dorsal spinous processes (same horse as in Figure 18.3).

Osteoarthritis

- Degenerative joint disease may affect the dorsal intervertebral joints in the mid-thoracic to lumbar regions.
- Some affected horses may show no clinical signs.
- If severe, may cause low-grade pain, stiffness and poor performance.
- Diagnosis: radiography; scintigraphy (bone scanning); ultrasonography; exercise before and after ultrasound-guided injections of local anaesthetic solution.

Osteomyelitis

- Osteomyelitis is an infrequent cause of back disease in the horse, and occurs more commonly in young horses and foals. Infection may occur by the haematogenous route or by extension of an abscess.
- Clinical signs vary with site and degree of spinal cord compression.
- Diagnosis: haematology (leucocytosis; hyperfibrinogenaemia, neutrophilia), radiography, scintigraphy. May be focal pain on clinical examination.
- Treatment: antimicrobial and anti-inflammatory agents.

Neoplasia

Tumours in the equine vertebral column or pelvis are very rare. Multiple myeloma (plasma-cell myeloma) may cause bone lysis, but other signs are usually more prominent.

18.9 SACROILIAC JOINT DISEASE

Acute sacroiliac injury

- Usually the result of trauma.
- May be asymmetry of the hindquarters with the affected side of the pelvis dropped near midline.
- May be severe pain or crepitation on deep palpation.
- Commonly a cause of lameness of the hind limb on the affected side.

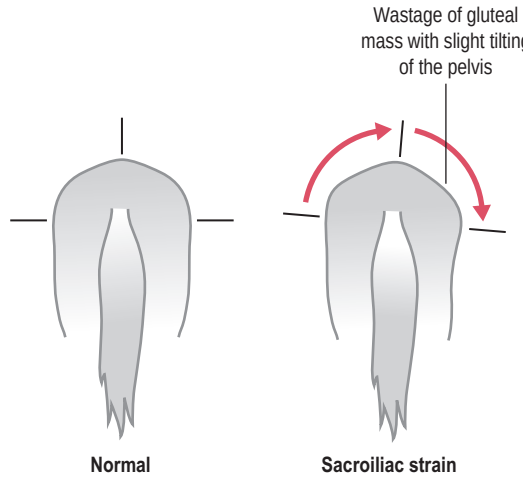


Figure 18.6 Diagram of a normal and a tilted pelvis subsequent to sacroiliac strain.
Reprinted from Jeffcott LB (1980) Equine Veterinary Journal 12(4):197–210, with kind permission of Professor Jeffcott and Wiley-Blackwell.

Chronic sacroiliac disease

Aetiology and pathogenesis

- Seen in large-framed horses with weak quarters.
- Presumed to result from low-grade instability at the joint after initial trauma.
- On post-mortem examination, the joint surface area is significantly increased by caudal or medial bony extensions.

Clinical signs

- Overt pain is rare; hind limb flexion tests are usually negative.
- Gluteal wastage and lowered tuber coxae on affected side usually present (Figure 18.6).
- Mild hind limb lameness or uneven gait that is best seen at slow trot.
- Discomfort when ridden which can include sudden kicking out.
- Support of the diagnosis can be provided by gamma scintigraphy where there is increased uptake abaxial to the tuber sacrale.

18.10 TREATMENT OF BACK CONDITIONS

The lack of effective conventional treatment for many cases of back pain and the frequent difficulty in coming to an accurate diagnosis have resulted in a wide range of 'alternative' therapies. Few treatment methods have been assessed objectively.

Medical treatment

Non-steroidal anti-inflammatory drugs (NSAIDs) (e.g. phenylbutazone, flunixin meglumine or suxibuzone) are appropriate to use in acutely painful cases, and to assist a controlled return to work. This is particularly important in chronic sacroiliac disease, where fitness and improved muscle bulk are vital to recovery.

Long-acting corticosteroids have been injected locally into sites of 'kissing spines' and into the affected sacro-iliac joint, with anecdotal evidence of beneficial results. Recently, descriptions have been published for the insertion of an 18-G spinal needle (20–25 cm, 10–12 inch) close to the sacroiliac joint with which to achieve more targeted injection (Figure 18.7). Sclerosing agents have also been used. Anti-inflammatory medication can also be injected into diseased dorsal intervertebral joints with the help of ultrasound localization. Tiludronate has also been reported to be effective for treating some cases of overriding dorsal spinous processes and osteoarthritis of the thoracolumbar verterbral column.

Surgical treatment

Resection of the tips of crowded dorsal spinous processes may be carried out following a positive result to local analgesic agent infiltration and where conservative treatment has failed to provide long-term alleviation of signs. This can be achieved under general anaesthesia or with the horse sedated and standing. A study that followed up over 200 horses that had had this surgery reported that over 70% were able to return to full work.

Rest

A sufficient period of rest is often the most important therapy in many cases. This should be box-rest for 3–6 weeks in severe cases, followed by turning out in a small paddock for 2 to 12 months.

Other treatments

Physiotherapy appears to be useful as an adjunct to a controlled programme of work. Manipulation of the back may work in some cases by altering muscle tension; however, the effect is usually temporary. Extra-corporeal shockwave therapy has also been recommended for treating back pain in horses.

Prognosis for back conditions

In a series of 190 horses diagnosed with chronic back pain, 57% recovered irrespective of the form of treatment adopted or diagnosis made. In cases of 'kissing spines', the prognosis for full recovery was reduced as the number of sites affected increased. Some horses may still perform satisfactorily despite a low-grade back problem, and complete spontaneous recovery is not uncommon.

Figure 18.7 Injection of the sacroiliac joint.

18.11 PELVIC FRACTURES

Aetiology and pathogenesis

Fractures of the pelvis are not infrequent and usually result from trauma or can be stress fractures in young racehorses. They may occur in a bad fall, or during high-speed galloping, particularly in young racehorses. Excessive abduction of the hind limbs ('the splits') can also result in pelvic fracture.

Fracture sites are most likely to be at the ilial wing (usually in horses over 6 years of age) or shaft, but may also occur at the acetabulum, pubic bone or ischium (Figure 18.8). Fracture of the tuber coxae is frequently caused by trauma inflicted while going through a narrow gate or doorway. The fracture is usually simple; rarely, the tuber coxae may be exposed by a wound. Fragments at the tuber ischia or tuber coxae only rarely sequestrate.

Clinical signs

The signs vary depending on the site of injury.

Unilateral or bilateral hind limb lameness is common. Pain or crepitus may be apparent on manipulation of the limb on the affected

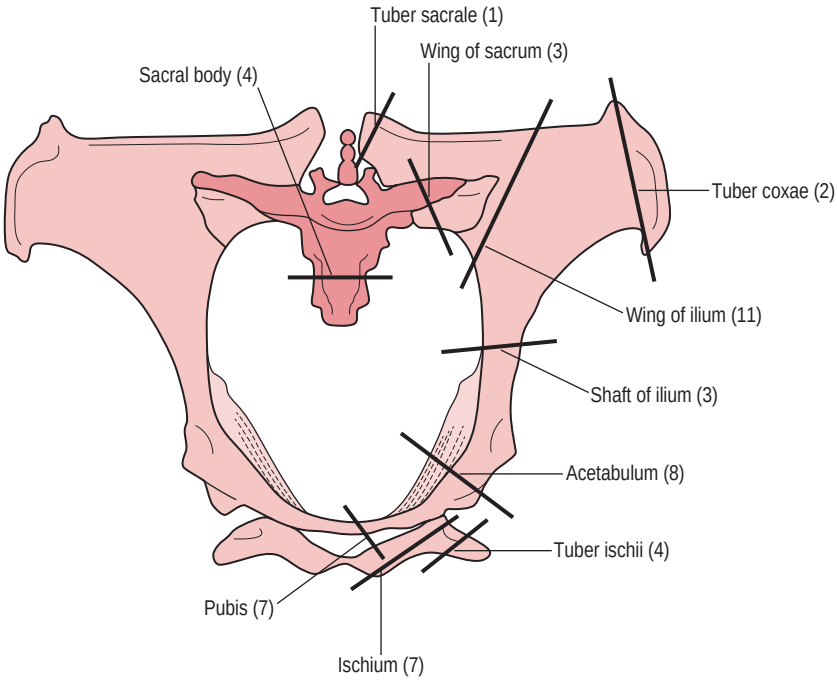


Figure 18.8 Diagram of the sites and incidence of 50 pelvic fractures in 41 horses.
Reprinted from Jeffcott LB (1982) Veterinary Clinics of North America: Equine Practice 3(3):34. Reprinted with permission from Elsevier Ltd.

side if the fracture is recent, in which case there is usually also associated soft-tissue swelling. In severe cases, with marked comminution or involvement of the acetabulum, the horse may be unable to rise.

In chronic cases, muscle wastage at the affected quarter is common and may be severe. Fracture of the ilial wing often results in asymmetry of the tubera sacrale, with the affected side lower; while a fractured tuber coxae is also be visibly 'dropped'.

Deep palpation over affected area may elicit pain. A fracture through the obturator foramen or pubic symphysis may cause a bilateral shortened hind limb stride.

A brief period of rest may result in rapid apparent resolution of the lameness, but recurrence at increased severity is likely on return to exercise. Accurate diagnosis and prognosis are therefore essential.

Diagnosis

- Examination and palpation: rectal palpation should always be performed, but is useful mainly in displaced fractures of the pubis or acetabulum, or to detect a large haematoma.
- Radiography (see Chapter 25): ventrodorsal views – with the horse anaesthetized and in dorsal recumbency and frog-legged – are required. A grid is used to reduce scatter. A technique to radiograph the pelvis in the standing horse has been described, but this requires a mobile Bucky and involves some risk to both the X-ray tube and the horse, as well as a high level of scatter radiation exposure to personnel. A full examination of pelvic and lumbosacral regions may require six radiographs, using large cassettes. By tipping each hip joint towards the cassette, better views of the dorsal acetabular edge and joint are obtained.
- Scintigraphy (see Chapter 25): this readily shows marked increases in bone turnover associated with fractures (see Figure 25.65).
- Ultrasonography (see Chapter 25): this is a reliable and simple technique to use in detecting some pelvic fractures, particularly of the ilium (Figure 18.9). Ultrasonography may also be used to monitor healing and to help determine when exercise can be recommenced.

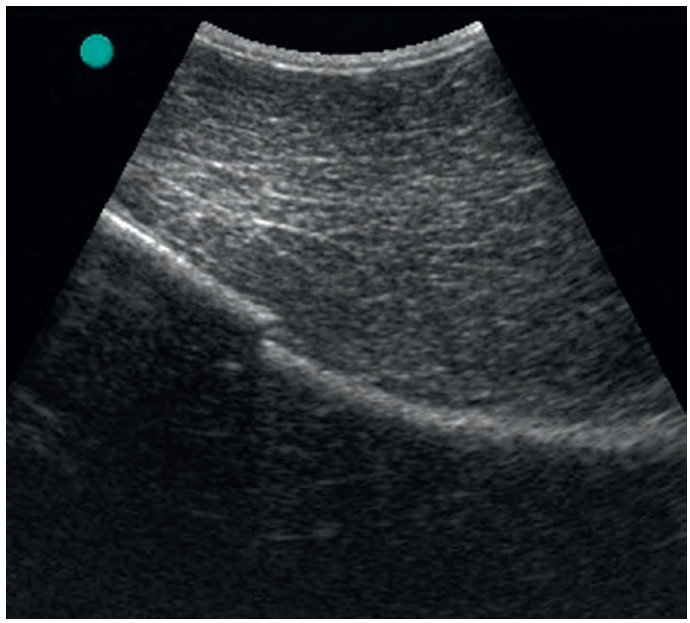


Figure 18.9 Ultrasonogram of an ileal wing fracture. The line of the bone surface has a step at the fracture site.

Treatment

The only surgical treatment currently undertaken for pelvic fractures is to remove tuber ischii or tuber coxae bone fragments in horses where there is sequestration or osteitis of the fragment(s).

All cases should be box-rested for a period of 1–3 months (optionally, maintained, cross-tied), followed by restricted exercise in a small paddock until healing has taken place. This may take up to 1 year, although for most minimally displaced ilial fractures 3–6 months are sufficient.

Prognosis

Horses with fractures of the tuber coxae, tuber ischii and wing of ilium carry a good prognosis for a full return to work. A poor prognosis must be given for cases of fracture through the body of the pubis, shaft of ilium or acetabulum.

Hip luxation

Hip luxation usually occurs after a fall. It can be difficult to distinguish from a pelvis fracture, which produces many similar clinical signs. In addition to these signs, a hip luxation causes the affected limb to be held outwardly rotated with the point of the hock higher on the affected side (because luxation is usually dorsal).

Diagnosis can be confirmed using the same radiographic techniques as those described above for pelvic fractures. Gamma scintigraphy is frequently negative unless the luxation is accompanied by an acetabular fracture.

If the luxation is recent (within 24–48 hours) reduction under general anaesthesia using ropes around the inguinal area and using the horse's weight with a hoist attached to the distal limb to assist in reduction can be successful. However, because of the shallow acetabulum of the horse, re-luxation is common. Very small ponies can be managed in an Ehmer sling.

For more chronic cases, surgical reduction with toggles and/or capsular imbrication can be used, although the forces involved, even in small ponies, can disrupt even the most secure repair. Excision arthroplasty has been attempted in small ponies.

18.12 CAUDAL AORTIC OR ILIAC ARTERY THROMBOSIS

Aetiology and pathogenesis

The pathogenesis of thrombosis of the caudal aorta or iliac arteries is unknown, but the migration of *Strongylus vulgaris* larvae has been implicated. Thrombosis occurs on the dorsal wall of the artery at the quadrification of the aorta and causes secondary emboli in the iliac or femoral arteries.

Signs result when an embolus obstructs a distal vessel, frequently at the popliteal artery bifurcation, and depend on the degree of obstruction. The condition may occur in all breeds and ages but is detected more frequently in young horses performing at a high level.

Clinical signs

In the early stages there is often the appearance of a subtle hind limb lameness or back problem. As exercise increases, clinical signs become more apparent: stiffness in affected limb, asymmetric hind limb gait, knuckling over or toe-dragging. Signs usually resolve after 20 minutes of rest.

On palpation, the affected limb is cooler than the contralateral limb, particularly distally. Arterial pulsation is reduced distal to the site of obstruction, and the superficial veins may be collapsed. Much less frequently, horses may present with acute pain and severely lame due to sudden and extensive thromboembolism.

Diagnosis

- Rectal palpation: the terminal aorta and iliac arteries should be palpated; typically, a firmness and reduction in pulse is detectable. Markedly occluded vessels may be enlarged and hardened. However, there may be aortic thrombosis and only peripheral embolism, with the iliac arteries unaffected.
- Ultrasonography: using a linear probe per rectum. Requires good familiarization with normal structures.
- Arteriography: requires specialized facilities.

Treatment

Analgesia, using NSAIDs, is indicated, particularly as controlled movement at a level below the threshold which results in clinical signs helps maintain collateral circulation. Aspirin can help to relieve pain associated with local vasoconstriction.

Although anthelmintics have been recommended, they do not resolve an existing thrombus. Surgical treatment (thrombectomy) may be possible; a recent publication recorded an incidence of success of 65% in 17 cases.

Prognosis

The outlook for a return to work without surgery is relatively poor. Most cases show gradual deterioration, with the level of work requiring progressive reduction.

FURTHER READING

Dyson S, Murray R (2003) Pain associated with the sacroiliac joint region: a clinical study of 74 horses. Equine Veterinary Journal 35:240–245

Engeli E, Haussler KK, Erb HN (2004) Development and validation of a periarticular injection technique of the sacroiliac joint in horses. Equine Veterinary Journal 36(4):324–330

Erichsen C, Eksell P et al (2004) Relationship between scintigraphic and radiographic evaluations of spinous processes in the thoracolumbar spine in riding horses without clinical signs of back problems. Equine Veterinary Journal 36(6):458–465

Haussler KK, Erb HN (2006) Pressure algometry for the detection of induced back pain in horses: a preliminary study. Equine Veterinary Journal 38:76–81

Henson FMD (2009) Equine Back Pathology. Diagnosis and Treatment. Wiley-Blackwell, Oxford, UK

Jeffcott LB (1979) Radiographic examination of the equine vertebral column. Veterinary Radiology 20:135–139

Jeffcott LB (1980) Back problems in the horse – a method of clinical examination. Veterinary Record suppl In Practice 5:4–15

Walmsley JH, Pettersson H et al (2002) Impingement of the dorsal spinous processes in 215 horses: case selection, surgical technique and results. Equine Veterinary Journal 34(1):23–28

Weaver MP, Jeffcott LB et al (1999) Back problems. Radiology and scintigraphy. Veterinary Clinics of North America Equine Pract 15(1):113–129, vii-viii

Chapter 19

Infectious diseases and parasitology

Sandy Love, Tim S. Mair

CONTENTS

19.1 Investigation of infectious disease 400
19.2 Diagnosis of viral and bacterial infections 400
Diagnosis of viral infections 400
Diagnosis of bacterial infections 401
19.3 Equine influenza 401
19.4 The equine herpesviruses 402
The viruses 402
Equine herpesvirus 1 (rhinopneumonitis) 402
Equine herpesvirus 4 (rhinopneumonitis) 403
Equine herpesvirus 3 (coital vesicular exanthema) 403
19.5 Equine viral arteritis (EVA) 403
19.6 Equine adenovirus 404
19.7 Equine picornaviruses (rhinitis viruses) 404
19.8 Equine infectious anaemia (EIA) 405
19.9 African horse sickness (AHS) 405
19.10 Vesicular stomatitis 406
19.11 Hendra virus 406
19.12 West Nile virus 406
19.13 Equine encephalosis 407
19.14 Rotavirus 407
19.15 Other equine viral infections 408
19.16 Salmonellosis 408
19.17 Clostridial diseases 410
19.18 Strangles 411
19.19 Potomac horse fever 413
19.20 Proliferative enteropathy (*Lawsonia intracellularis*) 413
19.21 Leptospirosis 414
19.22 Anthrax 415
19.23 Brucellosis 415
19.24 Tuberculosis 415
19.25 Glanders 415
19.26 Lyme disease 415
19.27 Equine granulocytic ehrlichiosis 415
19.28 *Corynebacterium pseudotuberculosis* 415
19.29 Tyzzer's disease 415
19.30 Other equine bacterial infections 415
19.31 Fungal infections 416
19.32 Investigation of parasite-associated disease 416
Introduction 416
History 416
Physical examination 416
Faecal worm egg count (FWEC) 416
Faecal egg count reduction test (FECRT) 416
Faecal larvae 416
Haematology/blood biochemistry 416
Serology 416
Bronchoalveolar lavage 417
Cytology/histopathology 417
Post-mortem examination 417
19.33 Control strategies for equine parasite infections 417
19.34 Strongyles 418
19.35 Ascarids 420
19.36 Tapeworms 420
19.37 Bots 420
19.38 Lungworm 421
19.39 *Strongyloides westeri* 421
19.40 *Oxyuris equi* 421
19.41 Piroplasmosis 421
19.42 Trypanosomiasis 421
19.43 Other equine parasitic infections 422
Further reading 422

© 2013 Elsevier Ltd
DOI: 10.1016/B978-0-7020-2801-4.00019-5

19.1 INVESTIGATION OF INFECTIOUS DISEASE

In equine practice most illnesses/disorders affect individual patients, and so the individual, rather than the group, is often regarded as the most important unit of concern. However, from time to time equine veterinarians have to deal with outbreaks and epidemics of disease. Increased international travel of horses as well as potential climate change increase the risks of spread of infectious diseases, including diseases traditionally considered to be 'exotic' to particular geographical areas. Early recognition of disease outbreaks is central to their effective control. There are some fundamental, conceptual aspects of investigating group disease which is often, but by no means always, of an infectious nature. The basis of the approach is simply a logical interpretation of biological, historical and clinical data to study the occurrence of disease within a population. The veterinarian will often subconsciously apply basic principles to a clinical scenario, but less commonly the investigation of a disease outbreak will need to be performed by use of sophisticated, specialist epidemiological techniques.

The following decision-making steps may be employed when investigating an outbreak/epidemic or apparent group condition:

1. Is there an apparent increase in the incidence and/or prevalence of disease above the normal levels? To confirm that the apparent increase is real it is essential to know:
   - how new cases are being clinically defined.
   - if awareness of a condition has increased the level of reporting, rather than a true increase.
   - the rate of occurrence (i.e. incidence) and/or proportion of the group/population affected (i.e. prevalence) prior to the apparent increase.
2. If possible, confirm the diagnosis by appropriate additional tests.
3. Examine the distribution of cases within the group to establish 'risk factors'. In particular:
   - look for associations:
     when did cases occur? e.g. post-weaning, at housing etc.
     where did cases occur? e.g. in a specific barn or pasture.
     which animals are affected? e.g. age, sex, breed, location.
   - obtain rate of disease occurrence in different animal categories, e.g. in foals versus mares.
   - obtain detailed information from a sample of individuals within different animal categories, e.g. duration/severity of illness in young versus old.
4. On the basis of analysis of information in 1 to 3 hypothesize why the increase in disease occurred, e.g. introduction of an animal incubating disease; change in environmental temperatures etc.
5. The purpose of going through the processes of 1 to 4 is to use the findings to formulate a control strategy. Ideally this would be by identifying the cause(s) of an increase in disease and by removing it (them) to prevent further cases. Most often control is based on:
   - initiation of immediate control measures pending confirmation of diagnosis and other investigative tests.
   - isolation/segregation of affected cases (and those with known direct contact prior to intervention).
   - treatment of known cases would seem logical but is not always appropriate and requires specific knowledge of the disease/condition.
   - protect high risk groups, e.g. administer hyperimmune plasma to foals during rotavirus outbreak.
   - institute measures to prevent recurrence, e.g. establish quarantine protocol for new intake animals.

These comments are perhaps rather obvious, but it cannot be overemphasized how easy it is to focus on the immediate clinical problem rather than to stand back and evaluate all the available information. The following information and guidelines on infectious and parasitic diseases of horses will be most useful if considered against the background of the foregoing comments.

19.2 DIAGNOSIS OF VIRAL AND BACTERIAL INFECTIONS

An accurate diagnosis is a prerequisite to specific treatment but may be especially important in infectious diseases where there is a risk of spread to other horses. The presumptive diagnosis of viral and bacterial infections may be easy in certain diseases, being made solely on the characteristics of the history, clinical signs and physical examinations. In other cases, these characteristics may lead to a suspicion of a certain infection, which needs to be confirmed by additional information, usually in the form of laboratory tests. In other situations, such as where subclinical infection or a carrier state exists, a diagnosis may be possible only by means of laboratory examinations.

A specific diagnosis will not be required in every case of suspected infectious disease. In respiratory viral infections, for example, there may be no therapeutic benefit to be gained from knowing which virus is involved; furthermore, the results of laboratory tests may take several weeks, by which time the affected horse(s) is likely to have recovered. However, in certain situations, a specific diagnosis may be necessary (e.g. in the face of an epidemic, or when a large population of horses is at risk, or if an exotic infection is suspected). A variety of laboratory tests for the accurate diagnosis of infectious agents is available, and it is recommended that the clinician discusses the requirements for sample collection and submission with an appropriate laboratory beforehand.

Diagnosis of viral infections

The three most common methods available for the diagnosis of viral infections are virus isolation, serology and direct detection of viral antigens by immunodiagnostic methods.

1. *Virus isolation*. Samples for isolation of viruses include swabs (e.g. conjunctival, nasal, nasopharyngeal etc.), body fluids (e.g. blood, semen, tracheal aspirates, etc.) and tissues. Samples often need to be obtained in the early stages of infection, and they should be collected into a special transport medium which contains antibiotics to prevent bacterial growth and proteins to stabilize the virus. The sample should be transported to the laboratory without delay, and may need to be kept at a low temperature (e.g. packed with ice).
2. *Serology*. A variety of serological tests is used to demonstrate serum antibodies. In many cases, the antibody titres present during the acute stage of the disease need to be compared with those in the convalescent stage 10 to 14 days later; a four-fold or greater increase in titre is generally considered significant.
3. *Detection of viral antigens and nucleic acids*. Viral antigens may be detected in blood, secretions, scrapings and tissues by a variety of immunodiagnostic techniques, such as immunofluorescence, immunoperoxidase staining, enzyme-linked immunosorbent assay (ELISA) and radioimmunoassay (RIA). Viral nucleic acids may be detected by techniques such as the polymerase chain reaction (PCR). These methods may provide a rapid diagnosis.

Diagnosis of bacterial infections

A number of techniques similar to those used for diagnosis of viral infections may be used. The most commonly used technique in bacterial infections is culture of body fluids (e.g. nasal swabs, pus, tracheal aspirates), faeces, tissues etc. As with viral infections, the collection of the correct type of sample, and correct handling and transport of the samples, are all essential to obtain accurate laboratory results.

19.3 EQUINE INFLUENZA

The virus

The virus is an orthomyxovirus of influenza A type. Two structural proteins (haemagglutinin, HA, and neuraminidase, ND) are the major antigenic determinants and form the basis of categorization into different subtypes. Two major antigenically distinct subtypes exist – A/equine/1 (H7N7) and A/equine/2 (H3N8). These viruses are considered to have originally been of avian origin. The H3N8 influenza viruses are currently the most common sub-types identified in horses worldwide.

Antigenic shift (major change in HA and NA antigenic structure – rare) and antigenic drift (minor change in antigenic structure – commoner) can occur, resulting in recurrent epizootics and the need to update commercial vaccines.

Epidemiology

A major and economically important cause of acute respiratory disease throughout the world – with the exception of a small number of island nations, including New Zealand and Iceland. In North America and some parts of Europe the virus is enzootic with local outbreaks occurring regularly.

Horses of all ages are susceptible, but infection is commonest in young (2–3 years) unvaccinated horses. Equine influenza has a low mortality rate but very high morbidity, which has a major economic effect due to disruption of equestrian activities. Infection may occur in vaccinated horses, although the severity of clinical disease and degree of viral shedding are reduced.

The extensive use of killed and subunit vaccines in Europe and North America has reduced the severity and morbidity of clinical disease. However, when new antigenic variants emerge, explosive outbreaks of disease can occur.

Large amounts of virus are aerosolized from affected horses due to the frequent cough. The incubation period is short (1–5 days) and virus is shed for up to 10 days.

Outbreaks are most common when large numbers of young susceptible horses are brought together at sales and shows, or for weaning and training.

Pathogenesis

Aerosolized virus is inhaled and deposits on the mucosa of the upper and lower respiratory tracts. The virus attaches to the epithelial cells and enters the cell cytoplasm where replication occurs. The epithelial lining of the entire respiratory tract is affected. Infected epithelial cells are damaged leading to inflammation, clumping of cilia and focal erosions. Mucociliary clearance mechanisms are impaired, and secondary bacterial infections are common, which can prolong the recovery period.

Clinical signs

- Cough (harsh, dry and hacking; sudden onset).
- Pyrexia (up to 41.7°C, 107°F).
- Depression.
- Lethargy.
- Inappetence/anorexia.
- Enlargement and tenderness of submandibular lymph nodes.
- Serous bilateral nasal discharge (becoming mucopurulent with secondary bacterial infections).

Other signs that may be observed include tachypnoea, tachycardia, congestion of conjunctivae, epiphora, limb oedema, muscle stiffness and weight-loss.

- The severity of the disease is related to the virulence of the virus strain and the immune status of the horse at the time of exposure. In uncomplicated cases, recovery occurs in about 10–14 days, but coughing may persist for several weeks.
- In some cases, secondary bacterial infection of the lower respiratory tract may give rise to bronchopneumonia with signs of dyspnoea, chest pain, reluctance to move (see Chapter 6).
- In a minority of cases, myocarditis occurs causing tachycardia, arrhythmia and severe exercise intolerance.
- Pregnant mares occasionally abort secondarily to pyrexia.
- In partially immune or vaccinated horses, the clinical signs are usually mild, or the infection may be subclinical, but they can still shed virus and serve as a source of infection to other horses. Performance horses may demonstrate only exercise intolerance.

Diagnosis

1. A presumptive diagnosis may be made on the basis of the clinical signs and rapid spread of disease, especially in unvaccinated horses; however, vaccinated horses may show few if any clinical signs, and laboratory analysis is required for diagnosis.
2. Haematology.
   - Anaemia, leucopenia and lymphopenia are seen early in the course of the infection (1–5 days).
   - Neutrophilia often occurs later as secondary bacterial infections arise.
   - Plasma fibrinogen and plasma viscosity may be elevated.
3. Virus isolation from nasopharyngeal swabs.
4. Serology – acute and convalescent serum samples (taken 10 to 14 days apart) demonstrate antibody rise (haemagglutinin inhibition and single radial haemolysis).
5. Rapid immunological tests to detect viral antigens/nucleic acid include enzyme and fluorescent antibody tests, and real-time PCR. An immunoassay developed to detect human influenza (e.g. Directigen Flu-A and Espline) may also be used. Real-time PCR is considered to be very sensitive and provides a diagnosis within hours.

Treatment

1. Complete rest for minimum of 3–4 weeks in clean, minimum-dust environment.
2. General nursing care and provision of palatable food.
3. Antibiotic (penicillin or trimethoprim/sulphonamide) treatment is necessary only if there is significant secondary bacterial infection. If bronchopneumonia is suspected, antibiotic selection should be based on culture of transtracheal aspirate.
4. Non-steroidal anti-inflammatory drugs (NSAIDs) such as phenylbutazone are helpful in horses with high fever, depression or muscle stiffness.
5. Immunostimulants, such as mycobacterial cell wall extracts, are reported to be beneficial.
6. Bronchodilators and mucolytics may be helpful in some cases.

Control of outbreak

1. Isolate infected horses as soon as possible (as soon as a temperature rise is identified).
2. Provide adequate ventilation and minimal dust conditions.

3. Avoid all contact between healthy and sick horses.
4. Cease exercise/training to minimize stress.
5. Maintain separate feeding, cleaning and grooming equipment, and personnel for sick horses.
6. Vaccinate healthy horses in face of the outbreak.

Prevention

1. Isolate new arrivals for 3 weeks.
2. Maintain adequate ventilation rates for all stabled horses, especially in barns.
3. Routine vaccination.
   - Current vaccines contain both influenza subtypes as either inactivated whole virus or subunit viral antigens.
   - Manufacturers' recommendations vary, but most advise two primary doses 3 to 6 weeks apart, followed by a booster dose 6 months later, a fourth dose after another 6 months and thence annually (or more frequently for high risk horses).

Since 2005, the Federation Equestre Internationale (FEI) requires all horses competing at their competitions to have been vaccinated in the previous six months. It is advisable to vaccinate pregnant mares during the last 6 to 4 weeks of pregnancy to ensure that their colostrum has protective levels of antibody. Foals born to seropositive mares usually have adequate passive immunity for 3 to 6 months.

19.4 THE EQUINE HERPESVIRUSES

The viruses

Nine herpesviruses (EHV) are known to infect equids. EHV1 infection is associated with respiratory disease, abortion, neonatal mortality, and neurological disease, whereas EHV4 is primarily associated with respiratory disease. EHV3 causes the sexually transmitted disease equine coital exanthema, and EHV2 and 5 have been associated with keratoconjunctivitis.

Equine herpesvirus 1 (rhinopneumonitis)

EHV1 infection is associated with respiratory disease, abortion, neonatal disease and neurological disease. The virus has a worldwide distribution. Numerous strains of EHV1 exist, and strain variations are likely to be associated with the different forms of clinical disease.

Pathogenesis and epidemiology

Necrotizing vasculitis with ensuing hypoxia and malnutrition of tissues, including fetal tissues, placenta and central nervous system, is the basic underlying pathophysiological mechanism responsible for the clinical manifestations. Infection occurs by inhalation of virus in aerosols. The virus replicates in the epithelium of the upper and lower respiratory tracts and associated lymphoid tissue (resulting in lymphadenopathy especially in young horses). EHV1 appears to have evolved a number of mechanisms that allow it to evade the host's immune defences. Systemic spread by a cell-associated viraemia can occur. Subsequent infection of endothelial cells may lead to vaculitis in the pulmonary, CNS and placental vasculature. A genetic mutation has been identified in the DNA polymerase of some EHV1 strains that appears to predispose to neurological disease.

- The incubation period is 2 to 10 days.
- Respiratory disease is commonest in young horses (up to 3 years). Older horses usually show mild or subclinical respiratory disease.

Immunity following infection or vaccination is short-lived, and horses may become reinfected on numerous occasions throughout their lives. There is good evidence to suggest that initial infection often occurs within weeks after birth and that unweaned foals are infected by asymptomatic shedding of virus by mares.

Consistent with many herpesviruses, latent infections by EHV1 occur, which may undergo recrudescence under conditions of stress or following administration of immuno-suppresive drugs, with the potential to cause disease or act as a source of infection to incontact horses. Latent virus may persist in lymphoid tissue and the trigeminal ganglion. A recent study in Kentucky identified latent EHV1 infection in the submandibular lymph nodes of 54% of broodmares.

Clinical signs

Respiratory disease

- Pyrexia (up to 41.1°C, 106°F) (may be biphasic).
- Serous nasal discharge (which becomes mucopurulent later).
- Coughing (variable; mild or absent).
- Depression.
- Enlargement of submandibular lymph nodes.

Uncomplicated cases recover in 8 to 10 days. Secondary bacterial infections may prolong the course of the disease.

Older horses show milder or subclinical disease – this may be associated with exercise intolerance or low-grade lower airway inflammation (see Chapter 6).

Abortion (see Chapter 14). Abortion may occur due to infection of the foetus and placenta after viraemia, or due to vasculitis in the maternal endometrium. It usually occurs in late gestation (7 to 11 months). The initial respiratory infection is often subclinical. Abortions due to EHV1 often occur as 'red bag' or premature placental separation deliveries.

Abortions may be sporadic or multiple (abortion 'storms').

Neonatal disease (see Chapter 20). Foals infected in utero may be affected by severe respiratory disease and are born ill and weak, dying within a few days. Alternatively, foals may appear normal at birth but develop severe illness after a few days. Pathologically, affected foals have necrotizing bronchopneumonia.

Neurological disease (see Chapter 11). Neurological disease is associated with vasculitis of spinal vessels and ischaemia of the cord. It may occur in association with or in the absence of respiratory disease or abortion. Onset of neurological signs may occur up to 8 days after an initial episode of fever.

In many parts of the world, EHV1 neurological disease is considered to be the most common infectious cause of neurological disease in horses. Any age group may be affected. Single, isolated cases or outbreaks may occur.

- Clinical signs vary in severity, but usually have a sudden, rapid onset, with maximal severity occurring within 48 hours:
  - Ataxia and paresis of hind limbs.
  - May progress to paraplegia or quadriplegia and recumbency.
  - Depression.
  - Hyperaesthesia.
  - Decreased tail and anal tone.
  - Urinary incontinence.
  - Occasionally a head tilt.
  - Fever.
  - Inappetence.
  - Limb oedema.

Non-recumbent horses usually recover, but full neurological function may not return for several months.

A genetic mutation affecting DNA polymerase is associated with the development of neurological disease; however, not all horses infected with such strains of the virus develop neurological disease.

Diagnosis

Respiratory disease

- Virus isolation from nasopharyngeal swab or citrated/heparinized blood.
- Serology.
- Demonstration of viral antigens in nasal swabs or tracheal aspirates (PCR).
- Haematology – non-specific changes.

Abortion

- Virus isolation from foetal tissues/placenta.
- Histopathology of foetal tissues/placenta (see intranuclear inclusions).
- Demonstration of viral antigens in foetal tissues/placenta (immunofluorescence, etc.).

Neonatal disease

- Virus isolation.
- Histopathology and immunofluorescence of post-mortem tissues.

Neurological disease

- History and clinical signs.
- Serology.
- Post-mortem examination and histopathology.

Treatment

Respiratory disease

- Rest for 3–4 weeks in clean, minimum dust environment.
- Anti-inflammatory drugs (e.g. non-steroidal anti-inflammatory drugs).
- Antibiotics as necessary to control secondary bacterial infections.

Abortion

- None.

Neonatal disease

- Antibiotics.
- General nursing and respiratory care.
- Prognosis is very poor.

Neurological disease

- General nursing care.
- Treatment of recumbent horses is difficult, and many cases require euthanasia due to complications of myositis, skin damage, pneumonia, etc. In general, horses that become recumbent for longer than 24 hours have a very poor prognosis. Some horses with severe neurological signs can recover completely whilst others may have residual defects.
  - Urinary catheterization.
  - Antiviral medications (e.g. acyclovir, valocyclovir) may be helpful.

Control of outbreaks and prevention

Aims:

- Minimize exposure of horses to virus.
- Maximize immune status.
- Decrease likelihood of latent virus recrudescence.

1. Isolate incoming horses for 2–3 weeks.
2. Age segregation of horses.
3. Pregnant mares should be separated from young stock and ideally kept in small groups according to the gestational stage.
4. Horses showing clinical respiratory disease should be isolated.
5. Personnel handling infected horses should be isolated from healthy stock.
6. Bedding of infected horses should be burned.
7. Contaminated stable equipment/clothing should be disinfected.
8. Aborting mares should be kept and managed in isolation. The foetus and membranes are potential sources of infection and must be handled and disposed of carefully.
9. In cases of abortion, all mares due to foal in the same season should remain on the farm until they have foaled. Horses that leave the farm should not be allowed contact with pregnant mares.
10. In cases of neurological disease, infected horses should be isolated, and all horses on the premises should be confined until 3 weeks after the identification of the last new case.
11. Vaccination. EHV1 and 4 vaccines are available but provide only partial and short-lived immunity. However, repeated vaccination can reduce the severity of respiratory disease and reduce the incidence of abortions. Two initial doses given several weeks apart are followed by regular boosters at intervals varying between 3 months and 1 year (depending on specific vaccine). Unlike the recommendation for influenza, it is usually inappropriate to administer EHV1 and 4 vaccines to healthy, at-risk animals in face of an EHV outbreak.

Equine herpesvirus 4 (rhinopneumonitis)

EHV4 causes respiratory disease indistinguishable from that caused by EHV1.

- The pathogenesis, clinical signs, diagnosis, treatment, control and prevention are similar to those described for the respiratory form of EHV1.
- EHV4 has been linked with sporadic abortions but has not been associated with abortion storms. It is not generally linked with neonatal disease or neurological disease.

Equine herpesvirus 3 (coital vesicular exanthema) (see Chapter 14)

This is a venereal infection causing small pustular vesicles on the vulva and penis, which subsequently ulcerate. The lesions persist 2 to 3 weeks.

The incubation period is 2 to 10 days.

Fertility is unaffected, but severe lesions on the penis may inhibit the stallion from covering mares.

Treatment consists of sexual rest and application of antiviral and/or antibacterial ointment to affected areas.

19.5 EQUINE VIRAL ARTERITIS (EVA)

The virus

Equine viral arteritis (EVA) is caused by equine arteritis virus (EAV), an RNA virus of the Togavirus family. Only one serotype is recognized, but there is marked variation in pathogenicity between different strains. EVA derives its name from the widespread vasculitis involving small blood vessels (especially arterioles) that is characteristic of the disease.

Epidemiology

EAV is widely distributed throughout the world, having been reported in North and South America, Europe, Africa, Asia, Australia and New Zealand. However, reports of clinically apparent EVA are relatively few, which is explained by a high prevalence of subclinical infection and confusion of the clinical disease with other viral infections such as

influenza and rhinopneumonitis. Serological surveys indicate that the prevalence of infection varies widely between countries and between different breeds in the same country. Higher rates of infection are frequently found in Standardbreds and various Warmblood breeds.

Transmission of the virus occurs via the respiratory tract (aerosolized particles) and venereally. EAV is shed in nasal secretions for up to 7 to 10 days. Stallions can transmit the virus venereally in the acute stage of the disease, and as long-term carriers. EVA-infected semen is an important potential means of spread of the virus.

The incubation period is 3 to 14 days.

Pathogenesis

Although only one serotype of the virus has been isolated so far, there appear to be differences in antigenicity and pathogenicity among different isolates of the virus. The virus multiplies within alveolar macrophages and then passes to the bronchial lymph nodes. This is followed by viraemia and widespread distribution to many tissues. Vasculitis involving the small arteries occurs initially in the lungs and then in other tissues.

Clinical signs

Infection may result in clinical disease or be subclinical. The clinical signs depend on the virus strain, viral dose, route of exposure, age and physical condition of the horse.

The clinical signs vary widely, but tend to be more severe in very young and old horses, or those in a debilitated condition.

The incubation period is usually 3–14 days.

- Pyrexia (up to 41 °C, 106 °F) for 2–10 days.
- Depression and anorexia.
- Limb oedema (especially hind limbs).
- Stiffness.
- Nasal or lacrimal discharge.
- Conjunctivitis or rhinitis.
- Periorbital/supraorbital oedema.
- Ventral abdominal, scrotal, preputial or mammary oedema.
- Vesicular-erosive stomatitis and hypersalivation.
- Urticarial-type skin rash (especially over the head, neck and pectoral regions).
- Abortion (any stage of gestation) – foetus usually partly autolysed.
- Coughing, respiratory distress.
- Diarrhoea.
- Ataxia.

The duration of clinical signs can range from several days to up to 2 weeks. Most infected horses make a full recovery, and the mortality rate is low. Secondary bacterial infections are uncommon. Fatal infections are most likely in neonatal foals congenitally infected by the virus and in foals a few weeks to months of age that succumb from a fulminating interstitial viral pneumonia.

Abortion can be a sequel to clinical or asymptomatic infection, and many strains of the virus are believed to be abortigenic. Abortion rates in unprotected mares can range from less than 10% to over 70%. Abortion can occur at any stage of gestation. Placental fluids, the aborted foetus or congenitally affected newborn foals are all highly productive sources of virus.

Carrier state

A carrier state is established in 30–60% of infected stallions. The duration of the carrier state varies from several weeks to a lifetime. Carrier stallions shed the virus constantly in the semen. There is no effect on fertility. Mares do not become carriers of EVA.

Diagnosis

- Clinical signs – EVA cannot be diagnosed solely on the clinical signs, so laboratory confirmation is essential.
- Haematology – leucopenia and lymphopenia.
- Virus isolation or identification of viral antigens or PCR. To optimize the chances of virus detection, samples should be obtained as early as possible after the onset of fever.
  - nasopharyngeal swabs.
  - conjunctival swabs.
  - citrated or EDTA blood.
  - placenta/foetal fluids/foetal lung and lymphoreticular tissue.
- Serology (acute and convalescent samples).

Potential carrier stallions are screened by serology (if unvaccinated) and virus isolation or PCR of semen.

Treatment

No specific antiviral treatments are currently available.

- Rest.
- Symptomatic treatments, eg non-steroidal anti-inflammatory drugs.

Prevention and control

Two vaccines are available (a modified live vaccine and an inactivated adjuvanted vaccine).The vaccines are safe and effective in stallions and non-pregnant mares. Prevention and control depend on management practices (as described for influenza and EHV1) and selective use of the vaccine, including vaccination of the at-risk stallion population. Stallions should be vaccinated annually at least 28 days before the onset of the next breeding season. Carrier stallions should be kept in isolation and bred only to seropositive mares. EAV is readily inactivated by heat, sunlight and many commonly used disinfectants. However, infectivity of the virus can be maintained for long periods of time below freezing temperature. Measures should be taken to prevent the spread of EAV in fresh or frozen semen used for AI.

19.6 EQUINE ADENOVIRUS

Equine adenovirus is a DNA virus which is widely distributed in the horse population worldwide. Most infections are probably subclinical. It can persist in the upper respiratory tract of adult horses in a carrier state.

- Infection of foals may result in mild respiratory disease characterized by nasal discharge, coughing, conjunctivitis, pyrexia and occasionally diarrhoea.
- Arabian foals affected by combined immunodeficiency syndrome (CID) (see Chapter 20) may develop severe, fatal, intersitial pneumonia (usually in conjunction with other pathogens).

19.7 EQUINE PICORNAVIRUSES (RHINITIS VIRUSES)

The equine picornaviruses include equine rhinitis A and B viruses (ERAV and ERBV) (previously classified as equine rhinoviruses 1, 2 and 3) and equine rhinitis B virus 3 (previously called acid-stable picornavirus).

The clinical significance of infection by these agents is unclear. ERAV has been associated with mild acute respiratory disease (rhinitis, coughing, enlarged submandibular lymph nodes, pyrexia), but most infections by ERBV appear to be subclinical.

ERV infections may be diagnosed by isolation of virus in tissue culture, PCR and demonstration of viral seroconversion using complement fixation or virus neutralization tests.

19.8 EQUINE INFECTIOUS ANAEMIA (EIA)

The virus

EIA virus (EIAV) is an equine-specific lentivirus of the retrovirus family, related to HIV-1.

The virus contains reverse transcriptase, an enzyme that can convert the viral RNA genome into DNA; this complementary DNA can insert into the host DNA where it persists, protected from the host's immune defences. Replication of EIAV occurs primarily in cells of the monocyte/macrophage lineage although some strains can replicate in endothelial cells.

Epidemiology

The virus has been identified on all continents. In Europe, it is most prevalent in the northern and central regions, and in North America it is most prevalent in the Gulf Coast and northern wooded regions of Canada.

EIAV is transmitted between horses by transfer of blood or blood products. Biting flies, especially tabanids, are the usual means of transfer, but it can also occur iatrogenically via contaminated blood, needles, surgical instruments, etc. Transplacental and colostral transmission can also occur. A recent outbreak in Ireland was linked to the use of an untested batch of equine plasma.

Pathogenesis

EIAV replicates in macrophages in liver, spleen, lymph nodes, etc. High numbers of virus are released into the circulation, which causes pyrexia. Cell-mediated and humoral immune responses clear the virus from the blood, but the virus persists within tissue macrophages.

Rapid antigenic variation allows the recurrent replication of large amounts of virus, which cause recurrent bouts of disease. Eventually, the host immune response is stimulated sufficiently to prevent bouts of viraemia.

Clinical signs

The clinical features are highly variable. Acute, subacute, chronic and subclinical syndromes are recognized. The severity of clinical signs varies according to the virulence of the strain of virus, the dose of virus, and the host response. Classical cases progress through acute, chronic and carrier phases.

The *acute syndrome* is characterized by:

- Pyrexia (up to 41.1°C, 106°F).
- Thrombocytopenia and petechiation of mucous membranes.
- Depression.
- Anorexia.
- Anaemia.
- Epistaxis.
- Oedema.

Most horses recover from the viraemia and appear normal for several months (often 12 months or longer), and then experience recurrent episodes of disease, termed the chronic phase.

The chronic phase is characterized by:

- Weight loss.
- Anaemia.
- Depression.
- Oedema.
- Death.

The frequency and severity of clinical bouts of disease decrease with time, and most horses eventually cease to show any clinical signs but remain infected with EAV and remain a reservoir for transmission. This is known as the inapparent carrier stage.

Not all infected horses demonstrate these classical signs. Some infected horses have *subclinical infection* with no overt clinical signs, but they can act as a source of infection to others. Others may develop severe peracute disease with persistent fever, plummeting platelet counts, and eventually death.

Diagnosis

1. Clinical features.
2. Haematology.
   - Thrombocytopenia.
   - Anaemia.
3. Agar gel immunodiffusion test (Coggin's test) and ELISA test.
4. Virus isolation is difficult.

Control

Carrier horses are identified and eliminated using a serological test such as the Coggin's test. Federal and State control measures operate in the USA. No vaccines are currently available.

19.9 AFRICAN HORSE SICKNESS (AHS)

The virus

African horse sickness viruses (AHSV) are RNA reoviruses of which there are nine serotypes. AHSV is morphologically similar to other orbiviruses such as bluetongue virus of ruminants and equine encephalosis virus.

Epidemiology

AHS is endemic in eastern and central Africa, but has spread elsewhere on a number of occasions, including Pakistan, the Middle East and Spain.

AHS is arthropod-borne and may spread via infected horses or by vector movement. The main vectors are *Culicoides* species of midges. Spread by contaminated needles or surgical instruments is also possible. The disease has a seasonal occurrence, and its prevalence is influenced by climate and other factors affecting the breeding of *Culicoides* spp. Zebras and donkeys can act as reservoirs of the virus (infection is often sub-clinical in these species).

Clinical signs

Four syndromes are recognized:

1. Peracute or pulmonary form.
   - Incubation period 3–5 days.
   - Fever (up to 40.5°C, 105°F).
   - Congestion of mucous membranes.
   - Dyspnoea and tachypnoea (respiratory rate may exceed 50 breaths/minute).
   - Cough.
   - Sweating.
   - Frothy blood-tinged nasal discharge (terminally).
   - High mortality rate (95%) with death in 1–3 days.
2. Subacute or cardiac form.
   - Incubation period 5–7 days.
   - Fever (up to 40.5°C, 105°F).
   - Congestion of mucous membranes.
   - Oedematous swellings of neck, chest, lumbar and pelvic areas.
   - Oedema of supraorbital fossa, eyelids, intermandibular space.
   - Petechial haemorrhages on tongue and conjunctivae.
   - Colic.
   - Mortality rate is 50% with death in 4–8 days.

3. Acute or mixed form (most common clinical form).
   - Incubation period 5–7 days.
   - The initial clinical signs are similar to mild pulmonary form but then followed by oedematous swellings and effusions and death due to cardiac failure.
   - Mortality rate 50–95% with death in 3–6 days.
4. Horse sickness fever form.
   - Occurs in partially immune horses and donkeys.
   - Incubation period 5–9 days.
   - Low-grade fever (up to 39.5°C, 103°F).
   - Conjunctival congestion.
   - Mild depression and inappetence.
   - Recover in 5–8 days.

Diagnosis

- Clinical signs.
- Haematology – leucopenia, thrombocytopenia, elevated haematocrit.
- Serology.
- Virus isolation, antigen capture ELISA, PCR.
- Necropsy.

Treatment

There is no specific treatment.

Control

Vaccination is used in endemic areas. Vigilance necessary given the possibility the disease may well appear in currently non-endemic areas, reflecting climate change and intermediate host movement between regions/continents.

19.10 VESICULAR STOMATITIS

The virus

The disease is caused by vesicular stomatitis virus (VSV) which is a lyssavirus of the rhabdovirus family. Two main serotypes exist. VSV is a potential zoonosis, so gloves should be worn when examining potential cases.

Epidemiology

- Endemic in central America and southern USA.
- Affects horses and other livestock.
- High morbidity and low mortality.
- Virus-infected insects and plants are believed to be important means of transmission.

Subclinical infections can occur. Horses and cattle less than one year of age may be less susceptible to clinical disease.

Clinical signs

Although the disease starts with the formation of vesicles in the oral cavity and on the lips, the condition usually has progressed to the ulcerative phase by the time it is recognized by the owner.

- May be an initial fever.
- Coalescing ulcers along lips, gums and tongue.
- May be ulcers on the turbinates and nasopharynx.
- Ptyalism and reluctance to eat.
- Epistaxis.
- Dysphagia.
- Ulceration of coronary band.
- Occasionally lesions on the udder or genitalia.
- Oral ulcers heal in 1–2 weeks.
- Coronary band lesions may result in hoofwall defects or laminitis.

Diagnosis

- Clinical signs (it is not possible to definitively diagnose VSV infection on the basis of clinical signs alone).
- Virus isolation (saliva, vesicular fluid, epithelium).
- Serology (competitive ELISA, capture ELISA, CFT, VNT).

Treatment

There is no specific treatment.

- Symptomatic – palatable soft food.
- Antibiotics to prevent secondary infections.

Prevention

There are currently no available vaccines.

19.11 HENDRA VIRUS

An outbreak of acute respiratory syndrome occurred in a training yard in Queensland, Australia in 1994. Eleven of 24 horses in the yard died as did one human (the trainer). Another 10 horses were affected on five other neighbouring premises, of which three died. The outbreak was associated with a novel virus, Hendra virus, then referred to as equine morbillivirus. The incubation period was 8 to 16 days. Clinical signs included pyrexia (up to 41°C, 105.8°F), depression, shallow respirations, cyanotic mucous membranes and a frothy nasal discharge. Postmortem examinations revealed severe pulmonary oedema and congestion. Hendra virus has a tropism for vascular endothelium.

Since the intial outbreak, there have been another eight outbreaks reported, in five of which only a single horse was affected. Flying foxes, fruit bats of the genus *Pteropus*, are the natural reservoir host of Hendra virus. How the virus infects horses is currently uncertain, although horse-to-horse transmission has been demonstrated.

19.12 WEST NILE VIRUS

West Nile virus (WNV) encephalitis is one of the leading causes of equine neurological disease in the United States. Since the introduction of WNV into the United States in 1999, over 25 000 clinical cases of disease have been confirmed in horses.

The virus

WNV is classified as a Flavivirus within the family Flaviviridae. It is an enveloped, single-stranded positive-sense RNA virus, approximately 50 nm in size. There are two distinct phylogenetic lineages of WNV. Lineage 1 viruses, which were introduced into North America, are generally considered more pathogenic, while lineage 2 viruses are considered less pathogenic.

Epidemiology

- WNV was first discovered in 1937 in the West Nile province of Uganda. The virus was introduced to North America in 1999, when a single point introduction of WNV occurred in New York City. Since then, the virus has spread to affect almost the entire western hemisphere, as far north as Canada and as far south as Argentina.
- West Nile virus causes a seasonal disease, with case occurrence corresponding with peak mosquito vector activity. In northern, temperate climates, peak activity occurs in the summer months (July and August). In tropical and subtropical climates, disease activity is high year-round, although it may fluctuate slightly depending on rainfall.

- WNV is maintained in a bird-mosquito-bird cycle. In North America, over 60 species of mosquito have been found to be capable of transmitting WNV, with Culex species being the main vector among birds.
- It was originally thought that Corvidae (i.e. crows, blue jays, ravens) served as the major reservoir for the virus. However, while these species of birds develop high viraemias, they quickly succumb to clinical disease. Other bird species, including the American robin, the Northern cardinal, the common grackle, and the house finch, may serve as the main reservoir hosts of WNV.

Pathogenesis

- After the bite of an infected mosquito, WNV is inoculated peripherally into the skin and muscle. Dendritic cells take up the virus and transport it to the regional lymph nodes, where an immune response is initiated. If the host is unable to neutralize the virus, it may gain access to the central nervous system through breach of the blood–brain barrier.
- Neuronal infection by WNV leads to host cell damage, both directly through the actions of the virus, and indirectly through the host immune response.
- WNV causes a polioencephalomyelitis (inflammation of the grey matter) mainly involving the midbrain, hindbrain, and spinal cord. This is characterized grossly by an increasing number of lesions progressing from the diencephalon through the hindbrain and down to the spinal cord. Spinal cord lesions become progressively worse caudally. Congestion of the meninges and haemorrhagic foci may be seen.

Clinical signs

- The majority of WNV infections are subclinical – only about 10% of horses naturally exposed to WNV actually develop clinical disease.
- The incubation period is 9–11 days.
- The initial signs include fever (38.3–39.4°C, 100.9–102.9°F), anorexia, and depression.
- The onset of neurological disease is usually abrupt, and there are changes in behaviour or mentation with an insidious onset of motor deficits.
- Spinal cord abnormalities include a stiff stilted gait, ataxia (involving two or more limbs, symmetric or asymmetric), flaccid paralysis, paresis, and recumbency.
- Muscle fasciculations (most notable around the muzzle but can involve the entire body) are often noted.
- Cranial nerve abnormalities include weakness of the tongue, muzzle deviation, head tilt and difficulty swallowing.
- Changes in mentation include a change in sensorium defined as 'a change in horse's normal habits, personality, attitude, reaction to environment' and hypaeresthesia.
- In addition, changes in behaviour including severe aggression, somnolence, and coma may be seen.
- Approximately 30% of horses with clinical signs of disease die spontaneously or are humanely euthanized (this number increases to 100% if the horse is recumbent). The remaining 70% of horses with clinical disease recover within 3–7 days. However, approximately 30% of the horses that recover recrudesce within two weeks. Of the horses that recover, 10% retain long-term complications, including weakness, ataxia, and fatigue.

Diagnosis

- Clinical signs.
- Haematology – normal or lymphopenia.
- Serum biochemistry – elevated muscle enzymes secondary to trauma, and hyponatremia.
- CSF analysis – elevated mononuclear cell population >7 cells/µg, protein concentration >70 mg/dL, and mild xanthochromia.
- IgM capture ELISA (serum and CSF) – high levels of IgM antibodies are quickly formed post-exposure to WNV by the horse and last for approximately 4–6 weeks.
- Plaque reduction neutralization test (PRNT).
- Post-mortem examination and testing for the virus in brain and spinal cord – virus isolation, PCR to detect viral antigen, and immunohistochemistry to detect viral antigens.

Treatment

- There is no specific treatment.
- Treatment should focus on providing supportive care and includes administration of flunixin meglumine, corticosteroids.

Prevention

Three vaccine types have been produced – inactivated whole virion, canarypox modified live, and modified live flavivirus chimera.

19.13 EQUINE ENCEPHALOSIS

Equine encephalosis (EE) is usually a mild or subclinical orbivirus infection of horses. Equine encephalosis virus (EEV) is transmitted by species of *Culicoides*, which are endemic to the temperate regions of Africa, and as a result, the epidemiology has much in common with African horse sickness (AHS).

The incubation period of the disease is between three and five days. Most (>90%) horses show either no obvious signs of infection or develop only very mild clinical signs. Initially most affected horses show either a slightly elevated rectal temperature (39°C, 102.2°F) for one or two days or a high fever (40 to 41°C, 104 to 105.8°F) for between one and five days. Fever may be accompanied by varying degrees of listlessness and inappetance. Occasionally, varying degrees of swelling of the eyelids and supraorbital fossae may be observed. Central nervous system involvement, respiratory distress and acute heart failure have been observed in a few cases.

Infection with EEV is usually subclinical, and the majority of infections are confirmed by seroconversion. In horses showing a febrile reaction or clinical signs a diagnosis can be made by isolation of the virus from blood or from tissues (spleen, thymus, liver, lung, and brain).

As infection with EE is usually subclinical no vaccine has been developed, and no control measures are usually implemented in endemic areas. The stabling of horses from an hour or two before the sun has set until an hour or two after it has risen the next day (the period when *Culicoides* midges are particularly active) is a useful control method.

19.14 ROTAVIRUS

The virus

Rotavirus is a genus within the family Reoviridae. The viruses are about 70–80 nm in size and on electron microscopy look like wheels (*rota* is derived from the Latin word 'wheel') with short spokes radiating from a wide central hub. Rotaviruses are all double-stranded ribonucleic acid (RNA), non-enveloped viruses. Rotavirus is subdivided into several groups (A through G). Only one group affects the equine species (Group A).

Epidemiology

Antibody prevalence studies indicate that many adult horses have been exposed to the virus. However equine rotavirus clinically only

affects foals. These studies were conducted in concentrated horse breeding regions, and therefore the true seroprevalence in adult horses in other regions of the world may be less.

The mode of transmission of rotavirus infection is assumed to be the faecal–oral route with an incubation period of 1–2 days. Transmission occurs directly by animal to animal and indirectly through personnel and fomites. Infected foals shed virus in high concentration at the onset of disease and continue for an average of 3 days after return of normal faeces. It is possible that healthy adult horses shed the virus chronically in low numbers. Rotavirus virus can remain viable for up to 9 months at room temperature, so inappropriate cleaning and disinfecting of the premises may permit a build up of the infective dose in the environment.

Pathogenesis

Rotavirus replicates in mature villous epithelial cells of the small intestine. This results in blunting of the villi and subsequent villous atrophy. The loss of the villi results in the loss of important disaccharidases, especially lactase, which is responsible for the digestion of disaccharides (lactose). Lactose therefore remains in the intestinal tract and is fermented in the large colon to produce volatile fatty acids, which results in a hyperosmotic solution leading to acute diarrhoea. Rotavirus-induced lactose deficiency may last up to 10–14 days. Subsequent to the villous atrophy, malabsorption and maldigestion of nutrients also potentiates the diarrhoea. Intestinal crypt cells are not affected and therefore can replicate and eventually replace the villi brush border destroyed by the virus resulting in a self-limiting disease. Chronic diarrhea is not typical of rotavirus.

Clinical signs

- Clinical signs of rotaviral diarrhoea vary from mild diarrhoea to severe watery diarrhoea and dehydration.
- Any age foal can be affected but younger foals are usually more severely affected.
- Fever may or may not be present.
- Marked bloating may be noted with ileus resulting in gastric reflux.
- Colic signs can be mild to severe depending on the amount of colonic tympany (secondary to fermentation of disaccharides in the hindgut). Colic signs can mimic a surgical lesion; therefore appropriate diagnostics (abdominal ultrasound, foecal rotavirus testing etc.) should be pursued before an exploratory celiotomy is considered.
- Common electrolyte imbalances include hyponatraemia, hypochloraemia, hypokalaemia and metabolic acidosis. Some clinicians have suggested an association between gastroduodenal ulcer syndrome/stenosis and rotavirus infection although the relevance of this is uncertain at present.
- Mortality is relatively low in rotavirus infections. Morbidity however can approach 100% in outbreak situations. Foals less than 2 weeks of age are more at risk to die secondary to metabolic derangements and dehydration. The infection produces little inflammatory response of the surrounding tissue; therefore there are no pathognomonic lesions noted on histopathology.

Diagnosis

Although rotavirus is the most common cause of severe diarrhoea in foals, there are no pathognomonic clinical manifestations. Diagnosis therefore requires laboratory confirmation.

- Electron microscopy is not routinely available and has almost been completely replaced with an enzyme-linked immunosorbent assay (ELISA).
- Latex agglutination kits have been produced to deliver rapid results within minutes of testing.

These tests have a high degree of specificity but less sensitivity than the ELISA. It has been recommended by some authors that a minimum of three negative tests are required to be confident that a foal does not have rotavirus.

Treatment

The cornerstone of therapy for rotavirus-induced diarrhoea is replacement of fluids and electrolytes. Isotonic fluid therapy to correct electrolyte imbalances is especially important in young foals and those that are significantly dehydrated. (Fluid therapy guidelines are covered in Chapter 26.)

Oral fluids and electrolytes may be sufficient in mild cases.

Prevention

- A vaccine containing an inactivated strain of equine rotavirus is available for administration to pregnant mares in endemic areas to aid in the prevention of diarrhoea in their foals. Foal vaccination is not indicated because currently there are no data to suggest that vaccination of the new born foal with a rotavirus inactivated vaccine elicits an appropriate immune response.
- If a farm is experiencing an outbreak of rotaviral diarrhoea in their foaling units, then mares should be allowed to foal elsewhere (e.g. in outside paddocks).
- Rotavirus is a non-enveloped virus that is naturally very resistant to disinfectants. The phenol disinfectants however are effective. Oxidizing agents such as Virkon (1% solution) are also likely to be effective as they have been shown to be active against bovine rotavirus. Bleach is not considered to be efficacious against rotavirus.
- Pressure washing should not be used in a barn or stable with potential rotavirus infection because of the risk of aerosolization of pathogens into the rafters.
- Improper stall cleaning techniques can result in rotavirus surviving for months in the environment. Therefore manure and bedding from stalls of affected foals should be considered infectious and not be spread out on the pastures. The bedding should be disposed of away from horses or composted.

Rotaviruses are excreted from diarrhoeic foals in large quantities (10^{11} particles/g) which can easily contaminate not only the environment but the hands of personnel. Hand hygiene (wearing gloves and washing hands) is critical for disease prevention.

19.15 OTHER EQUINE VIRAL INFECTIONS

- Equine viral encephalitides (Chapter 11).
- Japanese B encephalitis (Chapter 11).
- Borna disease (Chapter 11).
- Rabies (Chapter 11).

19.16 SALMONELLOSIS

The Gram-negative bacteria of the species *Salmonella enterica* are facultative intracellular anaerobes. There are six sub-species and over 2000 serovars. Infection with *Salmonella* spp. is not uncommon in the horse and is associated with a variety of clinical scenarios from asymptomatic states to peracute enterocolitis. Equine *Salmonella* spp. infections have zoonotic importance and may also be transmitted to or acquired from other animal species. Although any equid may develop clinical salmonellosis, foals are notably more susceptible, and well recognized risk factors in adult horses include recent general anaesthesia/surgery, gastrointestinal disease, antimicrobial therapy,

physical exhaustion, dietary change and transportation. Clinically normal horses may transiently shed *Salmonella* organisms, and a high proportion of horses with abdominal diseases shed the organism. With these risk factors often pertinent in hospitalized equidae, there are particular biosecurity implications for management of suspect salmonellosis cases whilst they are inpatients.

Aetiology

No *Salmonella* spp. are specific or adapted to the horse, and many different serotypes are sporadically isolated from equine specimens. The serotype most commonly isolated from clinical cases of salmonellosis is *S. typhimurium*, with *S. agona, S. enteritidis, S. krefeld, S. saintpaul, S. newport, S. infantis* and *S. anatum* also frequently isolated. The initial source of infection in individual horses or outbreaks is frequently difficult to identify.

Pathogenesis

The development of equine salmonellosis involves the interplay of a number of factors, including the degree of bacterial exposure, the virulence of the *Salmonella* organisms and the susceptibility of the host. Clinical disease generally requires ingestion of large numbers of *Salmonella* spp. organisms, followed by invasion of mucosa of the ileum, caecum or ventral colon. In susceptible horses there ensues a severe fibrinous ileitis/typhilitis/colitis which constitutes a protein-losing enteropathy (see Figure 19.1). There is reactive mesenteric lymphadenopathy, and if this immunological response is overwhelmed, bacteraemia occurs. In addition, there is endotoxaemia following absorption of intestinal bacterial endotoxins across compromised intestinal mucosa. Horses with impaction colic may be particularly at risk of developing salmonellosis.

Clinical signs

Signs range in severity from asymptomatic carriers to peracute death.

1. Carriers/shedders.
   - Usually carry/shed non-pathogenic *Salmonella* spp.
   - Generally asymptomatic.
   - 1–5% horses are active faecal shedders; usually transient but may be protracted excretion of *S. typhimurium* after acute colitis.
   - Up to 20% of all horses are silent (nonexcreting) carriers.
2. Mild infections.
   - Fever.
   - Anorexia.
   - Dullness.
   - Mild (cowpat) diarrhoea.
   - Self-limiting within 2–4 days.
3. Acute colitis.
   - Clinically indistinguishable from other acute colitides (see Chapter 3).
   - Early signs of dullness, anorexia and fever for 1–2 days.
   - Followed by moderately severe colic for next 1–2 days.
   - Diarrhoea – may be transiently mild progressing to profuse, watery (may be haemorrhagic in foals).
   - Cardiovascular compromise/clinically apparent endotoxaemia.
   - Peripheral oedema.
   - Weight loss.
4. Bacteraemia/septicaemia (especially foals)
   - Fever.
   - Concurrent diarrhoea.
   - Pneumonia.
   - Meningitis.
   - Septic arthritis and lameness (especially foals).
   - Sudden death (foals).

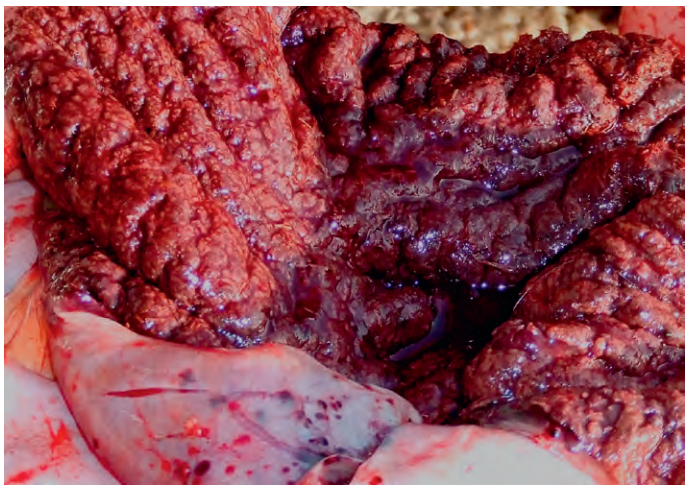


Figure 19.1 Post-mortem appearance of severe colitis in a horse affected by salmonellosis. The colonic mucosa is inflamed and severely congested.

Diagnosis

- Acute colitis associated with neutropenia, hypoproteinaemia and acid–base/electrolyte imbalance but not specific for salmonellosis.
- Isolation of *Salmonella* spp. organisms: culture in specific media, e.g. tetrathionate broth, selenite broth, brilliant green agar; suspect *Salmonella* spp. colonies tested by standard biochemical techniques, including H_2S production, urease activity etc.; serotype determined by slide agglutination at specialist reference laboratory. All confirmed isolates should be further classified by means of antimicrobial sensitivity testing and phage typing.

Faeces

- Small numbers in carriers/mild infections; culture of multiple sequential samples may be necessary.
- Poor isolation from rectal swab, so submit a large sample – minimum of 10 g of faecal material should be submitted. Salmonella organisms are more consistently shed in formed stool rather than diarrhoeic faeces.
- It may take 3–4 days to obtain a definitive culture result.

Tissues. Use rectal biopsy or intestinal mucosa, mesenteric lymph node, liver, spleen.

PCR tests are available for the detection of *Salmonella* spp. DNA. This can be applied to faeces or environmental samples. PCR techniques are extremely sensitive and may be overly sensitive for routine clinical application.

Treatment

Treatment is primarily supportive:

- IV fluids, electrolytes, acid–base therapies.
- Anti-endotoxic NSAID protocol, e.g. 0.25 mg/kg flunixin meglumine three times daily.
- Polymyxin B as an anti-endotoxin therapy (6000 IU/kg diluted in 5% dextrose iv every 8 hours).
- Possible hyperimmune plasma or purified endotoxin immunoglobin products.
- Monitor clinical status, plasma electrolytes/proteins, packed cell volume (PCV).
- Possible IV colloids or plasma transfusion if severe hypoproteinaemia.
- Antimicrobials – little evidence for either beneficial or detrimental effects in mild infections or acute colitis; ineffective in eliminating carrier state; indicated in foals; antimicrobial

resistance common so confirm sensitivity in laboratory; consider using trimethoprim sulpha, chloramphenicol, amikacin, ampicillin or gentamicin.
- Prophylaxis against laminitis e.g. frog support.
- Intestinal protectants e.g. bismuth subsalicylate, di-tri-octahedral smectite.
- Nutritional support.
- Probiotics/prebiotics (currently unproven benefit).
- General biosecurity protocols.

Prognosis

- Fifty per cent mortality in cases of acute colitis and especially foals.
- Small proportion of acute colitis cases develop chronic enteropathy with progressive weight-loss and persistent diarrhoea.
- Recovered colitis cases may shed Salmonella in faeces for up to 4 months.
- Possible complications include laminitis, thrombophlebitis and renal failure.

Prevention and control

- Minimize stress, e.g. associated with surgery, transportation etc.
- Strict isolation of affected horses.
- Monitor in-contact horses for early signs of dullness, inapettence or fever.
- No commercially available equine vaccines, but protection has been achieved using formalin-killed *S. typhimurium* and *S. enteriditis*.

19.17 CLOSTRIDIAL DISEASES

Clostridium spp. are important equine pathogens that can cause a wide-range of diseases. Many clostridial diseases can be rapidly fatal; however, most members of this genus can be found in healthy horses and are widely distributed in the environment. *Clostridium difficile* and *C. perfringens* are considered important causes of colitis in adult horses and foals, but other clostridia probably also play a role. Toxins produced by *Clostridium tetani* are the cause of tetanus (Chapter 11), and those produced by *Clostridium botulinum* cause botulism (Chapters 11 and 20) and have been implicated in equine grass sickness (dysautonomia) (Chapter 3). Various clostridia, including *C. perfringens*, can cause clostridial myonecrosis.

Clostridia are Gram-positive spore-forming anaerobic bacteria. Many clostridia are normal commensal components of the intestinal microflora of healthy horses and are likely to play an important role in maintenance of digestive tract health. However, some are opportunistic pathogens that can cause a wide range of diseases. The spore-forming nature of clostridia may play an important role in disease pathogenesis by allowing for prolonged survival in inhospitable conditions as well as being resistant to disinfectants.

Clostridial enterocolitis

Enterocolitis is a sporadic but potentially fatal disease in the horse. Numerous clostridia may be involved in its aetiopathogenesis, but the most commonly implicated are *C. difficile* and *C. perfringens*.

Clostridium difficile infection

Clostridium difficile is an important cause of colitis in some regions. Sporadic cases are most common; however, outbreaks can occur, particularly amongst foals on breeding farms and adult horses in veterinary hospitals.

A small percentage of healthy adult horses and foals carry *C. difficile* in their intestinal tracts. The prevalence of carrier status is higher among foals and horses treated with antimicrobials. The pathogenesis of disease involves proliferation of toxigenic strains of *C. difficile* in the intestinal tract, followed by production of bacterial toxins. Three major toxins may be produced by *C. difficile* strains, toxin A (an enterotoxin), toxin B (a cytotoxin), and CDT (a binary toxin). Factors that disrupt the intestinal microflora such as antimicrobial therapy may predispose to the proliferation of toxigenic strains of *C. difficile.*

The clinical presentation of *C. difficile* infection is highly variable. Signs range from mild diarrhoea with no signs of systemic illness to peracute and rapidly fatal necrotizing haemorrhagic enterocolitis. Signs of severe systemic illness may be associated with endotoxaemia and systemic inflammatory response syndrome (SIRS).

Diagnosis involves the detection of clostridial toxins in faeces. Tests that detect both toxin A and toxin B are preferred because some equine strains produce only toxin B. The concurrent detection of *C. difficile* by culture, antigen ELISA or PCR can be helpful.

Supportive care is the most important aspect of treatment. Di-tri-octahedral smectite may bind to clostridial toxins. Probiotic compounds have been suggested, but there is little evidence regarding their efficacy. If the diarrhea is considered to be associated with antimicrobial therapy, all antimicrobial treatments should be withdrawn, if possible. Metronidazole (15 mg/kg PO q8h for 3–5 days) is commonly used and may increase the survival rates.

Complications such as laminitis, venous thrombosis and disseminated intravascular coagulation (DIC) can occur, and these complicate both the treatment and the prognosis. There are no specific preventive measures. Judicious use of antimicrobials and other general practices such as providing a proper diet, minimizing diet and management changes and reducing 'stress' may be important. A vaccine is not currently available.

Clostridium perfringens infection

Clostridium perfringens is a ubiquitous species that can be found in the intestinal tract of many animals and in the environment. It is commonly implicated as a cause of colitis in adult horses and foals. *C. perfringens* can be classified into five types according to its ability to produce four major toxins.

The pathogenesis of disease is poorly understood but presumably involves proliferation of *C. perfringens* in the intestinal tract with associated toxin production. Whether this occurs following ingestion of *C. perfringens* or from proliferation of endogenous *C. perfringens* remains unclear.

Clinical presentation is variable and non-specific as described for *C. difficile*. It has been suggested that *C. perfringens* may cause severe haemorrhagic diarrhoea more often, but this has not been proven.

Definitive diagnosis is difficult. Isolation of *C. perfringens*, quantification of *C. perfringens* and analysis of faecal smears for clostridial spores are all non-diagnostic. Detection of enterotoxin is possible by ELISA or reverse passive latex agglutination assay (RPLAA). Neither has been validated for use in horses, and there are concerns about poor specificity of the RPLAA. Positive ELISA results can be treated as a presumptive diagnosis.

Treatment is basically supportive. Metronidazole (15 mg/kg PO q8h for 3–5 days) is commonly administered.

An equine vaccine is not currently available.

Duodenitis/proximal jejunitis (see Chapter 2)

This syndrome, also referred to as 'anterior enteritis', is a sporadic disease characterized by inflammation and oedema of the proximal small intestinal, with subsequent ileus, small intestinal distension, gastric distension and signs of moderate to severe colic.

Clostridium difficile has been implicated as a cause of this condition based on a high prevalence of isolation of *C. difficile* from gastrointestinal reflux of affected horses but not controls with gastric reflux from obstructive lesions. Despite this association, causation has not yet been demonstrated.

The association between *C. difficile* and duodenitis/proximal jejunitis suggests that empirical treatment for clostridial infection with metronidazole (40–60 mg/kg q8–12 hours per rectum or 10–20 mg/kg IV q 6-8h) or penicillin (sodium or potassium penicillin, 20 000–40 000 IU/kg IV q6 hours) could be considered along with supportive therapy; however, the efficacy is unclear.

Clostridial myonecrosis (see Chapter 21)

Clostridial myonecrosis, often referred to as clostridial myositis, gas gangrene and malignant oedema, is an uncommon but severe disease caused by growth of clostridia in skeletal muscle, with subsequent tissue necrosis and systemic inflammation. *Clostridium perfringens* has been most commonly implicated, but various clostridia can be involved, including including *C. septicum*, *C. sporogenes* and *C. sordellii*.

Clostridial spores can be present in healthy muscle but are dormant in the aerobic environment of healthy muscle. Severe tissue necrosis following the injection of irritatant substances or trauma can create an anaerobic environment that allows germination of spores. Alternatively, clostridial spores can be introduced into the muscle during injection or with penetrating trauma. If an anaerobic environment results, spore germination can occur. Most reported cases are associated with intramuscular injection. Intramuscular administration of flunixin meglumine has been implicated as a leading cause of clostridial myonecrosis.

19.18 STRANGLES

This is a common condition that may result in upper airway constriction ('strangles') due to enlargement of the lymph nodes of the head and neck. Acute, contagious disease is caused by *Streptococcus equi* subspecies *equi* (frequently referred to as *Streptococcus equi*) and characterized by inflammation of nasal and pharyngeal epithelium, followed by lymphadenitis, then abscess formation in draining lymph nodes.

Epidemiology

Strangles is more common and severe in young horses (especially 1–5 years), but horses of any age may be affected and are predisposed by stress, e.g. weaning, transportation. It is a group disease with high morbidity of almost 100% in horses with no previous exposure. Mortality is low – about 1% – but various complications may occur. It is usually introduced by a horse incubating disease or by 'carrier' horses. Although highly contagious, spread is slow compared with respiratory viruses and requires direct contact of contaminated equipment, tack etc. Copious purulent discharges result in rapid contamination of the environment, and *S. equi* organisms can survive for several months outside the host. Nasal shedding of the organism typically continues for 2–3 weeks, but recovered cases may be potential sources of infection for up to 6 weeks. A carrier state may develop in up to 10% of infected horses with infection of the guttural pouches. Some horses with infection of the guttural pouches. Some horses with infection of the guttural pouches may develop chronic empyema.

Pathogenesis

Streptococcus equi is a Gram-positive beta-haemolytic bacterium of Lancefield group C. Pathogenicity is related to properties of adherence, resistance to phagocytosis and toxin/enzyme production. Infection is by inhalation or ingestion of *S. equi* organisms, which colonize upper respiratory tract/pharyngeal/palatine mucosae. *S. equi* attaches to cells in the crypts of the lingual and palatine tonsils, and the follicular associated epithelium of the pharyngeal and tubal tonsils. The release of bacterial enzymes/toxins gives rise to rhinitis, pharyngitis and fever. This is followed by the spread of *S. equi* to local lymph nodes with resultant lymphadenitis and abscess formation. In some instances there is a bacteraemia which may be associated with spread of *S. equi* to satellite lymphoid tissue. Progressive physical expansion of infected lymph nodes results in rupture either externally to the skin surface (e.g. submandibular) or internally to the guttural pouch (e.g. retropharyngeal).

Clinical signs

The incubation period is 2–6 days and clinical course about 3 weeks if untreated.

- Pyrexia – 39.4–40.5°C, 103–105°F; often biphasic – first fever due to pharyngitis then second febrile response associated with lymph node infection.
- Anorexia, dullness, lethargy – improve following lymph node drainage.
- Nasal discharge; serous then mucopurulent then purulent.
- Lymphadenopathy – especially submandibular, retropharyngeal and parotid; infected lymph nodes progressively increase in size then soften and burst over 3–7 day period (see Figure 19.2).
- Respiratory stertor and dyspnoea. This may become so severe as to necessitate a tracheostomy.
- Dysphagia.
- Conjunctivitis and mucopurulent ocular discharge.
- Facial oedema, and/or abscess formation.
- Coughing is not usually a prominent sign.

Additional clinical signs occur as complications due to either local spread of infection, satellite abscess formation and metastatic spread of infection ('bastard strangles') or immune-mediated disease, e.g. purpura haemorrhagica.

Complications

Complications occur in approximately 20% of cases.

Figure 19.2 Strangles. Multiple subcutaneous abscesses around the head, some of which have ruptured through the skin surface.

Common complications

- Chronic nasal discharge – persistent guttural pouch empyema (see Figure 19.3) (resulting from retropharyngeal lymph node absecesses that rupture into the guttural pouches) or sinusitis.
- Purulent secretions that persist in the guttural pouch may form hard, inspissated concretions called chondroids (see Figure 19.4).

Undiagnosed guttural pouch empyema and chondroids can result in persistent shedding of *S. equi* into the environment.

Rare complications

- Colic/weight-loss – septic peritonitis following rupture of abscessed mesenteric lymph nodes.
- Peripheral oedema – purpura haemorrhagica: aseptic immune-mediated vasculitis associated with immune complex deposition within blood vessel walls. The clinical signs can vary from a mild transient reaction to a severe or even fatal disease.

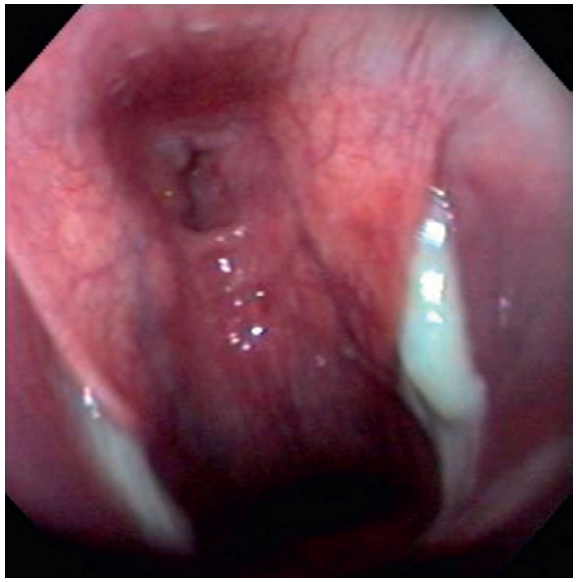


Figure 19.3 Endoscopic view of pus draining from the ostia of the guttural pouches into the nasopharynx – guttural pouch empyema.

Figure 19.4 Endoscopic view of chondroids within a guttural pouch.

Very rare complications

- Lameness/joint swelling – septic or immune-mediated arthritis and muscle damage secondary to purpura haemorrhagica.
- CNS dysfunction – brain/spinal cord abscess.
- Mastitis/agalactica.
- Abortion.
- Pneumonia.
- Renal failure – glomerulonephritis.

Diagnosis

- Clinical signs are generally observed within 7 days of infection.
- Culture of *S. equi*; swab nasopharyngeal/draining abscess but after 7–10 days secondary infections with *S. zooepidemicus* or *Streptococcus equisimilis* are isolated more easily than causative organism; atypical isolates of *S. equi* have similar characteristics to S. equisimilis.
- PCR to detect *S. equi* DNA – nasal/nasopharyngeal swabs, nasopharyngeal washes, guttural pouch lavage.
- Serology.
- Diagnosis of carriers is difficult since they may show no overt clinical signs of infection. Endoscopy can be useful to identify guttural pouch empyema and/or to obtain samples for culture/PCR by guttural pouch lavage.

Treatment (nursing)

- Soft foods.
- Hot pack/poultice superficial lymph nodes/abscesses.
- Lance/drain mature abscesses (taking care to avoid any large blood vessels).
- Bathe conjunctivae/abscess sinus tracts.
- Feed at floor level.
- Rest.
- Non-steroidal anti-inflammatory drugs to control pain and fever.
- Nutritional support; per stomach tube if dysphagic.
- Tracheotomy if severe dyspnoea.
- Guttural pouch lavage to treat empyema. Chondroids should be removed by copious lavage, suction, use of endoscopically-guided basket forceps or surgery.
- Use of antimicrobials are controversial because unsubstantiated belief that they increase complications. Treatment with antimicrobials early in the infection may prevent the infection from establishing, but it also probably prevents the development of protective immunity.
- Generally not treated with antimicrobials unless life-threatening or high risk of bacteraemia e.g. in young foals.
- Antimicrobials have no prophylactic benefit unless infection source removed/environment clean.
- Procaine penicillin 20 000 IU/kg twice daily, 7–10 days.
- Topical application of a penicillin gelatin solution into the guttural pouch can achieve high concentrations in cases of guttural pouch empyema and chronic carriers.

Prevention and control

- Isolation/segregation and handler hygiene should enable prevention of further spread because direct contact with infective material is necessary (compared with inhalation of aerosolized respiratory viruses).
- Monitoring rectal temperatures of in-contact horses and isolation at first sign of fever.
- Thorough cleaning and disinfection of contaminated housing environments before unexposed horses enter, but within grazing environment *S. equi* remains viable for several months.

- Prophylactic antimicrobial treatments generally not indicated unless at very early stages of an outbreak, before the environment has become contaminated.
- No movement of horses from the premises for at least 6 weeks, and all new intake horses enter only uncontaminated quarantine areas.
- Maintain separate groups, especially for high-risk horses such as foals.
- Vaccination at best reduces number and severity of cases. Modified-live intranasal vaccine, killed whole-organism or bacterial protein or bacterial enzyme vaccines are available in certain countries.
- Use of bacterial cultures/PCR to detect subclinical carriers.

19.19 POTOMAC HORSE FEVER

A common condition in regions of the USA and Canada – also known as equine ehrlichial colitis or equine monocytic ehrlichiosis. The causative agent is *Neorickettsia risticii*, and infection is associated with severe, acute enterocolitis, laminitis and abortion.

N. risticii is a Gram-negative coccus that grows in cell culture but not in conventional bacterial media.

Epidemiology

There is regional (Eastern States) and seasonal (summer/autumn) prevalence of seropositivity and clinical disease which implies arthropod vector transmission, but this has not been proven. It is believed that aquatic insects, such as caddisflies and mayflies, are the most likely source of infection. Under natural conditions, horses grazing near rivers/creeks ingest adult insects along with grass. Many horses have low-level seropositivity for antibodies to *N. risticii*, but clinical disease generally occurs sporadically, i.e. many infections are subclinical. The prevalence of abortion associated with *N. risticii* infection is unknown.

Pathogenesis

The organism has great affinity to blood monocytes and infects intestinal macrophages, especially those in the colon. The infected intestinal epithelial cells lose their microvilli, which leads to malabsorptive diarrhoea. The incubation period is approximately 1–3 weeks. The only directly affected tissues outside the gastrointestinal tract are the placentae of pregnant mares, and infection of the placenta can result in abortion between 190 and 250 days of gestation.

Clinical signs

Clinical signs of Potomac horse fever are those of acute enterocolitis, but there is considerable variation of severity from anorexia/dejection/ileus to fever/marked dullness/colic/profuse diarrhoea. The clinical disease in untreated cases has approximately a 5- to 10- day duration and a mortality of 5 to 30%.

- Pyrexia – often biphasic – first transient fever of 39.5–41 °C, 103.1–105.8 °F (due to neoricketsaemia) with a more persistent second phase 3 to 7 days later associated with obvious dullness and anorexia.
- Gastrointestinal stasis (ileus) and reduced borborygmi.
- Within 24–48 hours diarrhoea – varies from mild output to profuse, watery consistency.
- Mild colic.
- Peripheral oedema.
- Some horses develop severe endotoxaemia and systemic inflammatory response syndrome (haemoconcentration, metabolic acidosis, cardiovascular compromise, etc.)
- Laminitis is reported in 15–25% of cases.
- Abortions are associated with spontaneous loss of the foetus which appears fresh.
- Occasionally death of a foetus may result in maceration and mummification.
- Mortality varies from 5 to 30%.
- Long-term sequelae usually relate to laminitis.

Diagnosis

- Isolation of *N. risticii* from blood or faeces.
- Serology performed on paired samples, but interpretation is complicated by very short time-scale of antibody production – peak levels occur within a few days of infection such that a rising titre cannot always be detected if a serum sample is not collected very early in the clinical disease. In addition, vaccination results in high titres.
- Serological methods include indirect fluorescent antibody test (IFAT), enzyme-linked immunosorbent assay (ELISA) and competition enzyme-linked immunosorbent assay.
- Polymerase chain reaction (PCR) can be used for immediate, reliable accurate diagnosis – used on faeces or peripheral blood.

Treatment

- Oxytetracycline – 7 mg/kg twice a day for 5–7 days is effective if initiated early in course of disease (note: oxytetracycline is potentially causative of acute enterocolitis).
- Intravenous fluid/electrolyte therapy.
- Treatments for endotoxaemia and laminitis.
- Isolation – because of clinical similarity to acute salmonellosis and/or possible initiation of *Salmonella* spp. excretion secondary to Potomac horse fever colitis.

Prevention

- Vaccination with inactivated, partially purified, whole-cell vaccines gives fair but incomplete protection which is short-lived such that 4-monthly vaccination is required in endemic areas.
- Recovered clinical cases have a natural immunity of about 2 years' duration.

Isolation/hygiene

- Clinical cases may excrete *N. risticii* for several months; exclude healthy horses from contact with possible excretors or contaminated premises for least 6 weeks.
- Avoid crossover of equipment, tack.
- Handlers disinfect/clothing change.
- Contaminated premises steam cleaned, disinfected and vacated for 6 weeks.

19.20 PROLIFERATIVE ENTEROPATHY (*LAWSONIA INTRACELLULARIS*)

Proliferative enteropathy is an infectious disease of young horses caused by the bacterium *Lawsonia intracellularis*. This organism has been identified as an enteropathogen in numerous species, including pigs, hamsters, horses, rats, rabbits, monkeys, sheep and guinea pigs.

The organism is a microaerophilic obligate intracellular Gram-negative curved rod. It can be most readily identified in the enterocytes of infected animals using the Warthin-Starry silver stain.

Epidemiology

Equine proliferative enteropathy is a disease of young animals, with foals from 2 to 8 months most commonly affected. The disease is

typically sporadic in nature, but herd outbreaks have been reported. Given that the organism is shed in the faeces it is likely that environmental contamination with infected manure represents the route of transmission of the disease, although the actual source of *L. intracellularis* organisms in most cases remains unknown. It is possible that other domestic species or wildlife may play a role as carriers of this organism.

Pathogenesis

- *Lawsonia intracellularis* is transmitted by the faecal–oral route.
- Infections are associated with profound intestinal hyperplasia due to proliferation of infected crypt epithelial cells of the small intestine. This proliferation results in loss of the normal villous structure, leading to impaired intestinal absorption and subsequent protein-losing enteropathy.
- The development of clinical disease appears to require some degree of immune dysfunction, as weanling age horses are most susceptible. It has been proposed that the decline of maternal antibodies that occurs at several months of age in foals may predispose them to Lawsonia infection. Another possible immunosuppressive influence that may predispose foals to proliferative enteropathy is stress induced by management changes, such as weaning, conditioning and transport.
- Characteristic pathological changes include dramatic thickening and corrugation of the small intestinal mucosa. Findings on histopathology include the presence of numerous curved bacteria within the apical cytoplasm of the infected enterocytes, profound mucosal hyperplasia and the relative absence of an active inflammatory response.

Clinical signs

The presenting signs of proliferative enteropathy in foals/weanlings can include some or all of the following:

- Anorexia.
- Depression.
- Lethargy.
- Weight loss.
- Fever.
- Diarrhoea.
- Colic.
- Ventral oedema.

Oedema, colic, diarrhoea and fever are the commonest reported signs. Clinicopathological abnormalities at presentation may include panhypoproteinaemia, hypoalbuminaemia, leukocytosis, hyperfibrinogenaemia and increased haematocrit.

Diagnosis

- Definitive diagnosis of equine proliferative enteropathy requires histological examination of affected small intestine; however, surgical biopsy is rarely warranted.
- Serological tests include indirect fluorescent antibody test (IFAT), enzyme-linked immunosorbent assay (ELISA) and immunoperoxidase monolayer assays (IPMA). Both false-positive and false-negative results can occur; however, serology is considered useful by many clinicians as one component of the clinical evaluation of foals showing clinical signs suspicious of proliferative enteropathy.
- Faeces can be examined using polymerase chain reaction (PCR) techniques. PCR techniques have the benefits of being both highly specific, due to the identification of species-specific DNA, and highly sensitive, due to their ability to amplify and detect the target DNA even at very low concentrations. The primary limitation of PCR techniques is that they can detect the DNA of non-viable organisms, meaning that infection is not always present even when the DNA of the organism is detected. Another limitation of PCR techniques for the detection of *Lawsonia intracellularis* is that despite their high sensitivity these tests can yield false-negative results if the horse is not shedding the organism at the time of testing. In most cases, faecal PCR testing is combined with serological testing in evaluating suspect cases.
- Diagnostic ultrasonography can be helpful to evaluate the thickness of the small intestinal wall. A small intestinal wall thickness exceeding the normal value is considered suggestive of proliferative enteropathy in foals showing typical clinical signs.
- Clinicopathological evaluation can be helpful in supporting a diagnosis of proliferative enteropathy. Hypoalbuminaemia is the most common clinicopathological finding. Other common abnormalities are leukocytosis, hyperfibrinogenaemia and increased haematocrit.

Treatment

Treatment of proliferative enteropathy consists of supportive therapy in combination with specific therapies directed towards *Lawsonia intracellularis*. Supportive therapy is primarily directed towards correction of the hypoalbuminaemia, although fluid therapy, nutritional support and analgesic therapy are often indicated as well. Colloid administration is the cornerstone of the treatment of hypoalbuminaemia, and this is accomplished by the use of either equine plasma or a synthetic colloid such as hydroxyethyl starch.

Specific therapy requires the use of antimicrobials that are able to reach therapeutic concentrations within the cytoplasm of the infected enterocyte, due to the fastidious intracellular nature of this organism. Antimicrobial drugs that have been used include erythromycin (25 mg/kg orally every 6 to 8 hours), with or without rifampin (5 mg/kg orally every 12 hours), azithromycin (10 mg/kg orally once daily for 5–7 days, then every other day), clarithromycin (7.5 mg/kg orally every 12 hours), oral doxycycline (10 mg/kg orally every twelve hours) and intravenous oxytetracycline (6.7 mg/kg IV every 12 hours). From a limited evidence-base it appears that tetracyclines are the antimicrobials of choice in cases of proliferative enteropathy. Anecdotally, the incidence of complications from the use of orally administered erythromycin may be high in older foals with a more developed large intestinal microbiota.

Reported survival rates for affected foals/weanlings with appropriate treatment range from 81 to 93%. These results reinforce the fact that, with early detection and appropriate therapy, most foals should survive. The gastrointestinal abnormality that is characteristic of this disease is not permanent in nature, but the impaired growth that results from the protein-losing enteropathy does appear to slow the growth of affected foals. Most foals appear to regain normal growth rates after several months, and eventually may 'catch up' with their peers.

19.21 LEPTOSPIROSIS

Serological studies show that equine leptospiral infections occur worldwide, but there are regional variations in both prevalence of seropositivity and the predominant serovars. Leptospirosis is rarely recognized as a clinical entity in the horse with the exception of association with recurrent uveitis (see Chapter 12) and abortion (see Chapter 14). Leptospirosis is occasionally associated with renal disease, and with liver disease and jaundice. *Leptospira* serovar Pomona is responsible for most of the leptospiral-associated disease in north America, whilst serovar Grippotyphosa appears to be most common in Europe.

19.22 ANTHRAX

Clinical disease due to *Bacillus anthracis* infection is very rare in the horse. Infection is usually rapidly fatal with signs of fever, severe colic, and oedematous swelling.

19.23 BRUCELLOSIS

Brucellosis is a major disease of domesticated and wild animals worldwide, and is also an important zoonosis. Infection with *Brucella abortus* in the horse is rare but has been associated with infectious bursitis, arthritis or tenosynovitis, especially fistulous withers – a painful swelling of the supraspinous bursa of T2–T5 vertebrae with or without purulent discharge from a skin fistula. Affected horses may also show fever, lethargy and general stiffness. Less commonly vertebral osteomyelitis, abortion and infertility in stallions has been reported. Serological surveys indicate that many horses are exposed to *B. abortus* without developing clinical disease.

19.24 TUBERCULOSIS

Mycobacterium species are widespread in the environment, yet disease caused by these organisms is rare in the horse. Equine tuberculosis is an unusual multisystemic disease associated with signs of weight loss and neck stiffness: granulomatous lesions occur most commonly in the bowel, lung and cervical vertebrae.

19.25 GLANDERS

Synonyms are farcy and enzootic lymphangitis.

Glanders is a highly contagious disease caused by infection with the Gram-negative bacterium *Burkholderia mallei* (formerly known as *Pseudomonas mallei*). The disease occurs in some regions of Asia, the Middle East, south America and North Africa, but is exotic to Europe and the USA. An acute bronchopneumonia occurs in donkeys and mules which is usually fatal, whereas in the horse a chronic cutaneous form is more common. The lesions (nodules and ulcers) occur on nasal mucosa, ventral abdomen, distal limbs, face and neck. Lesions are similar to ulcerative lymphangitis, consisting of subcutaneous nodules that ulcerate and discharge a honey-like exudate. Lymphangitis and lymphadenitis occur. Both acute (especially in donkeys) and chronic (especially in horses) forms of the disease occur. Treatment is contraindicated because it may result in carrier animals. No vaccines are currently available. Glanders is a potential zoonosis.

19.26 LYME DISEASE

Lyme disease is caused by the spirochaete *Borrelia burgdorferi*. The infection has been recognized in horses in Europe, Russia, USA, China, Southeast Asia and South Africa. The organism is transmitted by ticks (*Ixodes* spp.). Serological studies indicate that exposure to the organism is common in endemic areas, but clinical disease is rare. Clinical signs associated with Lyme disease include fever, lethargy, arthritis, joint swelling, lameness, muscle pain, skin lesions and neurological/behavioural abnormalities. Diagnosis is confirmed by serology or western blot. Treatments include orally administered doxycycline, intravenously administered oxytetracycline or intramuscularly administered ceftiofur.

19.27 EQUINE GRANULOCYTIC EHRLICHIOSIS

This disease is caused by *Anaplasma phagocytophilium* (a coccobacillary Gram-negative organism); the disease is seasonal (autumn, winter and spring) and is recognized predominantly in northern California, although it has been recognized in other parts of the USA and in Europe. Tick (*Ixodes* spp.) transmission is likely, and the organism infects circulating neutrophils and eosinophils. Clinical signs include fever, depression, inappetence, limb oedema, petechiation, icterus and ataxia. Diagnosis is confirmed by visualising the characteristic morulae in the cytoplasm of neutrophils and eosinophils in a peripheral blood smear. Treatment consists of intravenous administration of oxytetracycline.

19.28 *CORYNEBACTERIUM PSEUDOTUBERCULOSIS*

Corynebacterium pseudotuberculosis is a Gram-positive rod-shaped, intracellular, facultative anaerobic bacterium with worldwide distribution. Infection of horses with the organism is most commonly seen in the western USA, particularly California. Infections occur both as sporadic cases on a farm or as outbreaks involving hundreds to thousands of horses in a region. Evidence exists that infection is increasing in prevelance, possibly associated with climate change. Three forms have been described in horses: ulcerative lymphangitis or limb infection, external abscesses and internal infection. Treatment for external abscesses requires establishment of drainage. Antimicrobials are indicated for horses with ulcerative lymphangitis and for horses with internal abscesses. Trimethoprim-sulfa or procaine penicillin are effective against external abscesses especially on the ventral midline. Rifampin in combination with ceftiofur appears highly effective for treatment of internal abscesses.

19.29 TYZZER'S DISEASE (see Chapter 20)

Tyzzer's disease is a rare, sporadic cause of hepatic failure in foals between 1 and 6 weeks of age. The disease is caused by infection with *Clostridium piliforme* (previously known as *Bacillus piliformis*), and appears to have a worldwide distribution. The disease is often peracute, with the time between the onset of signs to death being as short as 2 hours; many affected foals are found dead. Affected foals are typically jaundiced. Other signs that are commonly present include tachycardia, tachypnoea and seizures. Diagnosis can be achieved by demonstrating the organism histologically in liver tissue obtained at post mortem or by identifying the organism in faeces using PCR. Treatment should be aimed at aggressive antimicrobial therapy combined with intensive supportive management of hepatic failure.

19.30 OTHER EQUINE BACTERIAL INFECTIONS

Various other bacterial infections occur in the horse and may be associated with clinical diseases, some of which are described elsewhere in the text.

- Neonatal septicaemia (Chapter 20).
- *Rhodococcus equi* (Chapter 6).
- *Clostridium tetani* (Chapter 11).

- *Listeria monocytogenes*.
- *Actinomyces* spp.
- *Clostridium botulinum* (Chapters 11 and 20).
- *Taylorella equigenitalias* (Chapter 14).
- *Ehrlichia equi (Rickettsia equina)*.

19.31 FUNGAL INFECTIONS

Important fungal infections in horses include dermatophytosis, sporotrichosis, pythiosis, etc. Dermatophytosis ('ringworm') (see Chapter 13) is a highly contagious fungal infection caused most commonly by *Trichophyton equinum*. Transmission occurs by direct contact or through co-sharing of tack, etc. Lesions appear as spreading circular patches of alopecia surrounded by scaling and erythema. Papules and pustules may also be evident. Sporotrichosis (*Sporothrix schenckii* infection) (see Chapter 13) can result in single to multiple, firm, non-painful cutaneous nodules on the distal limbs, head, neck or trunk. Pythiosis (see Chapter 13) caused by *Pythium insidiosum* (a protistal organism) infection can affect horses in tropical and subtropical climates; it classically causes large, proliferative, ulcerated, pyogranulomatous, cutaneous and subcutaneous lesions of the extremities, ventral abdomen and face. Fungal organisms such as *Conidiobolus coronatus, Cryptococcus neoformans* and *Coccidioides immitis* can form granulomatous mass-like lesions in the mucosal tissues of the upper respiratory tract, while *Aspergillus* spp. tend to form superficial mycotic plaques. The most common clinical signs of upper respiratory fungal infection include unilateral or bilateral mucopurulent or serosanguinous nasal discharge and inspiratory or expiratory noise. Fungi can also occasionally cause discrete or diffuse pulmonary granulomas or fungal pleuropneumoia.

19.32 INVESTIGATION OF PARASITE-ASSOCIATED DISEASE

Introduction

Although many distinct, parasitic disease states occur in equids, the majority of equine parasite infections are of mixed species of intestinal worms, and the infections are either asymptomatic or contribute to non-specific symptoms of ill-thrift, anorexia, reduced performance and poor haircoat condition. In this chapter individual parasite species infections are described in conventional terms of epidemiology, pathogenesis, clinical signs etc. but in the field it is quite rare to encounter these mono-specific infections as discrete clinical entities.

History

The key points are:

- Previous confirmed parasitic diseases on the premises.
- Specific usage of anthelmintics and other parasite control measures for all horses on the property, i.e. *not* just the affected horse.

Often parasite control is haphazard, especially in situations of shared grazing boarding properties. Lack of synchronization of anthelmintic dosing times and/or products used are the likeliest cases of apparent failure to prevent parasitic disease, but it is also important to consider the possibility of anthelmintic resistance. Relevant points of history for the individual clinical case suspected of parasite-associated disease are:

- Age.
- Season of year.
- Grazing – yes/no, alone/shared?
- Parasite control programme – yes/no, specific nature (see below)?
- Anthelmintic dosing – frequency, products, date of last dosing and product used.

Physical examination

Few parasite-associated diseases have characteristic physical findings other than those affecting the skin (Chapter 13).

Faecal worm egg count (FWEC)

The most common quantitative technique is the modified McMaster method. It is notoriously difficult to relate level of FWEC to worm burden, and some species have particularly high fecundity, e.g. *Parascaris equorum*. Strongyle FWECs of ≤200 eggs per gram (epg) are considered low and ≥1000 epg are high whereas for *P. equorum* counts of >2500 epg are considered high.

- Efficacy of detection of tapeworm eggs using routine faecal flotation methods is somewhat insensitive but may be improved by specialized centrifugation/flotation techniques.
- Exclusion of parasitic disease on the basis of low or negative FWEC is a common, fundamental diagnostic error: the most pathogenic stages of many equine parasites are larval, i.e. not egg producing.
- Specialized flotation techniques and possibly faecal antibody estimation are required for accurate detection of *Coccidia* spp., *Cryptosporidium* spp., and *Giardia* spp.

Faecal egg count reduction test (FECRT)

The percentage of reduction of FWEC following anthelmintic dosing is calculated from the values obtained in samples on day of dosing and 10–14 days later. The FECRT is the most widely used method for monitoring the development of anthelmintic resistance. The inherent variability of FWEC together with the small sample numbers from horse properties (compared with a flock of sheep) mean that the FECRT is sub-optimally sensitive for early detection of anthelmintic resistance. Although not yet widely available, computer-based modelling methods for assessment of anthelmintic resistance have been developed. There are also in vitro methods of egg hatching and larval migration which have been used in research studies on anthelmintic resistance within cyathostomins, but these are not currently available for routine use.

Faecal larvae

Cyathostomin larvae may be grossly evident as 1-cm long, red or white worms and if present in large numbers are evidence of clinical cyathostominosis. Larval detection can be facilitated by dilution of faeces in water and screening under a microscope.

Lungworm larvae are recovered by a Baerman technique and identified using microscopy.

Haematology/blood biochemistry

Although certain parasitic diseases may result in haematological and plasma protein changes, none of these have diagnostic specificity with the possible exception of hyperbetaglobulinaemia, which is often associated with larval cyathostominosis.

Serology

A serological test (quantitative ELISA) for tapeworm infection is available in Europe.

Bronchoalveolar lavage

This is a useful technique for confirming lungworm infection (see Chapter 6).

Cytology/histopathology

In specific situations cytological examination of fluid samples and/or histopathology of affected tissues are applied to parasite-associated disease, e.g. cerebrospinal fluid (CSF) analysis (Chapter 11) or skin biopsy (Chapter 13).

Post-mortem examination

Definitive confirmation of parasitic disease is often only made on gross pathology and/or histopathology findings.

19.33 CONTROL STRATEGIES FOR EQUINE PARASITE INFECTIONS

Epidemiology of the important parasite species forms the basis of strategies for equine parasite control. It is important to take into account regional variations in parasite epidemiology as well as factors such as host age, group size/stocking density and introduction of new horses. In addition, management considerations such as costs, ease of sampling groups of horses and reliability of record keeping affect the decision to implement control strategies such as pasture hygiene or targeted dosing. Where possible, young stock should be grazed separately from mature horses because there is usually greater transmission of parasites within the younger groups which are more susceptible to acquiring both strongyles and ascarids.

There have been very few robust studies on the relative efficiency of different equine parasite control programmes, and current best practice advice is based on opinion rather than evidence. Overall the aim should be responsible use of anthelmintic to achieve optimal parasite control with minimal pressure for development of anthelmintic resistance.

It is impossible to make standard recommendations for control of parasites for every different system of horse management.

Summary options are included in Table 19.1 which can be modified according to the situation on a specific establishment with the guiding principle of minimizing total usage of anthelmintic without compromising the risk to horse health. Regardless of the control programme adopted, it is considered best to use separate broad-spectrum drug classes for consecutive 12-month periods, e.g. Year 1 pyrantel; Year 2 macro cyclic lactones (e.g. ivermectin/moxidectin); Year 3 benzimidazoles (only if known susceptibility to this group – in recent surveys 80–100% benzimidazole-resistant cyathostomes); Year 4 pyrantel; Year 5 macrocyclic lactones etc. It is important to emphasize to owners/managers that the rotation of anthelmintic class is required, not the rotation of individual products. It may be useful to incorporate a single boticidal drug (e.g. ivermectin) in late autumn/winter, and control of tapeworms can be achieved by an additional, specific praziquantel dose once or twice *per annum*. It is essential to monitor the efficacy of each broad spectrum drug class on at least an annual basis by performing 10- to 14-day post-treatment FWEC which are ideally incorporated into FECRT. Assuming accurate dosages were given and consumed, the likeliest cause of drug failure is anthelmintic resistance.

Table 19.1 Control programmes for equine parasites

Programme	Dosing regimen	Comments
Targeted dosing	– Monthly FWEC for all grazing horses. – Dose all that are worm egg count positive (or arbitrary level, say >200 eggs per gram) with anthelmintic. – Bi-annual tapeworm serology. – Dose all tapeworm positive horses with praziquantel.	– Minimizes total anthelmintic usage and so reduces pressure for development of anthelmintic resistance. – Appropriate on premises with: strictly controlled grazing management; mature horses only; minimal new intake horses; efficient and compliant owner/manager prepared to undertake frequent sampling.
Strategic dosing	– Only spring/summer anthelmintic dosing. – All grazing horses dosed at same time points. – Dosing repeated on two or three occasions at pre-determined intervals which vary with each anthelmintic group.	– Reduces anthelmintic usage and so may reduce pressure for development of anthelmintic resistance. – Regional variations in climate affect the total period for which suppression of faecal worm egg output is required. – Appropriate on premises in areas which have discrete periods of weather detrimental to parasite survival on pasture, namely dry heat sustained for several weeks.
Interval dosing	– Year-round synchronized dosing all grazing animals. – Dosing at pre-determined intervals which vary with each anthelmintic group.	– Intensive anthelmintic usage predisposes to development of anthelmintic resistance so requires vigilant post-treatment monitoring. – Appropriate for situations where: grazing group is casually managed, e.g. some multi-owner self-care boarding premises; young horses grazing; frequent new intake horses.
Non-chemical pasture hygiene	– Once (or twice) weekly removal faeces from grazing area.	– Often used as adjunct to anthelmintic dosing control but can work as sole means of worm control. Not suitable on breeding farms/studs as sole control method. – Widely advocated but not robustly assessed and, at least in theory, the reduction of pasture stages of parasites in refugia may actually increase pressure for development of anthelmintic resistance if performed as adjunct to anthelmintic dosing.

Table 19.2 Outline of a parasite control programme suitable for a breeding/rearing property

Animal group	Target parasites	Anthelmintic	Comments
Foals and weanlings	Ascarids Small strongyles	Pyrantel at 4-week intervals (or benzimidazoles at 6-week intervals but only if known susceptibility)	Ascarid eggs survive for years in soil and are a major threat to foal health as paddock infestation levels can build up in nursery paddocks over time.
Yearlings	Ascarids Small strongyles Tapeworm	Ivermectin at 6- to 8-week intervals and then moxidectin single dose only at c. 20 months of age Single annual dose with tapeworm dose (praziqauntel)	From perspective of parasite control, grazing in groups by age is appropriate. From ca, 12 to 18 months of age horses develop immunity to ascarids.
Two-year-olds	Small strongyles Tapeworm	Interval dosing: either benzimidazoles at 6-week intervals or pyrantel at 4-week intervals. Single-dose moxidectin at c. 32 months of age Single annual dose with tapeworm dose (praziquantel)	Benzimidazole resistance common so only use if known susceptibility.
Mares (and others)	Small strongyles Tapeworm	Follow general guidelines, see Table 19.1	Ideally targeted dosing (Table 19.1) as the preferred option but given that there will typically be lots of on/off movements, and that mares will be co-grazing with foals, there is overall a need to consider that this situation is a higher risk for transmission of parasites. Likely that in many instances interval dosing will be the practical option

1. Note that: annual rotation of anthelmintic class (macrocyclic lactones [ivermectin/moxidectin]; pyrantel; benzimidazoles) for each programme option. Piperazines, phenothiazines and organophosphates are available but are used infrequently.
2. Bots are not important pathogenic parasites, but owners are often aware of their existence. Drug of choice for bots is a single dose of ivermectin in late autumn/winter.
3. Anthelmintic resistance is widespread among small strongyle (cyathostomins) populations and, of the major drug classes it has been widely reported to benzimidazoles. There are increasing reports of pyrantel resistance and early evidence of development of macrocylic resistance. For practical purposes, anthelmintic resistance should be considered irreversible.
4. Apparent lack of efficacy, possibly due to ivermectin resistance, has been reported in ascarid infections on large breeding/rearing properties. On this evidence it appears that ivermectin is not indicated for ascarid control, and other classes of drug should be used for parasite control prorgrammes on properties with large numbers of foals/weanlings and yearlings.
5. The current consensus is that serial dosing with moxidectin during a single year should not be practised because this causes unacceptable pressure for the development of macrocyclic lactone resistance.
6. Praziquantel is narrow-spectrum (tapeworm only) available in combination products with macrocyclic lactones.
7. A pasture hygiene approach of removing faeces from pasture has the advantage of significantly increasing the proportional area of fields which are actually grazed in addition to decreasing pasture infectivity.

On properties such as breeding farms there will often be several sub-populations of host (foals, weanlings, youngstock, mares) with several separate parasite populations (ascarids, small strongyles, tapeworm) of varying importance in the different age groups. An outline of a parasite control programme which may be suitable for this type of property is shown in Table 19.2.

19.34 STRONGYLES

These are colloquially known as red worms and are common equine parasites (>85% prevalence small strongyle infection but previously widespread large strongyle infection now only around 5% in most developed countries). Young horses are more susceptible, but there is only limited immunity so strongyle infection can occur at any age. There is a long-established association of colic with strongyles (especially *Strongylus vulgaris*) and more recent recognition of severe disease entity of larval cyathostominosis which usually affects horses less than 5 years old in late winter or early spring.

Aetiology/epidemiology

There are two subfamilies:

1. Strongylinae (large strongyles): *Strongylus vulgaris*, *Strongylus edentatus*, *Strongylus equinus*, *Triodontophorus* spp.
   - Direct, *migratory life* cycle (*Triodontophorus* spp. non-migratory).
   - Adult stages – large intestinal.
   - Prepatent period – 6–10 months.
   - Seasonal variation of pasture infectivity – maximal late summer/autumn in temperate zones.
2. Cyathostominae (small strongyles/cyathostomins) – 8 genera; over 50 species, of which 10 occur commonly.
   - Direct, non-migratory life-cycle.
   - Adult stages – large intestinal.
   - Prepatent period – 6–20 weeks.
   - Arrested larval development in large intestinal mucosa – period of arrest may be from several months' to 2–3 years' duration (see Figure 19.5).
   - Pasture infectivity seasonal, as for large strongyles.

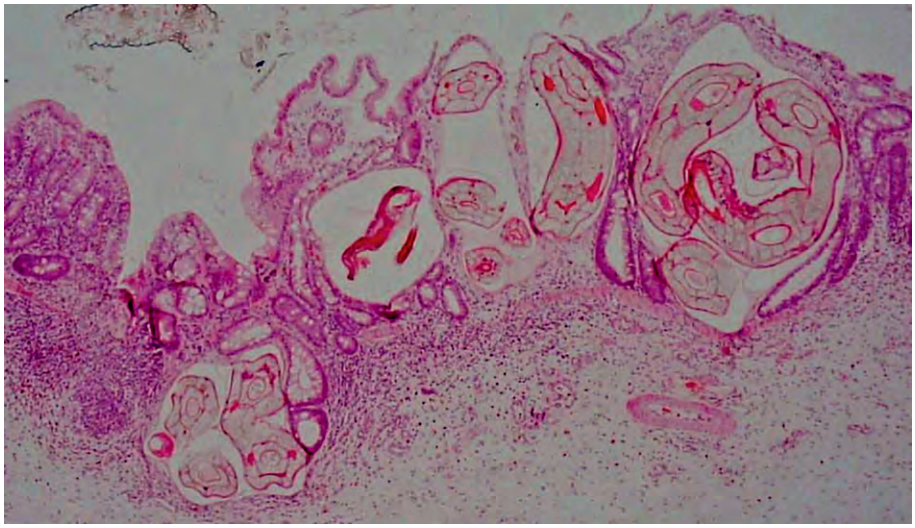


Figure 19.5 Photomicrograph of encysted mucosal cyathostomin larvae in the large colon.

Pathogenesis

Larval stages rather than adults are responsible for the main pathogenic effects. Various, inter-related changes involved in pathogenesis of these effects:

- Weight loss – protein-losing enteropathy.
- Colic.
  - Possible involvement of strongyles in many types of colic (Chapter 2).
  - Intestinal motility changes with large and small strongyle infection; detailed mechanisms not known.
  - Altered mesenteric blood flow in *S. vulgaris* infection associated with larval migration within intestinal blood vessels gives rise to non-strangulating intestinal infarction. Precise mechanisms not known but not thromboembolism.
  - Peritonitis, larval migration with *S. edentatus* and *S. equinus*.
  - Colitis/typhlitis and intestinal, mucosal oedema with cyathostominosis.
  - Caeco-caecal intussusception recognized (rare) with cyathostominosis.
  - Anaemia – adult large strongyles not blood feeders but cause incidental blood loss from mucosal bites.
  - Diarrhoea.
  - Synchronous, mass emergence into lumen of cyathostomin larvae arrested in development within intestinal mucosa causes typhlitis/colitis.
  - Mucosal injury at mucosal penetration of infective cyathostomin larvae – not a marked feature of large strongyle infections.

Clinical signs

Strongylosis, i.e. mixed strongyle species infection:

- Ill-thrift/weight loss/poor body coat condition ± diarrhoea.
- Colic may be severe and occur in recurrent bouts.
- Anaemia ± lethargy ± poor performance.

Cyathostominosis:

- Weight loss: marked and progressive; may precede onset of diarrhoea.
- Diarrhoea: sudden onset; possibly severe; usually becomes chronic; less severe systemic illness than other acute colitides (Chapter 3); may be recurrent diarrhoeic bouts in older animals.
- Colic: not always; usually mild.
- Oedema: variable; limbs, ventral, preputial.

Diagnosis

Faecal examination:

- FWEC is not reliable.
- Large numbers cyathostomins in diarrhoeic faeces/rectal scrape in cyathostominosis (note: cyathostominosis may initiate faecal excretion of *Salmonella* spp. which should be regarded as secondary/incidental).

Blood biochemistry/haematology:

- Variable, possibly within normal ranges.
- Hypoalbuminaemia.
- Hyperbetaglobulinaemia on serum protein electrophoresis.
- Neutrophilia.

Histopathology:

- Rectal biopsy occasionally produces evidence of cyathostomin larvae.
- Intestinal biopsy, rarely performed in live horse but ideal for definitive diagnosis of larval cyathostominosis.

Exploratory celiotomy:

- Non-strangulating infarction lesions grossly evident.
- Multiple intestinal biopsies to investigate weight loss/chronic diarrhoea (Chapter 3).

Treatment

- Anthelmintic.
  - Larvicidal doses of oral ivermectin 0.2 mg/kg; oxfendazole 10–50 mg/kg; fenbendazole 7.5–10 mg/kg on 5 consecutive days or 60 mg/kg as single dose; or oral moxidectin 0.4 mg/kg. From published anthelmintic efficacy studies, only the 5-day fenbendazole protocol and moxidectin have efficacy against cyathostomin larvae arrested within the large intestinal mucosa. The use of moxidectin, a lipophilic compound, is contraindicated in emaciated horses (and also in foals under 4 months of age).
  - Repeat at anthelmintic treatment at multiple 10 day intervals for cyathostominosis (off-label recommendation).
  - Possible exacerbation of colic.
- Analgesic – see Colic (Chapter 2).
- Surgery – resection infarcted bowel.
- Antidiarrhoeic – oral codeine phosphate; 1–3 mg/kg once, twice or three times daily to effect (see also Chapter 3).
- Corticosteroids – cyathostominosis – oral prednisolone 1 mg/kg once a day, before 9 a.m for 30 days then taper dose.

Prognosis

The prognosis is good for general strongylosis, fair for larval cyathostominosis (about 50% survive) and poor for non-strangulating infarction.

Prevention/control

This is summarized in Section 19.33.

19.35 ASCARIDS

These are important and common parasites in horses under 18 months old.

Aetiology/epidemiology

Parascaris equorum

- Direct, migratory life-cycle.
- Adult stages are in the small intestine.
- Prepatent period is 3 months.
- Prolific egg production leading to heavy contamination of grazing/stables.
- Eggs are resilient and are the infective stage.

Pathogenesis

Adult stages are responsible for main pathogenic effects

- Ill-thrift.
  - *Not* a catabolic condition (cf. strongyle infections).
  - Reduced digestive efficiency – worms feed on intestinal content.
  - Reduced feed intake.
- Colic.
  - *Rare.*
  - *Intestinal* impaction with adult worms and may lead to viscus rupture.
- Respiratory signs.
  - *Cough and nasal discharge during pulmonary phase of larval migration (see Chapter 6).*
  - *Not common* feature unless high levels of infection.

Clinical signs

- Poor body condition or emaciation.
- Bilateral nasal discharge and coughing (rare).
- Colic (rare): may present as acute abdominal crisis.

Diagnosis

- FWEC: large, round thick-shelled eggs; a sensitive and specific test in ascarid infection (unlike for other horse parasites)

Treatment

- Anthelmintic: most classes have good efficacy against adult stages but most less effective against migrating and/or immature larvae. Proven regimens against larval stages are: fenbendazole, 10 mg/kg on 5 consecutive days; ivermectin 0.2 mg/kg; or levamisole 8.0 mg/kg (not licensed for use in horses in some countries); or moxidectin 0.4 mg/kg.
- Supportive: high-quality ration.

Prognosis

The prognosis is good, but reinfection from highly contaminated environment (including stabling) is likely.

Control

On the basis of reports of lack of efficacy of ivermectin against ascarids, together with the widespread prevalence of benzimidazole resistance in cyathostomins, probably the best control programme for groups of foals is interval dosing with pyrantel and judicious, responsible use of a single dose of moxidectin when horses are 18–20 months of age. On properties breeding/rearing on annual basis the repeated use of the same nursery paddocks should be discouraged.

19.36 TAPEWORMS

Tapeworms are common (up to 80% prevalence); mainly asymptomatic infections.

Aetiology/epidemiology

Three species:

Anoplocephala perfoliata – caecum and distal small intestine (common).
Anoplocephala magna – small intestine.
Paranoplocephala mamillana – small intestine (and stomach) (rare).

- Indirect life cycle.
- Oribatid mites are intermediate hosts.
- Prepatent period is 6–10 weeks.

Pathogenesis

- Colic:
  - Mucosal lesions evident.
  - Possible altered intestinal motility resulting in ileocaecal disease, e.g. ileal hypertrophy or intussusception and some cases of spasmodic colic.

Clinical signs

- Asymptomatic infection in majority of infected horses.
- Colic.

Diagnosis

Faecal examination:

- Flotation/centrifugation methods somewhat insensitive.
- Morphological differences between eggs of *Anoplocephala* spp. and *P. mamillana*.
- Serology (quantitative ELISA) available in some countries.

Treatment

- Oral praziquantel 1 mg/kg, is drug of first choice.
- Double dose of either oral pyrantel embonate (38 mg/kg) or pyrantel pamoate (13.2 mg/kg).

Prognosis

The prognosis is good. If faecal excretion of tapeworm eggs persists suspect *P. mamillana* infection. The rate of decline of serological titre following successful treatment is slow, approximately 5 months.

Control

- If strong indication of specific problem on a property then recommendation is praziquantel once or twice per annum.
- Normal dosage pyrantel salts have fair efficacy against *A. perfoliata* such that tapeworm is incidentally controlled if pyrantel in use for strongyle prophylaxis.

19.37 BOTS

Bots are very common asymptomatic infection of equids.

Aetiology/epidemiology

There are four major species: *Gasterophilus intestinalis*, *G. nasalis*, *G. haemorrhoidalis*, *G. pecorum*.

- Stages within equine stomach are fly larvae (bots) found during winter months in temperate zones.
- Adult flies lay eggs on skin of limbs (*G. intestinalis*) or head (*G. nasalis* or *G. haemorrhoidalis*).
- Self-grooming/licking causes egg to hatch to larvae which penetrate oral mucosa and migrate to stomach where they persist for 10–12 months.
- Larvae are passed in faeces during spring, and adults emerge after 1–2 months pupation on ground.

Pathogenesis

- Essentially non-pathogenic other than causing nuisance/distress when adult flies are egg laying.
- Rarely aberrant migration occurs, e.g. to the central nervous system (Chapter 11).
- G. nasalis has been associated with rectal prolapse in working donkeys in Ethiopia.

Clinical signs

- Mainly asymptomatic except for presence on hairs of distal limbs of eggs which appear as 2–3 mm yellow elongated structures.
- Rarely, neurological signs.

Diagnosis

Diagnosis is not required, and is made incidentally by observation of eggs on limbs, or bots within faeces or stomach.

Treatment

Treatment is not required but is commonly administered during the winter months. The drug of choice is ivermectin, oral 0.2 mg/kg. Organophosphates are also effective, but toxicity and poor palatability limit usage.

Control

- *G. intestinalis* eggs can be induced to hatch by sponging with warm water or grooming or removed by clipping/shaving.
- Wintertime boticidal dosing may be incorporated into strongyle prophylaxis programme.

19.38 LUNGWORM

Donkeys are natural hosts for *Dictyocaulus arnfieldi* infection, and about 30% of UK donkeys have patent, asymptomatic infections. Horses/ponies are abnormal hosts for *D. arnfieldi*: infection is very uncommon and patency rare. Lungworm-infected horses/ponies show signs of chronic cough. Diagnosis suspected from historical information of grazing pastures contaminated by donkeys and confirmed by presence of large numbers eosinophils in bronchoalveolar larvae (Chapter 6). Treatment of choice is oral ivermectin at 0.2 mg/kg.

19.39 *STRONGYLOIDES WESTERI*

S. westeri infection is common in small intestine of foals but is usually asymptomatic. Unusual parasitic features: infection is by either ingestion of larvae in milk or skin penetration of larvae produced by free-living (i.e. non-parasitic) adult worms. Infections may be removed by anthelmintic treatments – higher dosages often required.

19.40 *OXYURIS EQUI*

O. equi (pinworm) infections commonly occur in caecum, colon and rectum and are of low pathogenicity. Adult worms infrequently cause anal pruritus during egg laying giving rise to self trauma of tailhead. Diagnosis is by identification of characteristic eggs in yellow/grey streaks of gelatinous material on perineum. Most anthelmintics have label claim of efficacy against *O. equi*, but anecdotal observations indicate that pinworm infections can be difficult to clear despite dosing with multiple classes of anthelmintic.

19.41 PIROPLASMOSIS (see Chapter 10)

Theileria equi and *Babesia caballi* are the causative agents of equine piroplasmosis. These tick-transmitted piroplasms cause haemolytic anaemia in acute phase of the disease. Clinical signs include fever, anorexia, pale to icteric mucous membranes, tachycardia and weakness. Transplacental transmission can lead to abortions or neonatal piroplasmosis which is invariably fatal. Diagnosis of acute disease is based on microscopic detection of parasites in red blood cells by thorough examination of Wright-Giemsa stained blood smears. Serology is used for detection of carriers. PCR is currently the most sensitive diagnostic technique. Imidocarb and diminazene are the most frequent used babesiacides for the treatment of clinical disease.

19.42 TRYPANOSOMIASIS

Equine trypanosomiases are acute or chronic infectious diseases caused by protozoan blood parasites. Three equine diseases can be distinguished, i.e. nagana, surra and dourine.

Transmission of trypanosomes can be the result of inoculation of infected blood (e.g. during vaccination campaigns), but they are usually transmitted cyclically, mechanically or sexually. Cyclical transmission occurs when the trypanosome undergoes a developmental cycle in the insect vector, the tsetse fly. In mechanical transmission, a haematophagous insect becomes contaminated with an infectious agent during normal feeding and the agent may persist on the mouthparts until the next feed without undergoing any biological development. On the African continent, cyclical transmission is the main mode of transmission for *T. congolense*, *T. brucei* s.l. and, to a lesser extent, *T. vivax*. Mechanical transmission is the only mode of transmission for *T. evansi*, but it can also occur with trypanosome species that are normally transmitted cyclically (i.e. *T. congolense* and *T. b. brucei*) and it is a common mode of transmission for *T. vivax*. *Trypanosoma equiperdum* is transmitted sexually, during copulation, from the stallion to the mare or vice versa.

Nagana

Trypanosomes causing nagana (or tsetse-transmitted trypanosomiasis) belong to the subgenera *Nannomonas* (*Trypanosoma congolense*, *T. simiae*, *T. godfreyi*), *Trypanozoon* (*Trypanosoma brucei brucei*, *T. b. rhodesiense* and *T. b. gambiense*) and *Dutonella* (*Trypanosoma vivax*). With the exception of *T. vivax*, which can also be found outside the African continent, they occur in sub-Saharan Africa.

The clinical signs of nagana in horses vary from lethargy and anorexia to sub-clinical infections:

- Parasitic peaks with large numbers of parasites in the peripheral blood in the early phase of infection are accompanied by pyrexia (rectal temperature may reach 40.5°C, 105°F), tachycardia and tachypnoea.
- Horses with acute nagana are depressed.
- Severe anaemia may develop with increasing weakness and signs of ataxia.
- Small skin nodules (urticaria) and oedematous plaques may form on the flanks.

- The mucous membranes may become icteric as a result of haemolysis.
- Equine trypanosomiasis due to *T. b. brucei* (in a single or mixed infection) is often acute and can lead to death in 2 weeks to 3 months.
- Infections with *T. congolense* or *T. vivax* are more chronic. In the chronic phase of the infection the parasitaemia is usually low, and parasites are difficult to detect using parasitological methods. The anaemia progresses with mucous membranes becoming pale. Horses further lose condition and weight and become extremely weak and show signs of ataxia. The subcutaneous oedema may extend to the head. Horses may become severely emaciated.
- In chronic *T. b. brucei* infections, the disease is often associated with nervous symptoms with ataxia and paralysis.
- Up to 20% of the horses infected with *T. brucei* may develop keratitits and corneal opacity.

Surra

Trypanosoma evansi is the causal agent of surra. Surra occurs mainly in North and Northeast Africa, Latin America, the Middle East and Asia.

There are two forms of surra. An acute form, which occurs mostly in horses and camels, and a chronic form, which is mostly seen in cattle and water buffalo.

- In acute infections, animals suffer from pyrexia, progressive anaemia, loss of general body condition and exhaustion, finally resulting in death.
- In chronic infections, subsequent waves of fever, associated with the parasitaemia peaks, are observed.
- Oedema, mainly of the distal limbs, urticarial plaques and petechial haemorrhages of the mucous membranes can develop.
- The mortality rate in horses can be very high (80–100%).

Dourine

Dourine is caused by infection with *Trypanosoma equiperdum*. The parasite is cosmopolitan, but Western Europe, Australia and the United States are considered to be free from dourine.

Trypanosoma equiperdum causes a chronic infection in horses that can persist for one to two years. Clinical signs usually appear within a few weeks of infection but, in some cases, may not be evident until after several years.

- In mares, the first sign of infection is usually a small amount of vaginal discharge. Swelling and oedema of the vulva develop later and extend along the perineum to the udder and ventral abdomen. Vulvitis and vaginitis with polyuria may occur. Significant abortion losses can be observed in infected herds. In stallions, the initial signs are oedema of the prepuce and glans penis, spreading to the scrotum and perineum and to the ventral abdomen and thorax. Swellings may resolve and reappear periodically. Vesicles or ulcers on the genitalia may heal and leave permanent white scars (leukodermic patches). Conjunctivitis and keratitis are often observed in outbreaks of dourine and may be the first signs noted in some infected herds.
- The second phase of the infection is considered to be pathognomonic for dourine. In this period typical cutaneous plaques can occur with variable sizes ranging from very small to the size of a hand and a thickness of one centimetre. These plaques usually appear over the ribs, although they may occur anywhere on the body, and usually persist for between 3 and 7 days.
- The final phase of dourine is characterized by progressive anaemia and neurological signs. Initially these signs consist of restlessness and a tendency to shift weight from one limb to another followed by progressive weakness and incoordination. Ultimately, paralysis (mainly of the hind limbs), paraplegia and death occur.

Acute equine trypanosomiasis must be distinguished from African horse sickness and anthrax. Repeated examination of blood allows for the differential diagnosis with other causes of severe pyrexia in horses such as equine babesiosis (*Babesia caballi*) and theileriosis (*Theileria equi*).

Diagnosis can be achieved by examination of wet or Giemsa-stained blood films. PCR and serological techniques are also used.

A number of drugs are available to treat trypanosomiasis including isometamidium, diminazene and quinapyramine sulphate. Specific drugs and dosages vary with the pathogen being treated.

19.43 OTHER EQUINE PARASITIC INFECTIONS

Various other parasitic infections occur in the horse and may be associated with clinical disease under certain circumstances, some of which appear elsewhere in the text.

Trichostrongylus axei
Habronema muscae (Ch. 13)
Draschia megastoma (Ch. 13)
Habronema majus (Ch. 13)
Thelazia lacrymalis
Micronema deletrix (Ch. 11)
Probstymaria vivipara
Sarcocystis neurona (Ch. 11)
Hypoderma spp.
Coccidia spp.
Onchocerca cervicalis (Ch. 13)
Parafilaria multipapillosa (Ch. 13)
Echinococcus granulosus (Ch. 3)
Fasciola hepatica (Ch. 3)
Eimeria leuckartii
Trichomonas spp.
Giardia spp.
Klossiela equi
Cryptosporidium spp. (Ch. 20)

FURTHER READING

Jacobs DE (1986) Colour atlas of equine parasites. Baillière, London

Mair TS, Hutchinson RE (2009) Infectious diseases of the horse. Equine Veterinary Education, Newmarket

Sellon DC, Long MT (2007) Equine infectious diseases. Saunders, St Louis, MO

Chapter 20

Diseases of the foal

Ramiro E. Toribio, Margaret C. Mudge

CONTENTS

20.1 Prematurity/dysmaturity 424
20.2 Systemic diseases involving multiple body systems 425
Sepsis 425
Shock 426
Perinatal asphyxia 427
20.3 Diseases of the cardiovascular system 428
Examination 428
Congenital heart disease 428
Acquired cardiac defects 429
20.4 Diseases of the respiratory system 429
Examination 429
Respiratory distress syndrome (RDS) 430
Meconium aspiration 431
Idiopathic tachypnoea and hyperthermia 431
Upper respiratory tract (URT) disease 431
Viral diseases 432
Bacterial disease 432
Interstitial pneumonia 433
20.5 Diseases of the nervous system 433
Examination 433
Ancillary diagnostic testing 434
Central nervous system disorders 434
Cranial nerve disorders 436
Cerebellar disease 436
Ataxia or paresis of the limbs 437
Paresis or paralysis of one limb 437
Other diseases of the nervous system 438
20.6 Musculoskeletal disease 439
Examination 439
Incomplete cuboidal bone ossification 439
Fractured ribs 439
Brachial plexus injuries and tendon ruptures 439
Angular limb deformities 439
20.7 Diseases of the alimentary system 439
Examination 439
Congenital problems of the alimentary system 441
Foal diarrhoea 441
Gastroduodenal ulcers 443
Meconium impaction/retention 443
Abdominal abscesses 443
Obstructive/strangulating lesions 444
20.8 Hepatobiliary diseases 444
Examination 444
Infectious hepatobiliary diseases of the foal 444
Toxic hepatobiliary disease 445
Congenital hepatobiliary conditions 445
Biliary obstruction/cholangiohepatitis 445
20.9 Diseases of the urogenital system 446
Examination 446
Congenital urogenital defects 446
Patent urachus 446
Omphalitis/umbilical remnant infections 446
Ruptured bladder 447
Primary renal disease 447
20.10 Endocrine/metabolic disorders 447
Neonatal hypothyroidism (goitre) 447
Adrenal insufficiency (hypoadrenocorticism) 447
Nutritional secondary hyperparathyroidism 448
20.11 Haemolymphatic and immunological disorders 448
Failure of transfer of passive immunity (FTPI) 448
Severe combined immunodeficiency (SCID) 449
Neonatal isoerythrolysis (isoimmune or alloimmune haemolytic anaemia) 449

© 2013 Elsevier Ltd
DOI: 10.1016/B978-0-7020-2801-4.00020-1

20.12 Miscellaneous conditions 450
Steatitis (yellow fat disease) 450
Glycogen branching enzyme deficiency (GBED) 450
Further reading 450

20.1 PREMATURITY/DYSMATURITY

Gestational length

- Normal gestational length varies from 320 to 360 days.
- Significant variations exist between horses, and gestational length alone is not a good indicator of readiness for birth.
- When assessing maturity, clinical appearance and gestational age must be evaluated in combination.

Definitions

1. *Prematurity*: foal less than 320 days' gestation.
2. *Dysmaturity*: signs of immaturity/prematurity in foals more than 320 days' gestation. Commonly low birth weight, despite normal gestational age.
3. *Postmaturity*: foal greater than 360 days' gestation. Normal to large axial skeletal size, but thin/emaciated.

Risk factors

1. Elective early induction of labour or early caesarean section.
2. Placentitis.
3. Systemic disease in the mare.
4. In utero disease of the foetus.
5. Fescue endophyte toxicity (risk for postmaturity).

Clinical characteristics of prematurity

- Small for gestational age.
- Soft/silky hair coat.
- Floppy ears.
- Increased range of joint motion; lax flexor tendons.
- Abnormal progression through normal events subsequent to foaling (e.g. standing, suckling).
- Weakness or 'floppiness'.
- Domed forehead.
- Poor thermoregulation.
- Increase chest wall compliance.

Clinical characteristics of postmaturity

- Large axial skeletal size, but thin or emaciated.
- Long hair coat.
- Erupted incisors.
- Flexor tendon contracture.

Pathophysiology and diagnostic findings

1. Immature adrenal cortical function:
   - Low cortisol concentrations.
   - Poor response to adrenocorticotrophic hormone (ACTH).
   - Neutropenia, lymphocytosis, decreased neutrophil:lymphocyte ratios.
2. Poor glucose regulation.
3. Poor vascular tone and responsiveness; hypotension is common.
4. Immature renal function, low urine output.
5. Pulmonary immaturity:
   - Decreased lung compliance.
   - Increased chest wall compliance.
   - Weak respiratory muscles.
   - Surfactant deficiency.
   - Thoracic radiographs: a diffuse, 'ground glass' appearance is seen with surfactant deficiency (Figure 20.1).
6. Gastrointestinal immaturity: unable to handle oral diet; poor absorption of colostrum.
7. Musculoskeletal immaturity: lack of cuboidal bone ossification seen on carpal and tarsal radiographs (Figure 20.2).
8. Susceptibility to infection; depressed immunity.

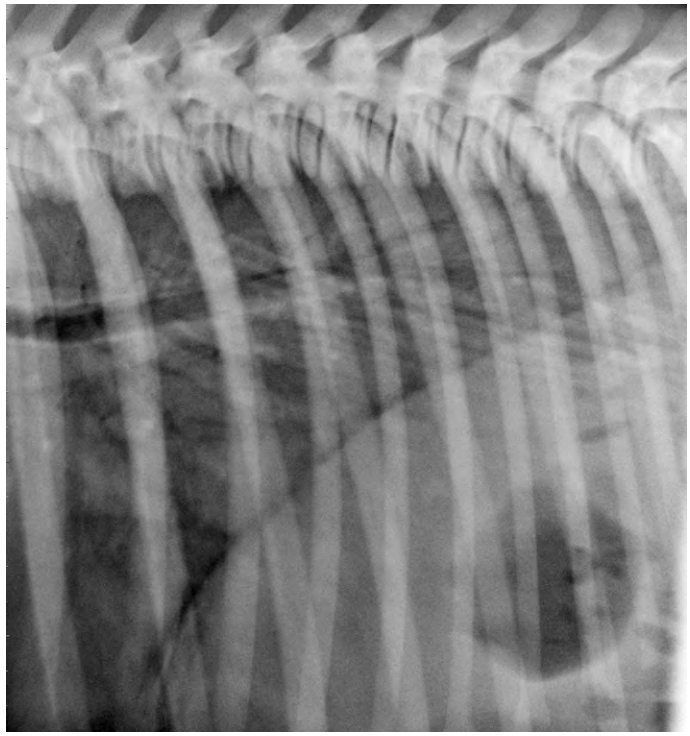


Figure 20.1 Thoracic radiograph of a premature foal (310 days) with evidence of pulmonary immaturity, including atelectasis and lack of alveolar expansion.

Treatment (see Chapter 26)

The treatment of premature foals remains one of the most challenging problems presented to the equine practitioner.

1. Supportive care is of primary importance:
   - Maintain warm environment: blankets, lamps, heating pads, etc.
   - General nursing care to prevent decubital ulcers.
2. Respiratory support:
   - Maintain sternal recumbency.
   - Intranasal oxygen if hypoxaemic.
   - Mechanical ventilation may be needed.
   - Surfactant treatment if deficient.
3. Cardiovascular support if necessary:
   - Intravenous fluids.
   - Vasopressors may be needed.
   - Maintain adequate perfusion.
4. Nutritional support:
   - Intravenous dextrose.
   - Enteral nutrition if gastrointestinal function is mature.
   - Parenteral nutrition if gastrointestinal function is immature or dysfunctional.
5. Adrenocortical axis support: corticosteroids may be used if adrenal insufficiency is suspected.

Figure 20.2 Radiographs of a premature foal (310 days) showing incomplete ossification of carpal cuboidal bones.

6. Physical therapy:
   - Bandages, splints, braces, etc., as needed for musculoskeletal support.
   - Exercise should be limited if hypoplasia of the carpal or tarsal bones is present.
7. Prevention of infection:
   - Determine adequacy of passive transfer of immunoglobulin.
   - Administer colostrum or plasma if necessary.
   - Broad-spectrum antimicrobials.
8. Determine the underlying cause of prematurity if possible:
   - Infection.
   - Placental abnormalities.
   - Cause is often undetermined.

Prognosis

- Survival of foals induced before 320 days is poor.
- Survival greater than 70% is reported for spontaneous birth between 280 to 322 days' gestation.
- Prognosis is better if cause of prematurity/dysmaturity is in utero infection or placentitis.
- Concurrent disease (e.g. sepsis, asphyxia) may reduce the prognosis.
- Respiratory immaturity is often the limiting factor to survival.
- The prognosis is guarded if the neonate does not develop a righting reflex or suckle reflex.

Prevention

Prevention is based upon good management practices and on identification of maternal problems that may influence foetal viability and/or the onset of parturition.

1. Identification and treatment of placental infections. Treatment may include antimicrobials, anti-inflammatory drugs, and progesterone.
2. Avoid induction of parturition unless necessary, as readiness for birth is very difficult to predict.
3. Corticosteroids have been successfully used in humans and sheep to encourage foetal maturation. They have not been shown to be effective in the mare.
4. Monitoring foetal heart rate can give an indication of foetal well-being.
5. To prevent postmaturity, remove mares from endophyte-infected fescue pasture or administer dopamine receptor antagonists.

20.2 SYSTEMIC DISEASES INVOLVING MULTIPLE BODY SYSTEMS

Sepsis

Sepsis is a common cause of morbidity and mortality in the neonatal foal, and is one of the most common reasons for neonatal foal presentation at referral clinics.

Definitions

1. Systemic inflammatory response syndrome (SIRS): characterized by fever, tachycardia, tachypnoea, and leucopenia or leucocytosis.
2. Sepsis: SIRS with a source of confirmed or suspected infection.

3. Septic shock: septicaemia with hypotension refractory to fluid resuscitation.

Aetiology

1. Although sepsis can also be secondary to viral or fungal infection, the term sepsis in the foal generally refers to systemic spread of bacteria through the bloodstream. Infection may arise in utero or postnatally.
2. Failure of transfer of passive immunity (FTPI) may predispose to infection (see Section 20.11).
3. Other risk factors include poor hygiene, maternal illness, prematurity, and immunosuppression.
4. Infections are generally a result of opportunistic organisms from the foal's environment, skin, or the mare's genital tract.
5. Portals of entry into the body include the respiratory tract, umbilicus, gastrointestinal tract, and placenta.
6. Gram-negative infections predominate. The most common organisms vary with the geographical location. Commonly encountered bacteria include:
   - *Escherichia coli*.
   - *Salmonella*.
   - *Klebsiella*.
   - *Enterobacter*.
   - *Actinobacillus*.
7. Gram-positive infections are common, but less frequent than Gram-negative infections. Commonly encountered bacteria include:
   - Beta- and alpha-haemolytic *Streptococcus*.
   - *Staphylococcus* spp.
   - *Clostridium* spp.

Clinical signs

1. Early signs are non-specific and include depression, weakness and decreased suckling.
2. Fever, hypothermia, or normothermia may be present, depending on the severity and stage of the disease.
3. Localizing signs of infection: uveitis, hypopyon, pneumonia, enteritis, meningitis, omphalophlebitis, septic arthritis, and osteomyelitis.
4. Injection, petechiation or ecchymotic haemorrhages on the mucous membranes. Subclinical coagulopathy is common.
5. Severe cardiovascular collapse (shock, stupor, coma), with associated tachypnoea, tachycardia, and hypotension. Bradycardia may be present in the late stages of septic shock.

Diagnosis

1. Blood culture. A positive blood culture is definitive, but a negative culture does not rule out sepsis; periods of bacteraemia may be intermittent and brief.
2. Culture of other body fluids may be beneficial, e.g. transtracheal aspirates, cerebrospinal fluid, faeces, urine, and synovial fluid.
3. History (risk factors) that may predispose to sepsis includes placentitis or vulvar discharge in the mare, maternal problems during gestation, dystocia, low birth weight, and prematurity.
4. Clinical findings: foals with early disease may present with few and subtle signs; advanced disease may progress to shock and coma.
5. Complete blood count (haematology):
   - Leucopenia (neutropenia) is common.
   - White cell count may be normal early in the disease.
   - Differential cell counts may be more useful than total cell count, e.g. relative neutropenia or neutrophilia, presence of band forms and toxic changes.
   - Repeat cell counts are helpful.
6. Other laboratory findings:
   - Hypoglycaemia.
   - Metabolic acidosis.
   - Low plasma immunoglobulin concentrations (FTPI).
7. Sepsis score includes historical, clinical, and clinicopathological information.

Treatment

1. Antibiotic therapy should be initiated as soon as possible. It is often prudent to suspect sepsis until proven otherwise.
   - The initial choice of antibiotics should be broad spectrum with an emphasis on the Gram-negative spectrum.
   - Changes in therapy can be made when and if culture and sensitivity results are available.
   - Therapy should be long-term in an attempt to prevent localization of infection (secondary complications).
2. FTPI should be treated with immunoglobulin therapy (plasma).
3. Supportive care (see Chapter 26):
   - Nursing care is critical.
   - Regulate environmental temperature (provide heat source).
   - Nutritional support; hypoglycaemia is often present; glycogen and fat stores are poor in the foal.
4. Address any underlying infections.
5. Cardiovascular support if needed.

Prognosis

1. Reported survival is in the range 30–80%.
2. Prompt recognition of sepsis and referral for appropriate treatment increases the prognosis.
3. Foals with multiple septic joints have a guarded prognosis for future athletic performance.

Prevention

Good management practices:

- Clean environment.
- Healthy mares.
- Check quality of colostrum and monitor for FTPI.
- Umbilical hygiene.

Shock (see Chapter 26)

Shock is inadequate cellular energy production that results in cell dysfunction and death. Rapid recognition and treatment for shock are important in the prevention of organ system failure. The recognition and treatment of shock in the foal do not differ significantly from those in the adult horse.

Classifications

It is rare for shock to be categorized under a single classification, but the following groupings are useful for categorizing the pathophysiology and treatment targets:

1. Distributive shock.
   - Decrease in systemic vascular resistance resulting from maldistribution of vascular volume.
   - Secondary to sepsis (septic shock), infection, strangulating intestinal lesions, anaphylaxis.
2. Cardiogenic shock.
   - Failure of the heart to act as a pump.
   - Congestive heart failure.
   - Cardiac arrhythmia.
   - Congenital heart disease.

3. Hypovolaemic shock.
   - Blood loss.
   - Third space loss.
   - Severe dehydration.
4. Hypoxaemic shock.
   - Severe pulmonary disease.
   - Anaemia.

Clinical signs

1. Early, compensated phase of shock.
   - Difficult to recognize.
   - Hyperdynamic: injected mucous membranes, rapid capillary refill time.
   - Tachycardia.
   - Tachypnoea.
   - Bounding pulses.
   - Decreased perfusion to gastrointestinal tract and other organs.
2. Decompensated phase of shock.
   - Prolonged capillary refill time.
   - Cold extremities.
   - Tachycardia may change to bradycardia.
   - Irregular respiration.
   - Poor pulse pressure.
   - Abnormal mentation.
   - Lack of urine production.

Treatment

1. Fluid administration is the single most important treatment for hypovolaemic and distributive shock.
   - The goal of fluid therapy is to optimize the vascular volume and restore circulatory function and tissue perfusion.
   - The most appropriate fluid choice is balanced electrolyte solution containing acetate or lactate (e.g. lactated Ringers solution, Hartmann's solution, Plasma-Lyte 148).
   - Polyionic fluids remain effective in restoring vascular volume if the PCV remains greater than 20% and the total protein greater than 35 g/L.
2. Hypertonic solutions cause rapid, transient volume expansion; however, hypertonic saline solution is not recommended, as neonatal foals are unable to handle large amounts of sodium.
3. Colloid fluids (Hetastarch or plasma) may be needed in cases of low colloid oncotic pressure.
4. The rate of administration of fluids is variable; crystalloid fluids may be given in boluses of 20 mL/kg, the boluses being repeated if needed after clinical reassessment.
5. In severe cases as much as 90 mL/kg may need to be administered rapidly.
6. Inotropes and vasopressors may be needed if the foal does not respond to fluid therapy:
   - Dobutamine can be used to increase cardiac output.
   - Norepinephrine can be used to increase systemic vascular resistance and increase stroke volume.
   - Vasopressin.
7. Anti-endotoxic therapy.
   - Polymyxin B.
   - Non-steroidal anti-inflammatory drugs (e.g. flunixin meglumine).
   - Anti-endotoxin plasma.
8. In haemorrhagic shock, whole-blood replacement may be necessary if adequate response is not achieved with crystalloids only.
9. Intranasal oxygen if hypoxaemic shock exists. Mechanical ventilation may be needed in cases of severe pulmonary disease.

Perinatal asphyxia

This is a mutifactorial problem that develops secondary to impairment of tissue oxygen delivery. It is most commonly seen in the neonate shortly after birth when oxygen delivery to the tissues is interrupted (see 'maladjustment syndrome' in section 20.5 of this chapter).

Causes

1. Prepartum.
   - Placental insufficiency.
   - Premature separation of the placenta.
   - Severe maternal disease.
2. Delivery.
   - Dystocia.
   - Caesarean section.
   - Compression/torsion of the umbilical cord.
3. Postpartum.
   - Severe cardiopulmonary disease in the foal.
   - Premature separation of the placenta.
   - Neonatal isoerythrolysis.

Pathophysiology

Two major mechanisms may result in cellular deprivation of oxygen, namely, ischaemia and hypoxaemia. Ischaemia (lack of blood flow) is generally more severe than hypoxaemia, because during hypoxaemia some blood flow and oxygen delivery persist. With a lack of blood flow, anaerobic metabolites are not removed from the tissues which may result in severe local and/or cellular acidosis.

Organ dysfunction

- Central nervous system: encephalopathy associated with cerebral necrosis, oedema and haemorrhage, although consistent cerebral abnormalities have not been reported.
- Renal: acute tubular necrosis.
- Gastrointestinal ischaemic necrosis; necrotizing enterocolitis.
- Cardiovascular and respiratory systems are less commonly affected.

Clinical signs

Signs range from mild depression/transient behavioural abnormalities to severe seizures and generalized organ failure. Many foals do not show obvious clinical signs until 12–24 hours of age. Disorientation, loss of suckle, and tongue protrusion are common physical exam findings.

Resuscitation

1. Airway:
   - Ensure patent airway.
   - Provide intranasal oxygen.
2. Breathing: if the foal is not breathing or if breaths are infrequent or shallow, provide mechanical ventilation with an endotracheal tube or mask (endotracheal tube is preferable).
3. Cardiovascular support and correction of metabolic disturbances (see previous section on the treatment of shock).

Treatment

1. Supportive care.
   - Provide nutrition if unable to nurse.
   - Prevent self-trauma.
2. Seizure control.
3. Prevent further brain injury.
   - Osmotic agents.
   - Magnesium sulphate.

4. Respiratory support if foal has hypoventilation.
5. Antimicrobial drugs to reduce the risk of bacterial translocation and septicaemia.

Prognosis

With appropriate care, prognosis is good (approximately 75%), although will worsen with severe organ dysfunction or concurrent disease (e.g. sepsis).

20.3 DISEASES OF THE CARDIOVASCULAR SYSTEM (see Chapter 7)

Examination

Physical examination

Part of the physical examination of the newborn should include a complete assessment of the cardiac and respiratory systems.

Rate and rhythm

1. Immediately after delivery the heart rate is 60–80 beats/min, increasing to 120–150 within hours to stabilize at 80–100 in the first week of life.
2. Sinus dysrhythmias are frequently found on auscultation and ECG in the immediate postpartum period, probably as a result of increased parasympathetic tone and myocardial hypoxia. Physiological (functional) murmurs are typically crescendo/decrescendo in time (grade 1–3/6), with maximal intensity over the semilunar valves.
3. Other dysrhythmias (ventricular premature contractions, ventricular tachycardia, supraventricular tachycardia) are occasionally auscultated immediately after delivery, but they disappear within 15–30 minutes postpartum.
4. To determine the clinical significance of any dysrhythmia, consideration should be given to the clinical, metabolic and haemodynamic status of the foal.

Murmurs

1. Because of the foal's thin chest, the apex beat is quite prominent, and heart sounds and flow murmurs are louder than in the adult horse. Murmurs are frequent in newborn foals, and in general are not pathological (i.e. functional). In the immediate postpartum period (15 min) most foals have a continuous murmur from blood shunting through the ductus arterious. This represents a transition from the intrauterine fetal circulation in which there is pulmonary hypertension, to an extrauterine circulation in which there is a decrease in vascular resistance from alveoli opening and prostaglandin release.
2. Murmurs are often heard in newborn foals but are not considered pathological unless they persist beyond 4 days of age. Holosystolic ejection-type murmurs are not uncommon and most of the time are physiological (innocent flow murmurs), rather than due to a patent ductus arteriosus (PDA).
3. Innocent murmurs may acquire unusual tones if the foal is in lateral recumbency, or is haemodynamically compromised.
4. Functional closure of the ductus arteriosus occurs shortly after birth in most foals.
5. Murmurs that persist after the first week of age should be further investigated. Likewise, murmurs associated with cyanosis should be evaluated immediately.

Echocardiography

See Chapter 7.

Electrocardiography

See Chapter 7.

Congenital heart disease

See Chapter 7.

Ventricular septal defect (VSD)

This is the most common and important congenital cardiac defect in the foal. There appears to be a genetic basis for VSD as it is more often documented in Arabians, Standardbreds, and Quarter Horses. VSDs are often incidental findings when foals are evaluated for other conditions. Foals with VSDs can appear normal or present for depression, respiratory disease, cyanosis, or failure to thrive. There is a grade 3–5/6 harsh, pansystolic murmur, typically louder on the right side, below the tricuspids valve. A left base ejection murmur from pulmonic stenosis is often present. Older foals may develop congestive heart failure, with or without atrial fibrillation. The intensity of the heart murmurs is not associated with the severity of the VSD. Other murmurs and dysrhythmias may be present if the VSD is associated with other developmental abnormalities. VSDs are present in tetralogy of Fallot, pseudotruncus arteriosus, and occasionally in foals with ASDs and tricuspid atresia.

Patent ductus arteriosus (PDA)

Functional closure of the ductus occurs shortly after birth in most foals (up to 72 hours in some). PDA is a rare cardiac abnormality in foals; however, a left-to-right PDA should not be considered pathological if it persists for a few days postpartum in an otherwise healthy foal. In some cases, a left-to-right PDA may become apparent a few days after birth, in particular in foals with systemic disease. Sick and premature foals with pulmonary hypertension as well as foals of mares treated with prostaglandin inhibitors are prone to PDA. Typical findings of a left-to-right PDA include a continuous, machinery-type murmur, with a thrill over the pulmonary artery.

Atrial septal defect (ASD)

Atrial septal defects are rare in foals and are in general associated with other congenital anomalies. Small ASDs may have no clinical significance (except for performance horses). If the ASD is large, with left-to-right blood shunting, right-sided volume overload ensues, leading to pulmonary over-circulation and atrial fibrillation as the foal ages.

Tetralogy of Fallot

Four characteristic lesions of tetralogy of Fallot include: VSD, right ventricular outflow tract obstruction (pulmonic stenosis), dextroposition of the aorta with overriding, and right ventricular hypertrophy. Foals with this complex pathology are small and ill-thrift. PaO_2 is decreased, and cyanosis is variable. On auscultation there is a loud systolic murmur over the left side from pulmonic stenosis, with a palpable thrill. A machinery-type murmur can be present from continuous blood shunting if there is a PDA. Prognosis is poor.

Tricuspid atresia

Atresia of the tricuspid valve is usually associated with a patent foramen ovale, ASD, VSD, hypoplastic right ventricle, hyperplastic mitral valve and left ventricle. There is cyanosis from right-to-left shunting through the ASD or the foramen ovale. Most of these foals die at an early age from severe hypoxaemia. The ones that survive for several months often have a VSD. These foals are in poor condition, intolerant to mild physical activity, and cyanotic. On echocardiography there is dilation of the right atrium, a small tricuspid valve, and a small right ventricle.

Truncus arteriosus

This condition is rare in foals. Early in development there is a failure of the truncus arteriosus to divide into the aorta and pulmonary arteries. The aorta, pulmonary, and coronary arteries originate from the single truncus. Clinical signs depend on pulmonary blood flow. If pulmonary vascular resistance is low clinical signs might be mild, with no evidence of cyanosis. In contrast, increased pulmonary vascular resistance leads to decreased pulmonary blood flow and hypoxaemia. The foals can be normal or lethargic and in poor body condition. There is a systolic murmur, with or without cyanosis. On echocardiography a single truncus is identified.

Patent foramen ovale

In the foetus, oxygenated blood from the placenta flows back to the heart via the caudal vena cava to the right atrium, and then through the foramen ovale to reach the left atrium. Lung inflation after birth leads to a drop in pulmonary vascular resistance, closure of the ductus arteriosus, and obliteration of the foramen ovale. Anatomic closure in foals occurs between 15 days and 9 weeks postpartum. Clinical signs of patency of the foramen ovale are rare unless associated with other cardiac defects, or with persistent pulmonary hypertension (reversion to foetal circulation) from various pulmonary diseases.

Acquired cardiac defects

Acquired cardiac defects are uncommon in the foal. Inflammatory or degenerative changes may be identified at necropsy. See Chapter 7 for more detailed descriptions.

Pericardial disease

Diseases of the pericardium are rare in foals. Exudate accumulation within the pericardial sac occurs from inflammation of the pericardial and epicardial surfaces. Pericardial effusion can also be present in foals with evidence of systemic inflammation/sepsis.

Myocardial disease

In foals, myocardial disease is associated with ischaemia, hypoxia, septicaemia, metabolic diseases, nutritional deficiencies (white muscle disease), and respiratory diseases. In white muscle disease (vitamin E and selenium deficiency – see Chapter 21), in addition to muscle weakness, dysphagia, and respiratory dysfunction, some foals may develop a dilated cardiomyopathy. The role of viral infections in myocardial disease is unclear. The clinical presentation of myocardial dysfunction is variable but includes murmurs from valvular incompetency, arrhythmias, atrial and ventricular premature depolarization, exercise intolerance, congestive heart failure, and sudden death. Arrhythmias are a common manifestation of myocardial disease. Diagnostics should include electrocardiography, echocardiography, assessment of serum electrolytes, creatine kinase activity (MB isoenzyme if possible), lactate dehydrogenase, and measurement of cardiac troponin I concentrations. If there is dysphagia or muscle weakness, measurement of vitamin E and selenium should be considered. Myoglobin can be present in urine if there is severe myocardial necrosis.

Supraventricular and ventricular arrhythmias

These are more common in adult horses.

Acquired heart murmurs

These may be due to aortic insufficiency, mitral insufficiency, tricuspid insufficiency, or endocarditis in septic foals.

White muscle disease

See Chapter 21.

Pulmonary hypertension/cor pulmonale

This is right ventricular hypertrophy in response to pulmonary hypertension; it can develop in response to severe pulmonary disease, in newborns with reversion to foetal circulation, and in left-sided heart failure. See section on asphyxia above.

Congestive heart failure

Clinical signs may include jugular vein distension, ventral oedema, pulmonary oedema, nasal discharge, and ascites.

20.4 DISEASES OF THE RESPIRATORY SYSTEM (see Chapter 6)

Examination

Respiratory rate

Respiration should begin immediately after birth. The respiratory rate of the neonatal foal is approximately 60–80 breaths/minute in the immediate neonatal period, and declines to 30–40 breaths/minute within 1–2 hours after birth.

Palpation

Palpation of the thorax should be performed to determine whether fractured ribs are present. One must be careful when assessing fractured ribs as pulmonary damage (pneumothorax, haemothorax) and cardiac damage can be induced. Most fractures are in ribs 3–6, and crepitus or a clicking noise can be heard on auscultation.

Thoracic auscultation

Thoracic auscultation is not as reliable an indicator of lower respiratory disease in the neonate as in adults, because often only subtle changes in lung sounds are appreciable on auscultation, even in the presence of very severe pulmonary disease. An elevation of respiratory rate, increased effort, and abnormalities of breathing pattern (nostril flaring, an abdominal component to the respiratory pattern, respiratory stridor) are more appreciable than auscultatory changes in foals with respiratory disease.

Lung sounds

Lung auscultation in the period immediately postpartum reveals crackles as airflow increases in a fluid-filled environment.

Arterial blood gas analysis

Arterial blood gas analysis aids in the characterization of the cause of lower respiratory disorders and cardiovascular disease and assists in the most appropriate course of therapy as well as response to treatment.

Radiography

Radiography provides a good diagnostic method to assess the type and extent of pulmonary disease. Evidence of pulmonary disease in the immediate postpartum period indicates *in utero* infections, septicaemia, peripartum stress (meconium aspiration), or prematurity.

1. Bacterial pneumonias may be characterized by alveolar, bronchoalveolar, and/or interstitial patterns. There is not a pathognomonic radiographic pattern produced by viral diseases, although in adult horses often an interstitial pattern is typical.

2. Ventral distribution of pathological changes may be indicative of bacterial or aspiration pneumonia (bacterial, meconium, milk). If there evidence of aspiration pneumonia, a complete evaluation of the mouth (cleft palate) and upper airways is indicated.
3. A 'cotton ball or nodular' pattern in the mid to dorsal thorax in young foals suggests fungal pneumonia. In older foals (>3 months) this pattern can be consistent with interstitial pneumonia. Foals with interstitial pneumonia can also have a miliary interstitial pattern.
4. Premature or dysmature foals with evidence of pulmonary disease may have pulmonary atelectasis ("ground-glass" appearance), indicating decreased surfactant production and/or bacterial infection.

Depending on the clinical presentation, thoracic radiography should be performed before trans-tracheal aspiration as pneumo-mediastinum may develop after trans-tracheal aspiration.

Ultrasonography

Thoracic ultrasonography has become a routine diagnostic method in foals with evidence of respiratory diseases (respiratory distress, diaphragmatic hernias, haemothorax) and systemic diseases (fever, sepsis).

1. In the foal with pulmonary disease it is preferable to perform ultrasonography with the foal in sternal recumbency or standing. This is particularly important for conditions such as diaphragmatic hernia, haemothorax, and pleural effusion.
2. Rib fractures can be identified as disruptions of the cortical surfaces of the rib, above but not involving the costochondral junction. Hypoechoic regions in the adjacent lung surface indicate pulmonary trauma. Homogeneous, slightly echogenic (cellular) fluid within the thorax is consistent with haemothorax. Pleural effusion is variable, from hypoechoic fluid when cellularity is low, to a hyperechoic fluid that can be homogeneous or filled with cellular debris and fibrin. The presence of gas echoes (hyperechoic) in the pleural fluid indicates either a pulmonary laceration (haemothorax) or a pleuritis/pleuropneumonia with involvement of anaerobic microorganisms.
3. Ultrasonography, like radiography, is not specific in the identification of pulmonary parenchymal disease. Thoracic ultrasonography in young foals with pulmonary disease, regardless of the aetiology, often reveals non-specific findings. Artefacts such as 'comet tails' from excessive reverberation indicate roughening of the pleural surfaces. Cavitary lesions (abscesses) and pulmonary consolidation are often identified by ultrasound.

Transtracheal aspirate

- The goal is to identify the aetiological agent and its antimicrobial sensitivity.
- Cytology can help differentiate the type of inflammatory response.
- The previous use of antimicrobial drugs decreases the success of a positive culture.
- Common sense should be used: in foals with severe pulmonary disease performing a transtracheal aspirate may be contraindicated.
- Perform after radiography.

Endoscopy

Endoscopy is indicated in upper-airway disease (obstruction, laryngeal dysfunction, guttural pouch disease) and malformations (cleft palate, laryngeal and tracheal malformation).

Thoracocentesis

Thoracocentesis is indicated in haemothorax, pneumothorax, and pleural effusion.

- A short intravenous catheter is safer than a needle. Depending on the type of exudates, a larger-bore catheter or a teat cannula can be used.
- Ultrasound guidance is preferable.

Lung biopsy

Percutaneous lung biopsy can be considered in conditions in which there is evidence of pulmonary parenchymal disease but routine diagnostic tests are negative. This is not a procedure routinely performed in young foals as its indications include pulmonary neoplasia and fungal pneumonia. Do not perform in foals with tachypnoea or evidence of poor ventilation, or in foals with infectious pulmonary disease. Haemothorax and epistaxis can develop after lung biopsy.

Respiratory distress syndrome (RDS)

Neonatal RDS, also known as hyaline membrane disease, is a respiratory disorder of premature/dysmature neonates in which there is pulmonary immaturity, with alveolar collapse, impaired alveolar gas exchange, hypoxaemia, hypercapnia, tachycardia, tachypnoea, and cyanosis. Reduced lung compliance and diffuse atelectasis are central to the pathogenesis of RDS.

Predisposing factors

1. Prematurity/dysmaturity.
   - Surfactant is critical for alveolar function. In foals, surfactant production begins at 100–150 days of gestation, being complete in most foals by 290–310 days of gestation, but in some foals it may not be complete until delivery. This process is variable, and gestational age should not be used as an indicator of pulmonary maturation. Foals with normal gestation length and evidence of incomplete organ maturation (respiratory, musculoskeletal) are considered dysmature.
   - Alveolar collapse and subsequent respiratory distress occur from the absence of surfactant.
   - Additional complicating factors in premature/dysmature foals with pulmonary disease include excessive compliance of the chest wall with poor compliance of the lung parenchyma, making the foal work harder during inspiration, which can lead to diaphragmatic fatigue.
2. Decreased respiratory efficiency.
   - Factors that lead to increased demand or fatigue.
   - Failure to establish functional residual capacity shortly after birth.
   - Increased vascular permeability, decreased surfactant, and oedema.
   - Cardiac anomalies (VSD, PDA, tricuspid valve atresia).
   - Gastrointestinal tympany (enteritis, meconium impaction), diaphragmatic hernia.
   - Is not unusual for premature/dysmature foals or foals with peripartum hypoxia to have a decreased respiratory rate and abnormal breathing patterns.
   - Viral or bacterial pneumonia.
3. Dystocia or other problems at or during foaling: premature placental separation, umbilical cord compression, fractured ribs, prolonged parturition, perinatal hypoxia.
4. Any condition that results in prolonged recumbency and atelectasis.
5. Reversion to foetal circulation.

Clinical signs

- Flared nostrils, tachycardia, tachypnoea, and increased respiratory effort.
- Auscultation reveals poor air movement.
- Cyanosis may be present.
- PaO_2 <50 mmHg. $PaCO_2$ >60 mmHg.

Treatment

- Oxygen therapy alone is often not sufficient, and mechanical ventilation may be necessary.
- Cardiovascular support and correction of metabolic disorders.
- Treatment of the underlying condition and surfactant replacement in premature foals.
- Glucocorticoid replacement therapy (hydrocortisone, dexamethasone) – with the rationale of increasing surfactant production – should be considered.

Meconium aspiration

Meconium is the first faecal material of the neonate and consists of ingested and intestinal epithelial cells, amniotic fluid, and mucus. The foetal lung is normally protected from aspiration by the continuous movement of fluids up the trachea. During vaginal delivery, compression of the thorax and closure of the glottis help prevent aspiration.

Aetiology

- Under physiological conditions meconium is not evacuated before birth. In utero, asphyxia, foetal stress, or mechanical factors during delivery may result meconium release into the amniotic fluid.
- Deep gasping or failure of protective mechanisms can result in aspiration.
- Meconium obstructs airways, resulting in air trapping and atelectasis, mechanical irritation, and chemical bronchopneumonia.

Clinical signs

- Meconium-stained foal with yellow-brown discoloration of the hair coat.
- Brown-tinged nasal discharge.
- Staining with meconium does not imply that aspiration has occurred, but therapeutic measures to prevent a potential pneumonia should be considered.

Treatment

- Respiratory and nursing care as needed.
- Antibiotics should be considered as secondary infections often occur.

Idiopathic tachypnoea and hyperthermia

This syndrome is seen in Arabian, Thoroughbred, Belgian, Friesian, and Miniature foals and is more frequent during hot and humid weather. The pathophysiology is unknown but may be related to a problem with the central control of thermoregulation and/or respiratory rate.

Clinical signs

- Sudden onset of clinical signs in an apparently healthy foal.
- Tachypnoea and fever (38.9–42.2°C, 102–108°F). The respiratory pattern may resemble panting, and the rate may be as high as 80 breaths/minute.

Diagnosis

- Other causes of respiratory disease must be ruled out via a complete respiratory evaluation. All other values should be within normal limits.
- Respiratory and systemic infections or pain must be ruled out.
- Haematology, fibrinogen concentrations, and serum chemistry may aid in the diagnosis as in most cases, except for an increase in haematocrit and a stress leukogram, these are normal.
- Metabolic acidosis and other causes of tachypnoea must be ruled out.

Treatment

- Temperature control: provide a cool environment; body clipping; alcohol baths.
- Antipyretics in general are not successful but should be considered.
- Antibiotics should be used if there is suspicion of infection.
- The condition usually resolves spontaneously in days to weeks.

Reversal to foetal circulation

This condition, also known as persistent pulmonary hypertension, is failure by the newborn foal to make the transition from foetal to extrauterine circulation.

Aetiology

There is right-to-left shunting through the foramen ovale and/or the ductus arteriosus, hypoxaemia, hypercapnia, and pulmonary hypertension. In neonates, pulmonary vascular resistance decreases, and pulmonary blood flow increases during and immediately after delivery. As gas exchange increases, pulmonary blood pressure decreases; however, a failure in the mechanisms that regulate the decrease in pulmonary blood pressure leads to hypertension. Reversal to foetal circulation can occur with intrauterine foetal diseases, premature placental separation, prematurity/dysmaturity, peripartum asphyxia, respiratory distress syndrome, meconium aspiration, severe sepsis, and pneumonia.

Clinical signs

There is a tachypnoea, nasal flaring in severe cases, and tachycardia with systolic murmurs of variable degrees. Cyanosis may be present.

Diagnosis

Clinical findings include respiratory acidosis, hypoxaemia, and hypercapnia. On echocardiography a persistent foramen ovale or PDA, with pulmonary artery dilation (with or without right ventricular hypertrophy), is a consistent finding of pulmonary hypertension.

Treatment

Treatment includes oxygen insufflation, correction of the respiratory acidosis, and mechanical ventilation in severe cases. Other treatments that have been proposed or used include vasodilators (prostacyclin, nitric oxide) and bronchodilators. Bronchodilators are controversial as they can induce ventilation/perfusion mismatch, worsening the clinical signs.

Upper respiratory tract (URT) disease

There are many causes of upper airway obstruction in the foal. However, the prevalence is low. Many obstructions of the URT are partial and may go unnoticed until the foal is exercised. A more thorough discussion of URT disease is found in Chapter 5.

Nasal passages and paranasal sinuses

- Deviation of the premaxilla (wry nose).
- Deviation or thickening of the nasal septum.

- Epidermal inclusion cysts.
- Paranasal sinus cysts.

Pharynx and guttural pouches

- Retropharyngeal infection.
- Persistent dorsal displacement of the soft palate.
- Pharyngeal cyst.
- Guttural pouch tympany.
- Choanal atresia.
- Pharyngeal collapse.

Larynx

Obstructive conditions involving the larynx of young foals are uncommon.

- Unilateral or bilateral paresis or paralysis.
- Fourth branchial arch defects.
- Congenital laryngeal web defect.

Trachea

- Tracheal rupture (traumatic).
- Tracheal stenosis and tracheal collapse.

Viral diseases

See also Chapter 19.

Equine herpesvirus 1 (rhinopneumonitis)

Aetiology

- EHV-1 is the main aetiological agent of herpes infections in foals.
- Occasionally associated with EHV-4.
- Infection occurs in utero or in the early postnatal period.
- IMPORTANT: foals suspected of EHV-1 or any viral infection should be placed in quarantine immediately.

Clinical signs

- Foals may be born normal and show acute onset of respiratory disease in the first days of life. A number of foals are born sick, weak, dysmature, depressed, with tachypnoea or respiratory distress.
- The virus infects other systems, and signs of gastrointestinal disease (diarrhoea) and liver disease (icterus) may be present. Leukopenia with lymphopenia is common from lymphoid tissue necrosis.
- Secondary bacterial infections are frequent in foals that live for a few days.

Diagnosis

- Clinical history and findings. It can present as a single sick foal, as an outbreak of sick foals, abortions, or horses with respiratory disease.
- Virus isolation (inconsistent).
- Serology, using paired samples. This may not be practical as most foals die within days. Serology should be performed in the dam and other horses in the premises.
- Polymerase chain reaction (PCR).
- Histopathology: inclusion bodies in cells of the respiratory epithelium, liver, intestinal mucosa and adrenal gland; interstitial pneumonia; hypoplasia or necrosis of the thymus/spleen; and hepatic abnormalities. Immunofluorescence and immunohistochemistry.

Treatment

- General nursing care.
- Antibiotics to prevent secondary bacterial infections.
- Antiviral drugs. Acyclovir and valacyclovir have been used in foals and adult horses. Valacyclovir has a better bioavailability than acyclovir.

Control measures

- Disinfect or dispose of tools and instruments that have been in contact with sick horses.
- Isolate sick foals and horses exposed to sick foals.
- Follow appropriate isolation protocols.
- Vaccinate non-exposed mature horses.
- Designate specific personnel to work with pregnant mares to avoid cross-contamination.

Equine viral arteritis

As with EHV-1, the equine viral arteritis (EVA) virus infects foals at an early age or *in utero*; the mortality rate is high. Most cases of EVA occur in adult horses. Foals with EVA infections are weak, depressed, with evidence of interstitial pneumonia and enteritis. Preventive and medical practices are similar to those for EHV-1; however, treatment is restricted to nursing support; no antiviral drug is effective against the EVA virus.

See also Chapter 19.

Adenovirus

Adenoviruses are considered opportunistic, and infections occur in horses with a compromised immune system. Adenovirus infections are almost limited to Arabian foals with severe combined immunodeficiency. Occasionally critically ill immunocompromised foals may get infected by adenovirus.

Bacterial disease

Bacterial infections are a common cause of pneumonia in the neonate and older foals. In the neonate, pneumonia is commonly associated with septicaemia. Severe pneumonia or septicaemia may lead to pleuropneumonia.

Routes of infection

- Placenta (placentitis); aspiration during birth; umbilicus.
- Inhalation, especially in dusty, poorly ventilated environmental conditions.

Common pathogens

- Gram-negative enteric organisms *(Escherichia coli, Salmonella, Klebsiella, Citrobacter)*.
- *Actinobacillus, Pasteurella.*
- *Streptococcus* spp.
- *Rhodococcus equi* (see Chapter 6).
- Mixed infections often occur.
- Anaerobes may be present in mixed severe infections.

Clinical signs

Variable and dependent on the severity of the infection and organs involved.

- Elevated respiratory rate and fever are highly suggestive. The absence of fever should not rule out bacterial pneumonia.
- Other signs that may or may not be seen: cough, nasal discharge, depression, mucous membrane changes.
- In chronic disease: weight loss, unthriftiness, poor doing.

Diagnosis

- Clinical signs.
- Abnormal auscultation (crackles, wheezes); deep inspiration may be necessary to identify sounds; the absence of abnormal sounds does not rule out pneumonia.
- Radiology: interstitial or alveolar pattern, consolidation, abscess(es), fluid accumulation.
- Ultrasonography is particularly useful to evaluate the superficial lung fields and to assess the pleural space for fluid or exudate accumulation.
- Transtracheal aspirates: cytology and culture.

Treatment

- Appropriate antibiotic therapy based on culture and sensitivity.
- Supportive care as needed which may include:
  - Intranasal oxygen.
  - Environmental temperature regulation.
  - Bronchodilators.
  - Non-steroidal anti-inflammatory drugs.
  - Nutritional support.

Interstitial pneumonia

This is an acute, highly fatal syndrome of foals between 2 and 6 months of age, associated with respiratory distress; most foals die within one week.

Aetiology

- Unknown.
- Many agents have been implicated including viruses (influenza, EHV-1, -4, -5), fungi, plants, toxins, *R. equi*, heat stress, atypical response to bacterial disease, and an exaggerated immune response (hypersensitivity).

Clinical signs

- These foals often are presented for respiratory distress.
- Foals have fever, nasal discharge, increased and harsh lung sounds and wheezing, respiratory effort (expiratory primarily, abdominal component).

Diagnosis

- History, physical examination.
- Haematological changes are non-specific (leukocytosis, neutrophilia), and fibrinogen can be normal or increased.
- Serology is non-specific.
- Ultrasonography may reveal pleural roughening or pulmonary consolidation.
- Radiography is the method of choice when there is diffuse interstitial infiltration. Some foals may also have a bronchoalveolar component (bronchointerstitial pneumonia). On radiography some lesions may look similar to those of fungal pneumonia or metastatic disease (structured pattern, cotton balls).

Treatment

- Oxygen.
- Glucocorticoids: systemic (dexamethasone, prednisolone) or inhaled (beclomethasone, fluticasone). This is central in the treatment for interstitial pneumonia.
- Non-steroidal anti-inflammatory drugs.
- Bronchodilators (clenbuterol, ipratropium, albuterol).
- Appropriate antibiotic therapy based on culture and sensitivity.

Prognosis

- Poor. Most foals with interstitial pneumonia die despite aggressive treatment.
- Foals that recover from acute disease may develop chronic pneumonia.

Necropsy

- Bronchointerstitial pneumonia, with pulmonary consolidation and atelectasis.

20.5 DISEASES OF THE NERVOUS SYSTEM

See also Chapter 11.

Examination

The neurological examination should not be considered in isolation from that of the other body systems as it is part of the general physical examination. This is particularly important in newborn foals where systemic diseases can be associated with neurological dysfunction. As in adult horses, the neurological examination should begin with a good clinical history and assessment of the mental status, progressing to evaluation of the head (position, orientation, movement), cranial nerves, trunk, limbs, and motion. While standing, normal foals have a base-wide stance.

Signalment and history

A good clinical history is central in recognizing neurological disease in foals. Critically ill neonates are often weak, depressed or recumbent, and may show neurological signs secondary to other diseases. These foals must be differentiated from those with primary neurological disease. Important information in the clinical history includes duration of gestation, pregnancy problems, history of premature lactation or placental separation, complications at partum, time to get up and nurse, IgG concentrations, fever, trauma, and duration of clinical signs. Behavioural observation is also crucial as disorientation or lack of maternal recognition can be the first detectable sign of neurological disease in some foals.

Physical examination

An important aspect of the neurological examination is observation (behaviour, response to external stimuli, stance). Healthy foals keep the head in a flexed position, and their response to stimuli is quick, exaggerated, with jerky movements, and some foals may show disorientation.

Cranial nerve evaluation

Evaluation of the cranial nerves is performed as in the adult horse. However, a few normal variations should be kept in mind:

- Immediately after birth the foal should have a strong righting reflex (withdrawal).
- The palpebral reflex is present shortly after birth.
- The menace response does not develop until several weeks of age; however, the foal should withdraw its head from a menacing gesture.
- The pupillary light reflex is slow in foals.
- Auditory response is present immediately after birth.
- Suckle reflex is present 5–15 minutes after birth.

- The capacity to swallow can be evaluated by observing the ability to nurse. Lip and tongue tone and recognition of the udder are also necessary for adequate nursing. If a foal appears to be nursing but large amounts of milk are seen coming from the mouth or nostrils, then a swallowing deficit should be considered (also cleft palate and white muscle disease).
- Facial innervation is assessed by observation (facial symmetry, eye and eyelid positioning, ear positioning) and response to stimulation (reflexes).
- The facial reflex evaluates trigeminal and facial innervation. A delayed response indicates lesions in the brain stem or in the nerve paths, which can be caused by CNS infection, trauma, or perinatal asphyxia.
- The cervicofacial reflex evaluates cervical sensory and facial innervation. A delayed response indicates lesions in the cranial cervical spinal cord, brain stem, or the nerve paths.
- Tongue tone should be strong in newborn foals.
- The slap test is unreliable in foals.

Reflex testing

Testing of reflexes is very useful in localizing a spinal cord lesion.

1. It is important to keep in mind that neonates and young foals have a hyperreflexive response to stimulation (compared to adults).
2. In the pelvic limb, the patellar reflex, flexor, gastrocnemius and cranial tibial reflexes can be tested.
3. The patellar reflex is tested with the foal in lateral recumbency and the limb to be tested supported in relaxed flexion. A brisk extension of the limb (stifle) is expected when the patellar ligaments are struck with the side of the hand. This reflex involves the L4–L5 spinal segments and is mediated through the femoral nerve.
4. The pelvic limb flexor or withdrawal reflex involves the L5–S3 spinal segments and is mediated through the sciatic nerve. Pinching the skin of the distal limb, the coronary band or the bulbs of the heel should elicit a withdrawal of the limb and/or a central recognition of pain.
5. The gastrocnemius reflex is performed by bluntly striking the gastrocnemius tendon and observing for extension of the hock. This reflex involves spinal cord segments L5–S3 and is mediated through the tibial branch of the sciatic nerve.
6. The cranial tibial reflex is performed by holding the limb in relaxed extension and balloting the cranial tibial muscle. The expected response is flexion of the hock.
7. Reflex testing in the thoracic limbs includes evaluation of the flexor and triceps reflexes. The triceps reflex is tested by holding the limb in relaxed flexion and tapping the triceps tendon above its insertion at the olecranon. This reflex is mediated by the radial nerve through the cervical intumescence (C6–T1).
8. The flexor or withdrawal reflex of the thoracic limb is mediated through the last three cervical and first two thoracic spinal cord segments. The expected response is a flexion of the digit, carpus, elbow and shoulder. A response to pain should be observed.
9. Thoracolumbar lesions can be localized by evaluating the cutaneous reflex along the lateral body wall:
   - Gentle pinching or poking of the skin along the lateral thorax elicits a twitching of the cutaneous trunci muscle.
   - The sensory input is carried to the spinal cord at the level of stimulation. It then travels cranially in the spinal cord white matter to the last thoracic and first cervical segments and synapses with the lower motor neurons of the lateral thoracic nerve.
   - The lateral thoracic nerve innervates the cutaneous trunci muscle. Damage to any portion of this pathway results in an absence of the cutaneous trunci reflex.
10. The perineal reflex evaluates the last sacral segments and the caudal spinal cord segments. Stimulation of the perineal area results in flexion of the tail and closure of the anus.

Gait

The gait of the foal should be examined for the presence of weakness, stiffness, and ataxia. The normal newborn foal has a choppy, hypermetric gait, and while standing it has a base-wide stance.

Ancillary diagnostic testing

Additional ancillary testing of the neurological patient includes blood analysis, radiography, cerebrospinal fluid analysis, myelography, computed tomography (CT), magnetic resonance imaging (MRI), electromyography, and electroencephalography (see Chapter 11).

Central nervous system disorders

Neonatal maladjustment syndrome (NMS)

The NMS represents a broad term associated with clinical manifestations of CNS dysfunction in newborn foals. Other terms used include dummy foal, wanderer, barker, hypoxic ischaemic encephalopathy, peripartum asphyxia syndrome, and neonatal maladjustment syndrome. NMS is the most common CNS disturbance in newborn foals. It is mportant to mention that NMS often manifests with involvement of other organs (gastrointestinal, renal).

Clinical signs

Clinical signs are variable and range from disorientation and inappropriate nursing behaviour to seizures and coma. Foals may wander, exhibit blindness, star gaze, or infrequently produce abnormal vocalizations. Some foals may appear normal at birth but exhibit CNS signs at 12–48 hours of age.

Diagnosis

Specific criteria have not been established. However, it is generally the working diagnosis for a foal exhibiting CNS signs without any other specific neurological disease.

- The history often suggests a problem during late gestation, parturition or immediately post-foaling.
- Blood analyses are non-specific. Elevated creatinine may suggest placental insufficiency.
- Sophisticated neurodiagnostic techniques (CT scans and/or MRI scans) may be helpful, if available.

Aetiopathogenesis

The aetiology remains unclear. It has been proposed that impaired oxygen delivery before, during or after birth, with subsequent hypoxic–ischaemic injury to the CNS, is the primary cause of the syndrome.

- Intracranial haemorrhage as a result of excessive pressures during foaling may be a causative factor.
- Impaired oxygen delivery may result from placental problems before birth (placental separation, placentitis, maternal diseases), problems during birth (dystocia, umbilical cord compression, etc.) or postpartum events (isoerythrolysis).
- Most of the brain damage occurs subsequent to the hypoxia during the period of reperfusion (12–24 hours after the initial insult). Oxygen deprivation leads to abnormal membrane ion gradients, with excessive sodium and calcium influx (cytotoxic).

In addition, the production of free radicals (reperfusion injury), activation of N-methyl-D-aspartic acid (NMDA) receptors by excitatory neurotransmitters (glutamate) with subsequent calcium influx, and decreased NMDA receptor blockade by magnesium have been proposed. These events lead to neuronal death.

- Foals with NMS are prone to sepsis for several reasons. Often they do not nurse because of neurological dysfunction, missing the timing for immunoglobulin absorption. If the NMS is the result of premature placental separation it is also likely that there was premature lactation and therefore no colostrum. If the cause was the result of hypoxia during or immediately after birth it is also possible that there was hypoxia to the intestinal tract, leading to epithelial denudation and bacterial entrance. A combination of these processes is common.

Treatment

Treatment is symptomatic and supportive:

- Control seizures. Diazepam (5–10 mg IV) or midazolam (0.1–0.4 mg/kg IV) are good initial choices as they work fast and are safe. Long-acting anticonvulsants such as phenobarbitone or phenytoin may be necessary as the duration of benzodiazepines is short. Phenobarbitone (2–10 mg/kg IV, over 15–20 min) is administered until seizures are controlled; it may not be necessary to repeat this for 12–24 hrs as it has a long half-life. Phenytoin is administered at 5–10 mg/kg IV q 6 h.
- Non-steroidal anti-inflammatory drugs (flunixin meglumine), antioxidants (vitamin E), free-radical scavengers (DMSO), magnesium sulphate, mannitol, thiamine, and more recently ketamine have been recommended.
- Fluid and nutritional support (fluids should be administered cautiously as excessive fluid therapy may worsen cerebral oedema).
- Supportive care, including antimicrobials.

Prognosis

Prognosis is dependent upon the degree of hypoxic damage:

- If severe, rapid death may ensue.
- If neurological signs are not severe or can be controlled, then the prognosis is good if nursing care is adequate and secondary complications can be avoided.
- Additional complications from systemic hypoxia, including gastrointestinal disease (enterocolitis, ileus, septicaemia) or renal disease may complicate the outcome.
- In general, the athletic future of foals that recover is not impaired.

Developmental disorders

See Chapter 11.

Hydrocephalus, anencephaly, spina bifida, meningomyelocele, hemivertebra.

Bacterial meningoencephalitis (septic meningitis, suppurative meningitis) (See Chapter 11)

- Meningitis may occur in foals with septicaemia (haematogenous).
- Often these foals have a failure of transfer of passive immunity.
- The clinical signs are variable but may include fever, depression, weakness, ataxia, disorientation, seizures, and cranial or spinal-cord nerve deficits. Similar signs may be present in foals with other neurological conditions (hypoxia, trauma).
- Aetiology: *Escherichia coli*, *Salmonella* spp, *Actinobacillus* spp, *Enterobacter* spp, *Klebsiella* spp, *Pseudomonas* spp, *Streptococcus* spp and *Rhodococcus equi* in older foals.
- The incomplete blood–brain barrier in the neonate favours bacterial invasion.
- The presence of bacteria in the CNS leads to inflammation and development of the clinical signs.
- Diagnosis: clinical findings and collection of cerebrospinal fluid (CSF) for cytology and culture are necessary for an appropriate diagnosis and subsequent treatment.
- Treatment: medical support, control seizures (diazepam, midazolam, phenobarbitone), non-steroidal anti-inflammatory drugs (flunixin meglumine, ketoprofen), and antimicrobials. Proper antimicrobial selection depends on bacterial sensitivity and antimicrobial properties. Most infections are caused by Gram-negative bacteria. Aminoglycosides and penicillins have poor CNS penetration unless there is extensive meningeal inflammation; third-generation cephalosporins (ceftiofur, ceftriaxone, ceftazidime), chloramphenicol, and trimethoprim–sulfonamides have good CNS penetration. If leucocyte count in the CSF is elevated, regardless of culture and Gram-staining results, a good initial option is a third-generation cephalosporin. The use of glucocorticoids remains controversial but should be considered in foals with evidence of severe inflammation.

Viral encephalitis (EHV-1)

EHV-1 is a rare cause of diffuse cerebral disease in the neonate.

Vascular disorders

Meningeal or parenchymal haemorrhages of the CNS can result in a variety of neurological signs depending on the location and severity. The aetiology is variable and includes metabolic disturbances, trauma, hypertension, endothelial dysfunction (sepsis, vasculitis), and blood-flow abnormalities.

Liver disease

See Chapter 3 and the hepatobiliary diseases section, below. Relevant diseases include hepatic encephalopathy, Tyzzer's disease, and porto-systemic shunts.

Juvenile idiopathic epilepsy (See Chapter 11)

Juvenile idiopathic epilepsy is a neurological condition of Arabian foals. Most foals show clinical signs in the first week of life, but it has been reported in foals up to 6 months of age. Typical signs include disorientation, tonic and clonic motor activity, unconsciousness, and blindness. The aetiology of idiopathic seizures remains unknown, but is more often reported in Egyptian Arabian foals, suggesting that it is heritable. Seizures can be controlled in the short term with benzodiazepines, but for the long term phenobarbitone or potassium bromide should be considered. Seizures usually cease by the time the horses is one year old.

Lavender foal syndrome

Coat colour dilution lethal, also known as the lavender foal syndrome (LFS), is a genetic disease of Egyptian Arabian foals characterized by inability to stand, weakness, recumbency, tetany, epilepsy, and death. The coat colour in these foals ranges from a dilute lavender to a pale chestnut. Most of the foals show neurological signs immediately after birth. This is in contrast to juvenile idiopathic epilepsy, another neurological disease of Egyptian Arabian foals, in which clinical signs show days or months after birth, and have no coat colour dilution. The other difference is that LFS is lethal, whereas most foals with idiopathic epilepsy survive with proper treatment.

LFS is a pathological condition of autosomal recessive inheritance caused by an inactivating mutation of the myosin Va (*MYO5A*) gene. Myosin Va is important in the movement of organelles and secretory granules in neurons and in melanosome transfer to keratinocytes. LFS should be suspected in any Arabian foal presented for weakness, inability to stand, neurological disease, and coat colour dilution. There is no treatment, and these foals die hours or days after birth or have to be euthanized.

Narcolepsy–cataplexy

- Narcolepsy, also known as the fainting disease, is a neurological disorder characterized by cataplexy (acute loss of muscle tone) and sleep. In foals, the condition primarily occurs in Suffolk horses (Suffolk Punch) and Shetland ponies, indicating that it is inherited. Occasionally it may present in other breeds. It is believed that complete inhibition of skeletal muscle tone during acute sleep episodes is associated with abnormal function of the sleep–awake centre. In horses, episodes of cataplexy can be triggered by grooming, initiation of various activities, changes in environment, etc. Information on the pathogenesis in foals or horses is limited.
- In general, clinical signs present in foals several months old. The foal may show weakness, or collapse. Sleep cycles can last from seconds to 10–15 minutes, and foals are normal between episodes.
- The diagnosis is clinical as most laboratory and imaging tests are non-diagnostic. Electroencephalography may assist in confirming the diagnosis, although this is a low-yield test. The physostigmine provocation test (0.05–0.1 mg/kg IV over 10 minutes) may precipitate episodes of cataplexy 3–10 minutes after administration in adult horses, but the effect in foals is unknown. Any condition associated with acute collapse, in particular heart disease, should be considered. Cerebral damage from peripartum hypoxia may produce similar signs in some foals. Imipramine (0.5–1.0 mg/kg IV) has been used in adult horses to treat narcolepsy with variable success. Atropine sulphate (0.04–0.08 mg/kg IV) may provide temporary control for up to 24 hours. Foals with early-onset narcolepsy (Thoroughbreds and miniature breeds) may recover, whereas in ponies the frequency of episodes may decrease although the condition persists throughout life.

Other causes of CNS disease

See Chapter 11.

- Toxins.
- Trauma.
- Rabies.
- Parasite migration.
- Heat stroke.
- Lightning strike.
- Leukoencephalomalacia.
- Metabolic disorders.
- Equine degenerative myeloencephalopathy (EDM).
- Tetanus.

Cranial nerve disorders

Visual dysfunction

1. Assessment of vision. Assessment of vision in the neonate is difficult as the menace response is not easy to evaluate. Signs of blindness may include attempts to nurse from incorrect sites, walking into obstacles, and signs of disorientation.
2. Causes of blindness.
   - Diffuse cerebral disease can result in bilateral blindness, usually with other signs of cerebral dysfunction.
   - Postictal blindness may occur subsequent to seizures.
   - Acute trauma, leading to cerebral lesions or optic nerve compression or avulsion.
   - Severe uveitis in septic foals may lead to blindness.
   - Congenital diseases, including stationary blindness, optic nerve hypoplasia, ocular dysgenesis, retinal dysplasia, microphthalmia, and cataracts.
3. Congenital stationary night blindness. This is a poorly understood condition that is most commonly seen in the Appaloosa but has been reported in other breeds including Thoroughbreds and Paso Finos.
   - Visual impairment varies from poor vision during reduced light conditions during the day with blindness at night.
   - A history of poor vision and an abnormal fundus allow for a presumptive diagnosis.
   - Other clinical signs may include dorsal medial strabismus, nystagmus, mild microphthalmia and holding the head in a 'stargazing' position when attempting to visualize objects.
   - In the Appaloosa this disease appears to be recessive and associated with the leopard complex of coat patterns, thus supporting a defect in neural crest cell (rods, melanocytes) migration during development.
4. Anterior segment dysgenesis. This is a condition described primarily in Rocky Mountain and Saddlebred horses, in which there is congenital megalocornea, ciliary and retinal cysts, hypoplastic iris, cataracts, and retinal detachment. Most of the time this condition goes unnoticed unless signs of visual impairment are detected.

Other cranial nerve abnormalities

See Chapter 11.

Cerebellar disease

See Chapter 11.

1. Cerebellar abiotrophy.
   - Cerebellar abiotrophy is the most common cerebellar disease in foals. It is a congenital disease of Arabian foals, with no gender predilection. Most foals develop clinical signs after 1 month of age, rarely immediately after birth. At birth the cerebellum appears to be normal in size, with normal neurons; however, as the foal ages there is degeneration and depletion of the Purkinje cells. The exact mechanism for the development of cerebellar abiotrophy is unknown, but decreased trophic stimulation and free radical injury have been proposed. In foals with severe disease the cerebellum is small.
   - Clinical signs include head tremors that worsen with external stimulation or blindfolding (intentional tremors), progressive ataxia, wide-based stance and gait, dysmetria with a hypermetric goose-stepping gait, hypometria, spasticity, inability to perform precise movements (apprehension, nursing), delayed or absent menace response, falling over, and inability to stand in severe cases. There are no changes in mentation, and nystagmus is rarely observed. These foals eventually have to be euthanized.
   - The diagnosis is clinical and post-mortem. The cerebellum can be normal or small in size, and on histology the Purkinje cells are absent or decreased in number. A genetic test to

detect carriers is currently available (University of California Davis Genetics Laboratory).
- A syndrome similar to cerebellar abiotrophy has been reported in Gotland ponies, Australian ponies, and Oldenberg foals.

2. Other cerebellar diseases.
   - Cerebellar hypoplasia has been reported in Haflinger, Arabian and Thoroughbred foals. A condition similar to the Dandy–Walker syndrome (midline cerebellar defect with cystic dilatation of the fourth ventricle, partial absence of the vermis) has been documented in Arabian and Thoroughbred foals. These foals are abnormal from birth, with difficulty rising, seizures, and absent suckle reflex, and their forehead may be domed. A progressive degenerative cerebellar disease has been reported in Thoroughbred and Paso Fino foals.

Ataxia or paresis of the limbs (See Chapter 11)

Different conditions can be associated with ataxia or paresis in foals, including infections, trauma, and brain, spinal cord, and nerve diseases.

Spinal cord and vertebral trauma

Trauma to the vertebral column, with or without vertebral fracture, is relatively common in the foal.

- Physical examination may identify external signs of trauma, such as skin abrasions, swellings or haematomas, epistaxis and haemorrhage from the ears. However, it is not uncommon to find no external evidence of trauma. With cervical trauma, neck pain may be present.
- Neurological signs may include varying degrees of ataxia and/or paresis to paralysis. A combination of thoracic limb extensor hypertonia and pelvic limb paralysis (Schiff-Sherrington phenomenon) has been reported with caudal thoracic vertebral fracture.

Vertebral osteomyelitis

Aetiopathogenesis

This disorder involves the systemic spread of bacteria which localize in the vertebrae, leading to vertebral pain and spinal cord compression as the condition progresses. Meningitis may be present. The history includes a previous infection, although this is often not noticed. In foals with septicaemia, Gram-negative bacteria (*Escherichia coli*, *Salmonella* spp.) are the most common cause of osteomyelitis. In older foals, Gram-positive organisms (*Streptococcus*) are more likely to be isolated. *Rhodococcus equi* is a common cause of osteomyelitis in endemic areas.

Physical examination

There may be a stiff neck, neck or back pain, fever, depression, and/or unthriftiness.

Neurological examination

- If spinal cord compression is present then paresis/paralysis or ataxia may be seen.
- Areas of hypo- or hyperreflexia, patches of sweating, lordosis, kyphosis, or scoliosis may be seen.

Diagnosis

- Radiographs may reveal bony changes; however, osteomyelitis is often not identified early in the disease.
- CT or MRI, if available, are great imaging modalities.
- Cerebrospinal fluid analysis may identify a neutrophilic pleocytosis.
- Scintigraphy may aid in localizing the lesion.

Occipitoatlantoaxial malformation (See Chapter 11)

This condition is primarily reported in Arabian foals but it also has been reported in Quarter Horses, Appaloosas, Standardbreds, Morgans, and miniature horses. It appears that the condition in Arabian horses is congenital. The atlas is fused to the occipitus, and its caudal aspect is rounded, while the axis looks more like the atlas. Clinical signs are primarily the result of spinal cord compression, typically spastic paresis, and the neck can be flexed or extended, but in a fixed position. On palpation there is no movement of the atlanto-occipital joint. Radiography reveals a malformation, and in general myelography is not necessary.

Myoclonia / myotonia

A form of stimulus-induced inherited myoclonia has been documented in Peruvian Paso Fino foals. Inactivation of the glycine receptor is believed to be responsible for this condition. Congenital myotonia occurs in a number of equine breeds, including Quarter horses and Ponies. A mutation in a chloride channel was documented in a New Forest pony.

See also Chapter 11.

Paresis or paralysis of one limb

See also Chapter 11.

Plexus and peripheral nerve trauma

Aetiology

Trauma to the brachial or lumbosacral plexus occurs most frequently at birth (during difficult deliveries) but can be caused by other physical accidents. The clinical signs are associated with the location of the lesion, type of injury, number of nerves involved, and severity of nerve damage (neuropraxia, axonotmesis, and neurotmesis). Neuropraxia is a mild nerve injury (concussion), with interruption of nerve conduction, but is reversible (days, weeks). In axonotmesis there is loss of axonal continuity with myelin layer disruption (more severe than neuropraxia), leading to Wallerian degeneration, with loss of motor and sensory function that can be reversible or not (weeks to months). Neurotmesis is a severe lesion leading to denervation.

Clinical signs

Signs include unilateral or bilateral hypotonia with depressed or absent reflexes, limb dragging, inability to get up or stand. If there is major involvement of motor innervation, muscle atrophy can become evident in 1–2 weeks.

Diagnosis

This is by physical examination, neurological examination, and assessment of motor and sensory activity. Signs include unilateral or bilateral hypotonia with depressed or absent reflexes, limb dragging, or inability to get up or stand. On electromyography, prolonged insertional activity with fibrillation potentials indicates muscle denervation of >7 days. Nerve conduction can also be evaluated in peripheral nerves. Depending on the location of the lesion, radiography should be considered to assess the extent of the lesion and to rule out fractures.

Treatment

- Non-steroidal anti-inflammatory drugs, glucocorticoids in the acute stage.

- Physical therapy.
- Splints may allow the foal to bear weight and prevent limb contracture and additional trauma.

Prognosis

The prognosis is dependent upon the location and severity of trauma. Spinal root injury has a worse prognosis than plexus or peripheral nerve injury.

Other diseases of the nervous system

See Chapter 11.

Botulism (shaker foal syndrome)

See Chapter 11.

Aetiology

- Botulism is a progressive muscular weakness caused by a Gram-positive, anaerobic, exotoxin-producing rod, *Clostridium botulinum*, a microorganism ubiquitous to the soil.
- Botulinum toxin is a neurotoxin that blocks the release of acetylcholine from the motor endplate at the myoneural junction causing muscular paralysis.
- There are seven neurotoxins identified: A, B, C, D, E, F, and G. In the USA, particularly in the mid-Western states and mid-Atlantic seaboard, most cases are caused by type B toxin. Type A botulism is primarily reported on the west coast. Type C is associated with decomposing carcasses and is less frequent. Types B, C and D have been reported in Europe.
- In foals, the disease results from the ingestion of preformed toxin or toxin production from the organism within the body. Necrotic lesions within the gastrointestinal tract may allow colonization; umbilical infections as well as wounds are other possible foci of infection.

Pathogenesis

- In foals, typically there is ingestion of spores which then produce toxin in the gastrointestinal tract. Unlike adult horses, the toxin can be detected in the faeces of 30% of foals. The toxin gets access to terminal ends and may travel to the CNS by retrograde axonal transport.
- *C. botulinum* toxin comprises a light and a heavy chain. The light chain is an endopeptidase that interferes with acetylcholine release by inhibiting proteins necessary for the fusion of synaptic vesicles to the neuromuscular junction. The binding of the toxin is irreversible, and therefore resolution of the clinical signs is dependent on the synthesis of new endplates.

Clinical signs

- Foals at any age can be infected, but they are usually more than 1 week of age.
- Presentation varies with the amount of toxin ingested or elaborated.
- The toxin produces a flaccid neuromuscular paralysis.
- Affected foals are centrally bright and alert. The initial signs include a stiff gait, with the foal spending only short periods standing, and/or dysphagia (milk dripping from the sides of the mouth). The foal may progress to trembling or shaking when standing to total paralysis. Some foals may present for respiratory disease from aspiration pneumonia. If severe, respiratory failure and rapid death may occur from diaphragmatic paralysis.

Physical examination

- Foals are bright and alert and typically afebrile unless there is a secondary infection.
- There is a flaccid neuromuscular paralysis, trembling, poor eyelid, tongue and tail tone.

Diagnosis

- Presumptive, based on clinical signs.
- Demonstration of *C. botulinum* spores or the preformed toxin in the gastrointestinal content would be ideal, but this rarely is possible despite clinical evidence of the disease.
- Attempts to identify the toxin in peripheral blood have been unsuccessful.
- Toxin can be identified in necrotic wounds.
- The mouse bioassay is the most sensitive method available to look for the presence of the toxin in serum.

Differential diagnosis

- Any neurological disease associated with weakness.
- White muscle disease.
- Electrolyte abnormalities.
- Cleft palate (no evidence of muscle weakness).
- Hyperkalaemic periodic paralysis.

Treatment

- Potassium or sodium penicillin (22 000–44 000 IU/kg IV QID) or metronidazole (20 mg/kg PO TID–QID). Caution must be taken when using drugs that may potentiate neuromuscular blockade (aminoglycosides, procaine penicillin, tetracyclines). Aminoglycosides may be necessary in foals with secondary infections.
- Good nursing and supportive care are essential. A nasogastric tube may be placed for feeding.
- Polyvalent antitoxin greatly increases the chance of survival, although it is very expensive. Alternatively, in regions where the most common type of botulism is known, monovalent antitoxin is a great alternative.
- Assisted ventilation may be necessary if respiratory paralysis has developed.

Prognosis

- Dependent on the severity. If severe and treatment is not aggressive, mortality is high.
- Mild cases can recover without antitoxin therapy, but antitoxin enhances the chance of survival.
- Severe cases can have a good prognosis if therapy is aggressive (mechanical ventilation, antibiotics, nutritional support, and meticulous nursing care).

Prevention

- Vaccination of mares with *C. botulism* toxoid to provide colostral immunity in endemic areas. A type B toxoid is available in the United States. Type C and D toxoids have been evaluated in Switzerland. It is recommended to vaccinate mares in the last trimester of gestation with two additional doses 2–4 weeks apart.
- If mares are unvaccinated then foals can be vaccinated early in life.

Tetanus

See Chapter 11.

20.6 MUSCULOSKELETAL DISEASE

See Chapters 15, 16 and 17.

Examination

1. In the newborn foal, examination of the musculoskeletal system evaluates maturity and/or gestational age. Floppy ears, absence of hair or a silky hair coat, and joint laxity can indicate immaturity, prematurity or dysmaturity.
2. Assessment for birth trauma involves palpation for rib, mandible, or limb fractures as well as any soft tissue injury.
3. Congenital abnormalities include flexural and angular limb deformities, lateral luxation of the patella, prognathia/brachygnathia, wry nose, polydactyly, kyphosis, and scoliosis.
4. Careful palpation for lameness, joint effusion, physeal pain/swelling, and heat over joints is critical as neonates are at risk of septic arthritis.

Incomplete cuboidal bone ossification

Radiographs are needed to diagnose incomplete ossification (Figure 20.2). Manage with strict exercise restriction until there is evidence of adequate ossification. Splints or casts may be used for cases of severe laxity, but do not incorporate the foot in the splint.

Fractured ribs

Fractured ribs (dystocia or trauma at birth) are one of the more frequent injuries in the newborn foal, although brachial plexus injuries and ruptured gastrocnemius tendons/muscle are seen occasionally.

- Fractured ribs may be bilateral but usually involve one side of the rib cage only.
- A single rib may be fractured, but multiple ribs are usually involved.
- Dislocations at the costochondral junctions may occur, and are of less clinical concern as the surrounding structures are usually left undamaged.
- Multiple dislocated rib fractures are easily identified, causing pain, swelling, and/or a flail chest.
- Careful palpation of the thorax can identify individual and non-displaced fractures. Swelling or crepitus may be present. Auscultation may reveal a rubbing sound.
- Fractured ribs may be difficult to identify radiographically. Ultrasound is useful to assess whether fractures are displaced, and to monitor healing.
- The importance of identifying fractured ribs should not be underestimated as they can result in numerous life-threatening complications.
- Internal fixation may be needed for multiple rib fractures or displaced rib fractures that cause respiratory compromise.

Brachial plexus injuries and tendon ruptures

Clinical signs of brachial plexus injuries vary with the severity of injury. However, they generally result in some degree of radial nerve paralysis. The foal is unable to bear weight on the limb and is unable to extend the carpus or digit. Rupture of the gastrocnemius muscle can occur during foaling and is identified as a swelling above the hock in the area of the gastrocnemius muscle. A complete disruption of the gastrocnemius allows flexion of the hock when the stifle is in an extended position. Ultrasonography identifies muscle tearing.

Angular limb deformities

See Chapters 16 and 17.

Angular limb deformity (ALD) is deviation of the limb from the normal axis in the frontal plane.

- Carpal valgus is the most common form of ALD.
- Perinatal factors include soft-tissue/ligamentous laxity and incomplete ossification of the cuboidal bones.
- Developmental factors include unbalanced nutrition, uneven weight-bearing, and excessive exercise or trauma.

Flexural limb deformities

Flexural limb deformity (FLD) is deviation of the limb from the normal axis in the sagittal plane and can be either hyperflexion or hyperextension.

- FLD may be due to congenital or acquired factors.
- Abnormalities at the distal interphalangeal, metocarpophalangeal, metatarsophalangeal and carpal joints are most common.
- Treatment for hyperextension consists of controlled exercise, protection of soft-tissue structures, and shoes with heel extensions.
- Treatment for hyperflexion in the neonatal foal consists of physical therapy, bandages, splints or casts, and oxytetracycline.

Septic arthritis

Septic arthritis in foals is most often haematogenous in origin and results in joint effusion, pain, and lameness.

- Joint fluid analysis usually reveals increased leukocyte count (>10 000/μL, and typically >30 000/μL with >90% neutrophils) and elevated total protein (>4 g/dL).
- Cultures of synovial fluid and blood are recommended.
- Treatment consists of joint lavage, local and systemic antimicrobials, and pain management.

Hyperkalaemic periodic paralysis

See Chapter 21.

20.7 DISEASES OF THE ALIMENTARY SYSTEM

Examination

Physical examination

1. External abdominal palpation. This can be of diagnostic benefit in small foals as impactions or other masses may occasionally be palpated.
2. Abdominal auscultation. Gastrointestinal sounds (borborygmi) can normally be heard on both sides of the abdomen. Fluid sounds may indicate enteritis. Gas distension can be confirmed by percussion with a resulting 'ping'.
3. Ballotment can be useful for determining the presence of free abdominal fluid.

Clinical pathology

Leukopenia, hypoglycaemia and metabolic acidosis are common findings in foals with enteritis.

Nasogastric intubation

Nasogastric intubation should be part of the assessment in foals with colic with or without abdominal distension. The gastric content can

be variable, from a normal white–yellowish material to a dark–haemorrhagic content in foals with severe enteritis. In some instances the gastric content can be submitted for bacterial culture (*Salmonella* spp., *Clostridium* spp.).

Radiography

1. Useful in cases of gastric reflux, abdominal distension, or signs of colic.
2. Abdominal radiographs are most easily taken with the foal in a standing position, although left or right lateral recumbency views are possible in small foals.
3. Abnormal radiographic findings:
   - Gas/fluid distension: ileus or intestinal displacements with resultant obstruction (intussusception, meconium impaction, congenital malformations).
   - Free peritoneal fluid: fluid lines can often be seen in standing radiographs.
   - Free peritoneal gas (pneumoperitoneum): may indicate gastrointestinal rupture.
4. Contrast studies are useful in certain cases of suspected gastrointestinal obstruction (duodenal strictures) or gastrointestinal ulceration.
   - Withhold food 4–12 hours or empty the stomach by nasogastric intubation prior to the study.
   - Barium sulphate suspension: 5 mL/kg via nasogastric tube.
   - Radiographs should be taken at 1, 15 and 30 minutes, then every 2 hours until the barium has passed the area of interest.
   - Barium should have left the stomach within 2 hours in a normal foal. Delayed gastric emptying can indicate pyloric outflow obstruction. However, sick foals may have delayed gastric emptying due to decreased gastrointestinal motility.
   - Barium swallow may be performed if oesophageal disease is suspected. Administer 100–120 mL barium sulphate suspension (liquid or paste) via dose syringe. This is contraindicated if oesophageal perforation or dysphagia is suspected.
   - Barium enema can be useful in diseases of the distal gastrointestinal tract such as atresia coli or other obstructive lesions (meconium impaction). Barium sulphate (180 mL) suspension is administered via an enema tube, and radiographs are taken immediately.

Ultrasonography

Ultrasound examination of the abdomen is useful for the evaluation of the bladder, umbilicus, kidneys, spleen, liver and gastrointestinal tract, as well as to look for the presence of peritoneal fluid.

- Free peritoneal fluid is dark and anechoic. Increased echogenicity may indicate peritonitis or gastrointestinal rupture. Presence of large amounts of anechoic fluid is highly suggestive of a ruptured bladder.
- An intussusception can sometimes be diagnosed with ultrasound by identifying the characteristic bulls-eye appearance on cross-section.
- Small intestinal distension caused by obstruction or ileus can also be identified with ultrasound.
- Assessment of intestinal wall thickness is important when assessing enteritis. In older foals this is important in the diagnosis of various forms of protein-losing enteropathies, including proliferative enteropathy caused by *Lawsonia intracellularis*.

Endoscopy

Endoscopic examination is useful to evaluate the oesophagus, stomach and pylorus.

- For optimal visualization, withhold feed or empty the stomach via nasogastric intubation prior to the examination.
- Gastric ulcerations are common in sick, stressed foals.

Abdominocentesis

1. Indicated in colic or where peritonitis or ruptured bladder is suspected.
2. The technique is the same as in the adult but with special considerations:
   - The foal must be adequately restrained.
   - The procedure can be performed with the foal standing or in lateral recumbency.
   - Care must be taken to prevent perforation of the bowel. The intestinal walls in the foal are thinner and more friable than the adult. Guidance with ultrasonography can be helpful.
3. Normal peritoneal fluid analysis is similar to that in the adult:
   - Colour: clear or pale yellow.
   - White blood cell count: $<3 \times 10^9$/L.
   - Protein: <25 g/L.
   Caution should be used in the evaluation of peritoneal fluid to differentiate medical and surgical cases, as elevations of protein and WBCs can occur in a number of conditions, as well as with enteritis.
4. Abnormal peritoneal fluid:
   - Increased protein and WBCs:
     - Enteritis.
     - Ischaemic intestinal lesions such as intussusceptions, volvulus.
     - Peritonitis.
     - Urachal or bladder infections.
   - Increased RBCs or haemorrhagic fluid:
     - Splenic tap.
     - Haemoperitoneum.
     - Haemorrhage from vessels in the abdominal wall.
   - Food material or ingesta:
     - Enterocentesis.
     - Bowel rupture.
5. Cytology is also useful to detect bacteria (which may indicate peritonitis, bowel rupture or bowel tap). Urine crystals can sometimes be seen in cases of uroperitoneum.
6. If uroperitoneum is suspected, the serum creatinine should be compared to the abdominal fluid creatinine.

Faecal analysis

1. Examination for parasites or protozoa.
   - Faecal flotation: *Stronyloides westeri*, *Parascaris equorum*, and strongyles.
   - Direct faecal examination: protozoa or oocysts (e.g. *Cryptosporidium*).
   - Normal foals older than 2 weeks should have an active faecal protozoal population.
2. Faecal cultures.
   - *Salmonella* spp. using selective media.
   - Multiple cultures should be performed as shedding of the organism appears to be intermittent.
   - Other organisms that can also be selectively cultured for include *Clostridium* spp, *Campylobacter* spp.
   - Polymerase chain reaction (PCR) for *Salmonella* spp, *Clostridium* spp, and *Lawsonia intracellularis*.
3. Assays for clostridial toxins.
   - *Clostridium difficile* toxins A and B.
   - *Clostridium perfringens* toxins.

4. Analysis for viruses, especially rotavirus.
   - Electron microscopy.
   - Enzyme-linked immunosorbent assay (ELISA).

Congenital problems of the alimentary system

Cleft palate

See Chapter 1.

Oesophageal stenosis

Congenital stenosis is rare and considered to be secondary to persistent right aortic arch.

Atresia

See Chapter 2.

The gastrointestinal tract is incomplete. A defect may occur at the anus (atresia ani), small colon (atresia coli) or rectum (atresia recti).

Lethal white syndrome (ileocolonic aganglionosis, overo lethal white syndrome)

Autosomal fatal disorder of white foals born from two overo American Paint horses. It is caused by a mutation of the endothelin receptor B. This results in absence of submucosal and myenteric ganglia in the small and large intestines. Most foals appear normal at birth, are white, with unpigmented skin, have blue irises, do not pass meconium and develop signs of colic with abdominal distension within hours to one day. These foals die or have to be euthanized. On necropsy there is intestinal impaction, distension, and a hypoplastic intestinal tract from the lack of intrinsic innervation (aganglionosis). This condition is similar to the Hirschsprung's disease of humans, in which there is a mutation of the endothelin B receptor, which impairs the proper migration of melanocytes to the skin and neurons to the intestinal tract. The diagnosis in foals is straightforward, based on clinical findings and abnormalities. There is a PCR-based test to identify carriers of this mutation.

Foal diarrhoea

Clinical signs

- Faeces can range from soft consistency to profuse watery, yellowish, dark haemorrhagic diarrhoea depending on the severity and cause.
- Increased borborygmi may be noticed before the onset of diarrhoea. Some foals may have decreased intestinal sounds.
- Depression, fever, anorexia, colic, dehydration, congested mucous membranes.
- Abdominal distension may be present.

Laboratory data

- Increased PCV and serum creatinine concentrations from dehydration and/or prerenal azotaemia. The presence of azotaemia (increased BUN and creatinine) should be addressed as it can be prerenal or renal. This is relevant to prognosis but also to the treatment plan, in particular for drug selection.
- Neutropenia with a left shift is a frequent finding, in particular in foals with evidence of endotoxaemia. Neutropenia can also be present in foals with viral diarrhoea from intestinal inflammation and absorption of endotoxins.
- Electrolyte imbalances include hyponatraemia, hypochloraemia, hypokalaemia, and metabolic acidosis.
- Hypoglycaemia is a frequent finding.
- Low total serum protein: total protein can be increased in foals with severe dehydration but is rare.
- L-lactate concentrations are increased in foals with poor perfusion.

Aetiologies of diarrhoea

Identifying the aetiological agents is often difficult. Predisposing conditions include peripartum asphyxia (maladjustment), failure of transfer of passive immunity (FTPI), septicaemia, and poor farm management.

1. Infectious causes of diarrhoea.
   - Bacterial:
     - *Salmonella* spp are a common cause of bacterial enteritis. Can cause enterocolitis with severe diarrhoea, colic, depression and fever. Concurrent sepsis, septic arthritis, or osteomyelitis can occur.
     - *Escherichia coli* is probably not a primary cause of diarrhoea in foals, but it is frequently a cause of sepsis, and diarrhoea can develop secondarily to this.
     - *Clostridium perfringens* (types A and C*)* and *C. difficile* are the most common causes of clostridial diarrhoea in foals. Usually sporadic, but occasionally can present as outbreaks. In general the diarrhoea is severe may be haemorrhagic, and may cause rapid death. Some foals may have severe necrotizing enterocolitis and present for acute colic with no evident diarrhoea (per acute death). *C. difficile* diarrhoea can present in foals as early as 1 day of age, and faeces vary from yellowish to haemorrhagic.
     - Other bacteria that have been associated with diarrhoea in the neonatal foal include *Rhodococcus equi*, which can cause diarrhoea in foals 1–4 months of age; it is more commonly associated with pneumonia and lung abscesses, and can also cause septic arthritis, physitis, uveitis and abdominal abscesses. *Lawsonia intracellularis* (proliferative enteropathy) occurs in foals older than 2 months; affected foals are presented for poor body condition, weight loss, ventral oedema, and diarrhoea; treatment is administration of a macrolide (erythromycin or clarithromycin) with rifampin or a tetracycline (oxytetracycline or doxycycline) for 3 weeks. Response to this treatment is good. Plasma transfusion may be required. (See Chapter19*: L. intracellularis*.) *Actinobacillus equuli* and *Klebsiella* spp are usually associated with septicaemia.
   - Viral causes of diarrhoea:
     - Rotavirus is the most common viral cause of diarrhoea in foals. It causes a profuse watery diarrhoea in foals usually <1 month of age; most foals are younger than 2 weeks of age. Many foals on the farm are affected within 3–5 days. Diarrhoea is usually profuse and watery. The virus induces denudation of the epithelial cells of the proximal intestine, resulting in increased secretion and decreased absorption of fluids and electrolytes. These cells also produce disaccharidases such as lactase; thus, lactose is not digested in the small intestine but is broken down by bacteria in the large intestine. This worsens the diarrhoea as glucose pulls water into the intestine (osmotic diarrhoea).
     - Blood abnormalities include metabolic acidosis, hyponatraemia, hypochloraemia, and hypokalaemia.
     - As gastroduodenal ulcers may develop, anti-acid drugs should be considered, especially when entral feeding is decreased.
     - As foals with rotavirus infection can dehydrate quickly, rapid intervention is required. Therapy should include

balanced electrolyte solutions and sodium bicarbonate. Enteral feeding should be restricted or reduced.
    - Other viruses that have been isolated from foals with diarrhoea include coronavirus, parvovirus and adenovirus. Their pathogenicity in foals is unknown. These viruses are typically isolated from immunocompromised foals and do not cause outbreaks.
  - Parasitic causes of diarrhoea (see Chapter 19).
    - *Strongyloides westeri* is a nematode that may cause mild diarrhoea in the foal at 1–4 weeks of age.
    - Heavy infestation with strongyles or *Parascaris equorum* may cause diarrhoea but uncommonly.
    - *Cryptosporidium* may occur in immunocompromised foals. This has zoonotic potential. Treatment is by supportive care.
2. Foal heat diarrhoea.
   - Self-limiting diarrhoea that occurs between 6 and 14 days of age.
   - Affected foals are not systemically ill, and usually no treatment is necessary.
   - Resolves in <1 week.
3. Nutritional causes of diarrhoea.
   - Over-eating or over-feeding, sudden changes in diet, or ingestion of sand or dirt can cause diarrhoea.
   - Lactose intolerance is uncommon as a primary condition. Usually this is a transient condition secondary to infectious diarrhoea.
4. Antibiotic-associated diarrhoea.
   - The administration of antimicrobial drugs can alter the normal microflora of the gastrointestinal tract which can lead to colonization by pathogenic bacteria. This is rarely a problem in newborn foals, but can occur in foals at a few weeks of age.
   - Antibiotics associated with diarrhoea include macrolides (erythromycin, azythromycin), oxytetracycline, trimethoprim–sulphonamides, penicillins, and third-generation cefalosporins.
   - There is a low incidence of antibiotic-related diarrhoea in foals; therefore these antibiotics should still be used if needed. If diarrhoea develops, consider discontinuing the antibiotic.

Diagnosis

- Diagnosis of the aetiological agent is often difficult despite testing. In most cases of foal diarrhoea an aetiological agent is not identified.
- Faecal cultures, faecal analysis for parasites, viruses, pathogen DNA (PCR) and blood cultures are indicated.

Treatment

Initial treatment is supportive with the goal of correcting hydration, acid–base and electrolyte imbalances, and hypoglycaemia.

1. Intravenous fluids. There are many options on the market, but balanced isotonic replacement solutions such as lactated Ringer's solution, Plasma-Lyte 148, Normosol R, or Isolyte S are good initial options. Saline solution (0.9%) can be used in some cases. Another aspect to consider when using these solution is the pH; while replacement solutions have a pH of >6.0, pH of 0.9% saline solution is 4.0. This may be relevant in foals with metabolic acidosis.
   - Administration of KCl should be considered in severe hypokalaemia. KCl administration should not exceed 0.25–0.5 mEq/kg/h as a rapid rate may lead to cardiac dysrhythmias. Mild hypokalaemia is often corrected with restoration of the hydration status, and intravenous or oral supplementation of KCl may not be necessary.
   - Supplementation becomes more important in foals with metabolic acidosis, as low pH leads to overestimation of serum K concentrations. As the pH increases K moves into the cells and *vice versa*.
   - Severe acidosis may require intravenous administration of isotonic sodium bicarbonate. It is preferable to administer sodium bicarbonate alone than to mix it with other solutions, in particular those with calcium and magnesium salts as they may precipitate. Rapid administration of sodium-containing fluids to foals with severe hyponatraemia may lead to neurological signs.
   - Dextrose may be added to fluids for hypoglycaemic foals. Typical solutions include 5% dextrose or 2.5% dextrose + 0.45% saline. Dextrose can also be added to other solutions to create a 2.5–5% concentration. Injectable hypertonic dextrose comes as a 25% or 50% solution.
   - Hypocalcaemia is a frequent abnormality in foals with gastrointestinal disease. Isotonic solutions can be supplemented with calcium gluconate (23%) at 10 mL/L.
2. Oral electrolyte solutions.
   - Not as effective in correcting severe electrolyte and acid–base imbalances, especially if absorption is impaired from enteritis. However, oral supplementation of potassium chloride and sodium bicarbonate is an effective and cheaper way to correct electrolyte concentrations in foals with mild abnormalities.
   - Older foals may be able to maintain hydration and correct mild electrolyte imbalances if free-choice water and electrolytes (in water or salt block with trace minerals) are provided.
   - If the foal is not interested in drinking, fluids and electrolytes may be administered via nasogastric tube.
3. Nutrition/caloric intake is very important in foals. Supplementation should be considered if the foal is not nursing or eating, of if the foal has evidence of proximal intestinal disease or lactose intolerance (idiopathic, rotavirus).
   - If the foal is interested in eating, controlled nursing is important as excessive milk intake may worsen the clinical signs. Excess of lactose in a dysfunctional small intestine may lead to bacterial breakdown of lactose to glucose and galactose. Glucose is osmotically active, drawing more water into the intestine. In addition, this increase in luminal glucose can lead to bacterial overgrowth.
   - If the foal is not interested in nursing, a small-bore feeding tube can be placed. These tubes have the advantage that the foal can nurse without problems, and they can be left in place for many days.
   - Bottle feeding should be done by experienced personnel as in some situations it may lead to aspiration pneumonia.
   - If there is no evidence of improvement and the foal does not tolerate enteral feeding, parenteral nutrition should be considered.
4. Antibiotics.
   - There are several factors to take into consideration in the selection of antimicrobial drugs, including current disease, aetiological agent, drug interactions, toxicity, and cost. The initial choice should be a broad-spectrum combination, unless the aetiological agent and sensitivity are known.
   - A combination of penicillin (or ampicillin) and an aminoglycoside (gentamicin or amikacin) is the most frequently used combination. It is important that the foal is well hydrated and that there is no evidence of renal disease, as aminoglycosides are nephrotoxic, particularly if there is dehydration and concurrent use of other nephrotoxic drugs (e.g. flunixin meglumine).

- Third-generation cefalosporins such as ceftiofur (4.4–8.8 mg/kg IV BID), cefotaxime (40 mg/kg IV TID–QID), ceftazidime (40 mg/kg IV TID–QID), or ceftriaxone (10 mg/kg IV BID).
- Metronidazole (20 mg/kg PO TID–QID) should be considered in foals with haemorrhagic diarrhoea, as this is a common finding in clostridial diarrhoea.

5. Anti-endotoxin therapy.
   - Flunixin meglumine (0.25 mg/kg IV TID) or polymyxin B (3000–6000 IU/kg IV BID, in 5% dextrose solution) should be considered in foals with severe sepsis or evidence of endotoxaemia.
6. Intestinal protectants. The use of these products is controversial, but protectants to consider are bismuth subsalicylate, kaolin, pectin, and activated charcoal. More recently, smectite-based products (Bio-Sponge) have been claimed to show good results in treating foal diarrhoea.
7. Non-steroidal anti-inflammatory agents.
   - Flunixin meglumine if there is evidence of endotoxaemia (0.25 mg/kg IV TID) or fever, pain, inflammation (0.25–1.1 mg/kg IV).
   - Ketoprofen (1.1–2.2 mg/kg IV) is a good choice in foals with fever and pain. It is less ulcerogenic than other non-steroidal drugs.
8. Plasma transfusion may be indicated in cases of FTPI or in foals with hypoproteinaemia.
9. Additional therapeutic measures.
   Oral supplementation with lactase should be considered in foals with watery diarrhoea, foals with rotavirus infection, or in foals that do not tolerate enteral feeding. Anti-acid drugs such as cimetidine, ranitidine, or omeprazole can be used as foals are prone to developing gastric ulcers.

Gastroduodenal ulcers

Predisposing factors include stress, previous illness, decreased feeding, and treatment with non-steroidal anti-inflammatory drugs.

Clinical signs

- Diarrhoea and abdominal pain are the most common signs.
- Signs of colic/pain, bruxism (grinding teeth), salivation, dorsal recumbency.
- History of stress such as recent shipping or previous illness.

Diagnosis

- Often based on clinical signs.
- Faecal occult blood test can be positive but is not diagnostic.
- Endoscopic examination is the test of choice. Feed intake should be restricted for 4–6 hours in foals. The pyloric region and duodenum can be difficult to visualize in foals. Ulcers frequently occur in the squamous mucosa near the margo plicatus and along the lesser curvature and cardia in older foals.
- Contrast radiographic studies may be indicated if duodenal stricture is suspected.

Treatment

1. Drugs to decrease gastric acid secretion.
   - Histamine type-2 receptor antagonists: cimetidine and ranitidine, available in oral or IV preparations.
   - Omeprazole: blocks HCl secretion by inhibiting the hydrogen ion pump and thereby blocking all receptor types for gastric acid secretion. May be indicated when gastric ulcers are not responsive to H2 blockers. A cheap way to give it is by using the adult formulation and dosing on the basis of body weight.
2. Mucosal protectants.
   - Sucralfate. There is no evidence that sucralfate is effective in foals unless the presence of gastric ulcers is confirmed. It has been suggested that sucralfate should be given 1 hour before H2 blockers because it is most effective in an acid environment. It can be expensive.
   - Anti-acids such as magnesium hydroxide, aluminium hydroxide, or calcium bicarbonate are rarely used in equine practice.

Prognosis

The prognosis is good with treatment and reduction of stress, unless complications develop.

Sequelae

- Perforation: gastric or duodenal ulcers may perforate, resulting in severe peritonitis and death.
- Oesophageal stricture: secondary to chronic reflux oesophagitis.
- Duodenal stricture: results in delayed gastric emptying. These foals present for chronic abdominal pain. Surgery to bypass the stricture may be attempted.

Meconium impaction/retention

Meconium consists of the intestinal secretions, cellular debris, and digested amniotic fluid that accumulate in the intestinal tract during gestation. Normally it is completely passed during the first 24–48 hours of life.

Clinical signs

- Straining to defecate: the foal usually stands with the back arched upwards, whereas foals straining to urinate stand more stretched out.
- Tail swishing, restlessness.
- Decreased nursing.
- Abdominal pain. With time, abdominal distension develops.

Diagnosis

- In mild cases, diagnosis is often based on the clinical signs and response to treatment.
- Digital rectal examination may reveal firm meconium in the rectum, and help to rule out congenital abnormalities such as atresia.

Treatment

1. Enemas. Care must be taken not to irritate or damage the rectal mucosa. Over-distension of the rectum can result in rectal tears.
   - Warm water and soapy water enemas.
   - Commercial phosphate enemas. Should be used with care as some foals may develop hyperphosphataemia.
   - Acetylcysteine retention enemas are effective for refractory impactions. Acetylcysteine (4% solution) is administered via Foley catheter and retained for 10–30 minutes.
2. Oral laxatives.
   - Mineral oil via nasogastric tube.
3. IV fluids if the foal is dehydrated.
4. Surgical therapy may be required in cases with severe abdominal distension or bowel compromise.

Abdominal abscesses

Aetiology

Most frequent aetiological agents include *Streptococcus equi*, *S. zooepidemicus* and *Rhodococcus equi*; occasionally enterobacteria are involved.

Clinical signs

- Depression, fever, anorexia. Depending on the duration, poor body condition.
- Colic due to compressive effects on the intestines or adhesion formation.
- Peritonitis due to inflammation or rupture of abscess.

Diagnosis

- Haematology may show neutrophilia with left shift. High fibrinogen.
- Ultrasound or radiology may be helpful in locating an abscess.

Treatment

- Medical treatment with long-term antibiotic therapy is often unrewarding.
- If there is bowel involvement or if medical therapy is unsuccessful, surgery may be indicated to attempt resection of the abscess. Resection is ideal but may not be possible. In these cases, debulking and establishing drainage are performed by marsupialization or drain placement.
- Antimicrobials should have good penetration. To consider for *Streptococcus* spp. and *Rhodococcus equi* abscesses are macrolides (erythromycin, clarithromycin, azythromycin) alone or in combination with rifampin, sulphonamides/trimethoprim, and tetracyclines. Penicillins and aminoglycosides have poor cure success for abscesses. Care should be taken when using macrolides in foals as the mares may develop clostridial enterocolitis.

Obstructive/strangulating lesions

These are similar to those in adults in causing signs of colic, tachycardia and abdominal distension. See Chapter 2.

Intussusceptions

These usually involve the small intestine: jejunojejunal or ileocaecal. Ultrasound may help diagnosis. Exploratory surgery with resection of the affected area is indicated.

Hernias

These may be diaphragmatic, inguinal/scrotal, intra-abdominal (mesenteric rent) or umbilical. Inability to reduce an umbilical or scrotal hernia constitutes an emergency, and surgical repair should be performed immediately to minimize bowel compromise.

Small intestinal volvulus

This strangulating lesion causes acute, severe pain, and rapid cardiovascular deterioration.

Large colon displacement and volvulus

These are not common in foals, but large colon displacements and volvulus can occur.

Functional obstruction: ileus

Ileus can result from electrolyte abnormalities (especially hypocalcaemia and hypokalaemia), intestinal irritation, enteritis and peritonitis. Ultrasound and radiography can be useful to determine the extent of abdominal distension. It is important to rule out mechanical obstruction as the cause of ileus. Treatment is aimed at removing the underlying cause and correcting any electrolyte imbalances.

Surgical treatment

Surgery is indicated if the foal is persistently in pain or has evidence of complete obstruction or strangulation. Reported short-term survival for foals undergoing colic surgery is approximately 65%.

20.8 HEPATOBILIARY DISEASES

Examination

Physical examination and clinical signs

See Chapter 3.

Laboratory investigations

See Chapter 3.

Ultrasonography

Ultrasonography is a very useful non-invasive tool.

- The liver can be visualized on the right side of the ventral abdomen from the 6th to the 15th intercostal spaces.
- Portal and hepatic vessels can normally be visualized in the parenchyma. Veins are very prominent in foals. Bile ducts are very small in normal liver and are often not seen.
- In cholestatic disease it may be possible to see distended bile ducts.
- In hepatocellular disease, inflammation or diffuse cellular infiltration results in increased echogenicity of the liver parenchyma.
- Focal hepatic diseases such as abscesses can also be diagnosed using ultrasound and appear as masses with increased echogenicity.

Liver biopsy

Liver biopsy is a procedure rarely performed in foals unless severe parenchymal disease is suspected. See Chapter 3.

Infectious hepatobiliary diseases of the foal

Tyzzer's disease

This is an acute, highly fatal infectious hepatitis of foals.

Aetiology

The infectious agent is *Clostridium piliforme* (*Bacillus piliformis*), a Gram-negative (despite being a *Clostridium* sp.), filamentous, intracellular, anaerobic bacterium. Infection occurs via ingestion of contaminated soil or faeces. The mare is suspected to be the source of infection.

Clinical signs

- Usually occurs in foals of 40–60 days of age.
- Onset and progression of disease is very rapid, from hours to days.
- Signs include depression, anorexia, recumbency, ataxia, seizures, icterus, fever, diarrhoea, coma, or sudden death. Classical signs are consistent with hepatic encephalopathy. Faeces may be white to grey.
- Laboratory data are suggestive of liver failure and include increased liver enzymes, increased serum bilirubin, increased ammonia, hypoglycaemia, low BUN, prolonged clotting times, metabolic acidosis, neutropenia or neutrophilia.

Diagnosis

- As this is an acute disease, antemortem diagnosis is difficult.
- Liver biopsy may provide a diagnosis with identification of groups of parallel bacilli within hepatocytes, in particular around necrotic areas.
- The organism is difficult to culture, but PCR methods are available.

Treatment

- Usually not successful.
- Intravenously administered antibiotics. The organism is susceptible to penicillins, oxytetracycline, streptomycin, erythromycin, and metronidazole.
- Supportive treatment: IV fluids (with dextrose if hypoglycaemic).

Prognosis

The prognosis is grave despite treatment. Most foals die within 24 hours.

Equine herpesvirus 1

In utero infection during late gestation with EHV-1 most commonly causes abortion, but can cause interstitial pneumonia and necrotizing hepatitis in neonates. Clinical signs include weakness, depression, respiratory signs, liver failure and icterus. See Chapter 19.

Septicaemia

- Bacteria (enterobacteria, *Actinobacillus, Listeria monocytogenes, Clostridium* spp.) can be isolated from the liver of septicaemic foals. Septicaemia and endotoxaemia interfere with normal hepatocyte function.
- Hepatitis can also develop secondarily to septicaemia via the haematogenous route or ascending cholangitis.

Liver abscesses

- Uncommon but can occur in the foal secondarily to septicaemia or via ascending infection of the bile ducts.
- Clinical signs include weight loss, intermittent colic and fever.
- Ultrasonography can be very beneficial in diagnosis. Liver enzymes can be elevated.

Toxic hepatobiliary disease

Iron fumarate toxicosis

Aetiology

Administration of iron (fumarate oral supplement) prior to colostrum intake, when intestinal absorption is high, has been associated with fatal hepatic necrosis. Foals are much more susceptible to iron toxicity than adults. There is no need to supplement healthy foals with iron.

Clinical signs

- Usually occurs 2–5 days after administration of iron fumarate.
- Signs include depression, icterus, hyperexcitability, blindness, ataxia, coma and death.

Diagnosis

- History of iron supplementation.
- The liver is smaller than normal.
- Histopathology shows severe hepatic necrosis.

Treatment

Treatment is supportive.

Prognosis

The prognosis is poor if there is liver failure.

Pyrrolizidine alkaloid toxicosis

This is very rare in foals. See Chapter 3.

Congenital hepatobiliary conditions

Portacaval/portosystemic shunts

- Few cases have been documented in the literature.
- Portosystemic shunts can be internal or external. Shunts allow blood from the portal system to bypass the liver and drain into systemic circulation, via the caudal vena cava or the azygous vein.
- Most affected foals are older than 1 month.
- Foals present because of neurological signs, including ataxia, disorientation, and blindness associated with poor body condition.
- On ultrasound the liver is small and hyperechoic. Occasionally external shunts may be observed.
- Laboratory abnormalities include increased ammonia, increased bile acids, low BUN, normal liver enzymes.
- Liver biopsy reveals hepatocyte atrophy, fibrosis, and biliary tract hyperplasia.
- Portography (intraoperative) can be performed.
- Treatment is surgical, and prognosis is poor.
- On necropsy the liver is small, and the shunt can be observed (if external).

Biliary atresia

- This is a rare condition in which there is incomplete formation of the biliary ducts.
- Foals will present for depression, anorexia, poor body condition, icterus, fever, colic, polyuria, and polydipsia.
- Laboratory tests reveal increased GGT (gamma-glutamyl transferase), increased direct bilirubin, and increased sorbitol dehydrogenase activity.
- The liver is normal or larger than normal on necropsy. Canaliculi are distended, there is biliary hyperplasia, and the portal triad is not properly developed.

Biliary obstruction/cholangiohepatitis

(See Chapter 3)

- Ascending disease of the biliary tract and hepatic parenchyma associated with gastroduodenal ulcers that lead to duodenal strictures, common bile duct obstruction and cholestasis.
- Strictures distal to the opening of the common bile duct into the duodenum (hepatopancreatic ampulla) are associated with cholestasis and reflux of ingesta into the common bile duct. Chronic cholestasis leads to cholangiohepatitis.
- Depending on the degree of inflammation or obstruction, direct bilirubin and GGT concentrations are elevated.
- Cholangiohepatitis from strictures should resolve with surgical correction, unless permanent hepatic damage has occurred.

Hepatic lipidosis

- Sick pony foals, miniature horse foals, and donkey foal may develop hyperlipaemia from different diseases. Some foals may accumulate large amounts of triglycerides in the liver (hepatic lipidosis) leading to liver failure, encephalopathy, and death.
- Clinical signs include depression, anorexia, blindness, seizures, ataxia, diarrhoea, and sudden death.
- Laboratory abnormalities are those of liver disease, and serum triglycerides are elevated.
- Medical treatment is supportive, based on dextrose and insulin to reduce the rate of fat mobilization.

20.9 DISEASES OF THE UROGENITAL SYSTEM

Examination

Physical examination

- The umbilicus should be carefully examined for any abnormalities such as urine leakage (indicating patent urachus), swelling or discharge (which may indicate umbilical infection).
- Abdominal ballotment can be useful in detecting free abdominal fluid.
- The external genitalia should be examined for any abnormalities such as congenital malformations. Some colts will have a persistent frenulum for the first week of life.

Serum biochemistry and electrolytes

See Chapter 8.

Urinalysis

See Chapter 8.

Healthy, nursing neonatal foals have a low urine specific gravity (1.001–1.010).

Radiography

1. Loss of serosal detail and presence of a fluid line (with standing radiographs) indicate free abdominal fluid.
2. Contrast studies can be helpful in determining the site of urinary tract disruption prior to surgery.
   - A water-soluble contrast agent must be used (e.g. iohexol).
   - The urinary bladder is catheterized (using aseptic techniques), and a suitable amount of contrast agent is injected to distend the bladder.
   - Lateral and ventrodorsal (if possible) radiographs are taken. Contrast material within the peritoneal cavity indicates a ruptured bladder or urachal defect. Occasionally urachal tears can occur outside the peritoneal cavity and contrast material can be seen in the subcutaneous tissue around the umbilicus.
3. Excretory urography such as an intravenous pyelogram may be useful in diagnosing ectopic ureters.
   - Caution should be used as the contrast material is potentially nephrotoxic, especially if there is dehydration or impaired renal function.
   - Iodinated, water-soluble contrast material is used.

Ultrasonography

- Very useful in evaluating the umbilicus for infection or abscesses.
- Free abdominal fluid can be visualized to aid abdominocentesis. Free abdominal fluid and lack of visualization of a fluid-filled bladder are supportive of bladder rupture. However, visualization of a fluid-filled bladder does not rule out the possibility of a rupture as some cases may involve small tears, and the bladder is able to partially fill.
- The kidneys should also be evaluated especially if nephritis or congenital abnormalities are suspected.

Abdominocentesis

1. Should be performed in cases of excessive free peritoneal fluid and when uroabdomen is suspected.
2. Uroperitoneum:
   - Peritoneal fluid creatinine should be compared to serum creatinine to diagnose uroperitoneum. A ratio >2:1 is highly suggestive of uroperitoneum, although a normal ratio does not rule it out.
   - Increased WBCs, increased total protein and/or bacteria in cases of uroperitoneum may indicate septic peritonitis.

Congenital urogenital defects

Ectopic ureter

1. Rare, but the most common congenital anomaly of the equine urinary tract. More common in fillies.
2. Primary clinical sign is persistent urinary incontinence which is present at birth. Affected foals often develop urine scalds in the perineal region and down the backs of the limbs.
3. Definitive diagnosis is by excretory urography or endoscopy.
4. Surgical correction is successful in some cases.

Cryptorchidism

See also Chapter 8.

Occurs in 2–8% of male horses. This condition is thought to have a heritable component.

Congenital hernias

See also Chapter 4.

Neonatal foals should be evaluated for the presence of congenital inguinal and umbilical hernias.

1. Small umbilical hernias usually close within the first few weeks of life. Large umbilical hernias and those that persist warrant surgical repair. Intestinal incarceration in an umbilical hernia requires emergency surgery.
2. Congenital inguinal hernias usually involve the small intestine, are easily reducible, and rarely require surgery. Non-reducible and ruptured inguinal hernias require immediate surgical repair.

Rare congenital defects

1. Renal dysplasia/hypoplasia: diagnosis by renal ultrasound and biopsy.
2. Rectovaginal and rectoureteral fistula: often associated with atresia ani.
3. Intersex: abnormal external and internal genitalia. The XX-male pseudohermaphrodite is the most common intersex in horses.

Patent urachus

See also Chapter 8.

1. Urine drips from the umbilicus when the foal urinates. The urachus may be patent at birth or may reopen due to infection.
2. The umbilicus should be kept clean using dilute chlorhexidine solution (0.5%), and systemically administered antimicrobials are used to treat or prevent infection.
3. Surgical resection of the umbilical remnants is recommended if infection is present or the urachus has not closed after 5–7 days of medical treatment.

Omphalitis/umbilical remnant infections

Infection can occur in umbilical remnants or urachal remnants.

Clinical signs

- Swelling of the umbilicus and occasionally purulent discharge can be expressed from the umbilicus. Lack of external swelling does not rule out umbilical remnant infection as infection/abscess can occur internally.

- Intermittent fever, leukocystosis, neutrophilia, and increased plasma fibrinogen are non-specific signs of infection that can be seen with umbilical infections.

Diagnosis

- Can often be made on the basis of the clinical signs.
- Ultrasonography is very helpful to diagnose infections of the internal portions of the umbilical remnants and to determine the extent of infection. Any foal with signs of inflammation and infection that cannot be localized should have an ultrasound evaluation of the umbilicus.

Treatment

- Medical therapy with long-term, broad-spectrum antibiotics.
- Surgical removal of infected umbilical remnants is indicated if there is not an adequate response to medical treatment, or the umbilicus is severely enlarged, or there is leakage of urine from the urachus.

Ruptured bladder

See Chapter 8.

Clinical signs

- Frequent attempts to urinate. Progressive abdominal distension and colic.
- Weakness, recumbency, and cardiovascular compromise occur as electrolyte abnormalities progress.
- Signs are seen in the first 24–72 hours of life if the rupture occurs during parturition. Rupture can occur at days to weeks after birth when it is secondary to sepsis.

Diagnosis

- Ultrasonography usually reveals a large amount of free abdominal fluid and a small urinary bladder.
- Abdominocentesis reveals clear-yellow fluid with a creatinine concentration greater than twice that of the serum creatinine.
- Electrolyte derangements include hyperkalaemia, hyponatraemia, and hypochloraemia.

Treatment

- Surgical repair of the defect is not performed until the foal is medically stabilized.
- Correction of electrolyte abnormalities, drainage of the uroperitoneum, and evaluation for concurrent disease, such as sepsis.

Primary renal disease

See also Chapter 8.

Aetiology

1. Sepsis-associated.
   - Most common cause of renal failure in foals.
   - Sepsis can cause renal failure via ischaemic damage and direct infection of the kidneys by the causative organism.
   - Common organisms involved include *Actinobacillus equuli*, *Escherichia coli* and *Klebsiella*.
2. Nephrotoxic renal failure.
   - Uncommon cause of renal failure but results from overdoses of potentially nephrotoxic drugs or administration of normal doses to a dehydrated patient.
   - Aminoglycosides can cause renal tubular damage. Gentamicin is reportedly more nephrotoxic than amikacin.
   - Oxytetracycline, especially when given at high dosages for treatment of contracted tendons, has been reported as a cause of renal failure in foals.
   - Non-steroidal anti-inflammatory drugs (prostaglandin inhibition) can cause renal failure by reducing blood flow, resulting in ischaemia. They can also cause renal papillary necrosis (phenylbutazone).
   - Haemoglobin in foals with haemolytic anaemia (isoerythrolysis). Haemoglobin causes renal vasoconstriction, acute tubular necrosis, and renal failure.
3. Ischaemic renal failure.
   - May be a consequence of perinatal asphyxia, severe dehydration, volume depletion.

Clinical signs

- Depression, lethargy, anorexia, tachycardia, fever.
- Oliguria.

Diagnosis

It is important to differentiate renal failure from urinary tract disruption such as ruptured bladder or obstruction.

- Laboratory findings: azotaemia, hyponatraemia, hyperkalaemia and metabolic acidosis are commonly seen.
- Urinalysis: urine gamma-glutamyl transferase/urine creatinine ratio may be elevated. This method is not considered reliable, but is used by some clinicians. Fractional excretions may be useful, but values are not as well established in foals. Urine cytology may reveal casts indicating renal tubular damage.

Treatment

The goals of therapy are to correct the underlying causes such as dehydration or sepsis, to correct electrolyte and metabolic imbalances, and to maintain urine output.

1. Fluid therapy. Care must be taken not to over-hydrate, especially in the oliguric patient.
   - Fluid overload can result in oedema, hypertension, CNS swelling and death.
   - Monitoring central venous pressure (CVP), urine output, PCV, and body weight can be helpful.
   - Potassium-free fluid such as 0.9% saline solution is the best choice in hyperkalaemia.
2. Diuretics in foals with evidence of acute tubular necrosis (haemoglobinuria) may be used to try to improve urinary output in oliguric patients.
3. Diazepam or midazolam may be indicated to control seizures.
4. Peritoneal dialysis or haemodialysis has been proposed in anuric renal failure patient.

20.10 ENDOCRINE/METABOLIC DISORDERS

Neonatal hypothyroidism (goitre)

See Chapter 9.

Adrenal insufficiency (hypoadrenocorticism)

Adrenal insufficiency, also known as hypoadrenocorticism, is a poorly understood condition in foals. It occurs in premature, dysmature, and septicaemic foals. The dysfunction can be at the hypothalamus, pituitary gland, or adrenal gland. Most often it is the adrenal gland that has a poor response (cortisol release) to adrenocorticotropin (ACTH) stimulation. The adrenal cortex is not fully developed or functional in premature foals.

Cortisol has many essential functions, including regulation of intermediary metabolism (carbohydrates, proteins, lipids), immunomodulation, controlling inflammation, and in late gestation/early postnatal period is important for organ maturation. Aldosterone, another adrenal corticoid that is essential in regulating fluids and electrolytes, can be low.

1. Clinical signs: evidence of prematurity or dysmaturity, septicaemia, and respiratory dysfunction (see section on prematurity).
2. Diagnosis is based on clinical signs with supporting laboratory data.
   - Normal plasma cortisol in healthy foals of 1–7 days of age is >2 µg/dL. Values of <2 µg/dL are suggestive of adrenal insufficiency but are not diagnostic unless a functional test is performed.
   - ACTH stimulation test: no or minimal response to 100 µg of synthetic ACTH (cosyntropin). In foals with a normal adrenal gland function cortisol increases by 200% with this ACTH dose.
   - Neutropenia and lymphocytosis may be present.
   - Hyponatraemia and hyperkalaemia if aldosterone concentrations are low.
3. Treatment: glucocorticoid (hydrocortisone, dexamethasone) supplementation remains controversial but should be considered in foals with immature lungs and electrolyte and glucose abnormalities. Also necessary is supportive medical treatment.

Nutritional secondary hyperparathyroidism

See Chapter 9. This is not a problem in newborn foals.

20.11 HAEMOLYMPHATIC AND IMMUNOLOGICAL DISORDERS

Failure of transfer of passive immunity (FTPI)

Passive transfer of maternal antibodies does not occur transplacentally in the horse (epitheliochorial placenta). The mare's colostrum contains high concentrations of antibodies (primarily IgG); it usually contains up to five times the IgG concentration of plasma. The foal must ingest colostrum and absorb the antibodies. The foal's gastrointestinal tract is able to absorb proteins intact via specialized villus epithelial cells in the small intestine. Maximum absorption occurs within 8 hours following parturition, and the specialized epithelial cells are replaced by more mature cells unable to pinocytose proteins within 24–36 hours.

Causes of FTPI

1. Inadequate colostrum or inadequate concentration of IgG in colostrum.
   - Mares that prematurely lactate (>24 hours prior to parturition) tend to have lower IgG concentration in the colostrum.
   - Premature parturition: mares foaling prior to 320 days may not have adequate colostral antibodies.
   - Mares on fescue pasture during late gestation can have agalactia, thickened foetal membranes, premature lactation, retained placenta, and weak foals.
2. Failure of the foal to ingest and absorb colostrum within the first 12 hours. Any illness or abnormality which causes the foal to be weak can result in lack of nursing.

Clinical signs

- There are no signs specific for FTPI, but these foals are predisposed to septicaemia and other infections.
- Neonatal maladjustment (dummy foals): these foals generally do not have a suckle reflex, are disoriented and unable to nurse.

Diagnosis

1. Immunoglobulin G concentrations should be measured at 24 hours of age (after antibody absorption is complete). Values <400 mg/dL are considered diagnostic of FTPI; 400–800 mg/dL is considered partial failure; >800 mg/dL is considered adequate.
   - Methods to measure IgG include the zinc sulphate turbidity test, glutaraldehyde precipitation, ELISA (CITE®, SNAP®), and agar gel immunodiffusion (AGID, radial immunodiffusion). The zinc sulphate test is the least expensive; however, it is not a sensitive test, and false positives often occur. AGID is considered the most accurate method.
2. Measurement of IgG content of the colostrum.
   - The specific gravity of colostrum directly correlates with the IgG content. Colostrum with a specific gravity >1.06 usually contains sufficient IgG.
   - The specific gravity can be measured with a colostrometer.

Treatment

The treatment is dependent on the reason for FTPI and the age of the foal.

1. If the foal is <18–24 hours old:
   - Frozen colostrum can be administered via bottle or nasogastric tube. At least 1 L should be administered within the first 12 hours of life, divided into several feedings. Frozen colostrum should not be thawed in a microwave.
   - Lyophilized IgG is an oral IgG supplement. Variable absorption has been reported.
   - Plasma can be administered orally, but it is not considered to be as effective as intravenous administration.
2. If the foal is >24 hours old or if colostrum is not available:
   - Plasma transfusion.
   - Approximately 20–40 mL/kg plasma is necessary to raise IgG levels to >400 mg/dL. If the foal is already sick, additional amounts are necessary.
   - Ideally the plasma should be cross-matched if commercial cell-free plasma from anti-RBC negative donors is not being used.
   - Serum IgG levels should be tested at 2–24 hours following administration, depending on the health status of the foal. Additional plasma should be administered if IgG concentration remains low.
   - Caution should be taken not to cause fluid overload, especially in sick or very small foals.
   - Before administering a large volume of plasma, a small amount (20 mL) should be given to assess for anaphylactic reactions. This manifests as tachycardia and tachypnoea, usually within 10–15 minutes. Urticaria is rare in foals.
   - Avoid fluid overload, especially in sick or small foals.

Prevention

- Mares should be removed from endophyte-infected fescue pastures 30–60 days before the expected foaling date.

- The colostrum should be checked in mares. Stickiness is a better indicator of good quality than colour or thickness. It is best to measure the specific gravity.
- The foal should be watched to ensure that it stands and nurses within a reasonable time after birth.
- If there is any doubt, the foal's serum IgG levels should be measured.
- Large breeding farms should establish a colostrum bank; 200–400 mL of colostrum can be taken from a normal mare without jeopardizing her foal's IgG levels. Also mares that die or lose their foals should be milked out and the colostrum frozen.

Severe combined immunodeficiency (SCID)

See Chapter 10.

- Primary immunodeficiency of Arabian foals in which there is a failure to produce functional B and T lymphocytes.
- The condition is autosomal recessive. There is a defect in an enzyme (DNA protein kinase) responsible for V(D)J recombination necessary for lymphoid tissue differentiation, and T-cell receptor and immunoglobulin production.
- Foals with SCID are normal at birth, but over time and depending on maternal antibodies titres, develop infections (fungal, viral, bacterial, protozoal). Frequently these foals have infections by *Cryptosporidium* spp, *Pneumocystis carinii*, and *Rhodococcus equi*. Adenovirus is often isolated from the intestinal, respiratory, and urogenital systems.
- Clinical signs are those of respiratory and gastrointestinal infections, including diarrhoea, fever, weight loss, nasal discharge, dyspnoea. Most foals die by 6 months of age.
- Laboratory tests reveal lymphopenia and neutrophilia. Immediately after birth IgG and IgM concentrations can be normal (maternal), but after 2–4 weeks IgG is low and IgM is undetectable.
- The diagnosis is clinical. Breed, lymphopenia, infections, no IgM, low IgG. There is a genetic test.
- Treatment is supportive and questioned on an ethical basis.
- As important is client education.

Neonatal isoerythrolysis (isoimmune or alloimmune haemolytic anaemia)

- Haemolytic condition of neonatal foals in which the mare develops antibodies against the foal's erythrocytes.
- Occurs when the foal inherits paternal RBC antigens that the mare does not have but has previously been exposed to these antigens.
- In general multiparous mares. Exposure usually occurs in a previous gestation from the same stallion. Primiparous mares can develop antibodies if they have been exposed to antigens from a blood transfusion, a biological product, or from a 'leaky placenta'.
- After the foal nurses and absorbs colostral antibodies, severe haemolytic anaemia occurs.

Pathogenesis

- The foal must have RBC paternal antigens different from those of the mare.
- A highly antigenic blood group is necessary to mount a high immune response by the mare.
- Factors Aa and Qa are the most immunogenic in horses, but it can happen with other blood groups. A 'donkey factor' is responsible for antigenicity in mule foals.
- Mares that are negative for Aa or Qa are at high risk of producing foals that will develop isoerythrolysis.

Clinical signs

- Foals are usually normal at birth. Signs develop from 8 hours to 4 days following parturition.
- Weakness, icterus, tachycardia, tachypnoea. Urine can be dark.
- Neonatal isoerythrolysis should be suspected in any foal younger than 1 week with icterus,.
- Differentials for icterus include liver disease, anorexia, and haemolysis.
- Seizures can occur if the anaemia/hypoxia is severe.
- Foals may develop diarrhoea and septicaemia with severe hypoxia, compromising the intestinal mucosa.

Diagnosis

- Laboratory findings include anaemia (PCV of 10–15%), leukocytosis from a stress response, hyperbilirubinemia (indirect), icteric to red-tinged plasma, and abnormalities from secondary complications.
- Haemoglobinuria: in severe cases foals may develop acute tubular necrosis.
- Indirect Coombs test to detect the presence of antibodies on the surface of the foal's RBCs.
- Minor cross-match (foal's RBCs + mare's serum): positive agglutination suggests neonatal isoerythrolysis, although non-specific autoagglutination is frequent in foals with isoerythrolysis.

Treatment

1. If the foal is less than 24 hours old at the time of diagnosis, it should not be allowed to nurse the mare until it is 24–48 hours of age.
2. Blood transfusion:
   - A transfusion should be considered if the anaemia is severe (PCV <15%) or if the foal is weak.
   - Sources of blood for transfusion include the mare's blood (RBCs must be washed to remove any antibodies); other donors should be cross-matched with the foal's blood prior to transfusion. If a cross-match cannot be achieved, a gelding donor is less likely to cause a reaction.
   - 20–30 mL/kg of packed red blood cells usually raise the PCV to 15–20%.
   - Transfused red blood cells have a very short-half life (2–4 days), therefore the PCV needs to be closely monitored as further transfusions may be needed.
3. Plasma transfusions may be necessary if the serum IgG concentration is low. Often foals with isoerythrolysis have a high IgG concentration.
4. Broad-spectrum antibiotics should be given to protect against opportunist infections.

Prevention

- Blood-typing of mares: alloantigens Aa and Qa are responsible for most cases; therefore mares without these alloantigens are more likely to be at risk.
- Mares can be tested late in gestation. The mare's serum can be tested against samples with known alloantigens (stallion). If there is consistent evidence of agglutination, colostrum should be withheld and the foal supplemented with colostrum from another mare that has not produced a foal by the same stallion.
- Testing at birth: the foal can be tested at the time of birth, and colostrum withheld until the test results are known.

Immune-mediated thrombocytopenia and neutropenia

- Syndrome of foals younger than 1 week of life. These foals appear clinically normal until they become depressed and develop spontaneous bleeding. They can present with thrombocytopenia alone, or thrombocytopenia and neutropenia. Often they have signs of sepsis.
- The pathogenesis appears to be similar to neonatal isoerythrolysis, but antibodies are directed to the platelets and neutrophils. These foals rarely develop immune-mediated anaemia, although it may occur. Anaemia, if present, is often the result of blood loss.
- Petechiation, epistaxis, haematomas, spontaneous bleeding.
- Laboratory abnormalities include low platelet and neutrophil counts, as well as abnormalities secondary to organ complications (acidosis, electrolyte imbalances, sepsis).
- As with isoerythrolysis, these mares are at risk of having foals with this condition in future gestations.
- Foals should not be allowed to nurse colostrum. They should receive colostrum from another mare or transfused with plasma. After 48 hours they can be placed back with the mare. The colostrum should be removed from the mare.
- These foals should be treated with antibiotics immediately as they have an incompetent immune system from the leukopenia and likely septicaemia.
- In foals with neutropenia, the use of granulocyte colony-stimulating factor has been suggested.

Ulcerative dermatitis, thrombocytopenia, neutropenia syndrome of foals

- Foals of <4 days of age present with ulcerative dermatitis, low platelet count (<30 000/μL), leukopenia (<3000/μL) and neutropenia (<2000/μL).
- Reported in Thoroughbreds and Paint horses.
- Lesions consist of oral, gingival and lingual ulcers, and erythema and exudative dermatitis on the face, around the eyes, mouth, inguinal, axillary, and perineal areas.
- Petechiation is frequent. Spontaneous bleeding (haematomas, epistaxis, melaena, haematochezia) can be present.
- Pathogenesis is unclear but it is believed that paternal antigenic stimulation of the mare and colostral antibodies are involved.
- Treatment consists of glucocorticoids (dexamethasone), antibiotics, platelet-rich plasma transfusion, and blood transfusion.
- With early intervention the prognosis is good.

Other immunopathies

Selective IgM deficiency

This has been documented in Arabians, Quarter Horses, and apparently in other breeds. Affected horses have enteritis, pneumonia, sepsis, and weight loss.

Transient hypogammaglobulinaemia

In this condition there is a delay in IgG production; it has been reported in Arabian and Thoroughbred foals. Foals develop bacterial and viral infections.

Agammaglobulinaemia

A condition – reported in Thoroughbreds, Standardbreds, and Quarter horses – in which the foals have no B cells and fail to produce immunoglobulins. Clinical presentations relate to impaired immune function and include gastrointestinal and respiratory disease. IgA and IgM are usually undetectable, and IgG is low (probably maternal).

20.12 MISCELLANEOUS CONDITIONS

Steatitis (yellow fat disease)

Steatitis is a rare disease seen primarily in pony and donkey foals, usually between 1 and 6 months of age. The aetiology is uncertain but might be associated with selenium and/or vitamin E deficiency. Clinical signs include pyrexia, inappetence, depression, tachypnoea and weakness. Firm, indurated subcutaneous plaques are palpable over the body, and the ligamentum nuchae is thickened, hard and painful. Other signs that may be present include hyperlipaemia, ventral oedema and diarrhoea. There is no effective treatment.

Glycogen branching enzyme deficiency (GBED)

This is a lethal, autosomal recessive condition of fetuses and foals of Quarter horse and Paint horse breeds. It is caused by a mutation of the glycogen branching enzyme, disrupting the metabolism of glycogen. GBED is characterized by abortion or mortality, immediately post-partum. These foals are weak, have contracted tendons, develop seizures, or die suddenly from hypoglycemia or cardiorespiratory failure. Some foals may live for a few weeks but eventually have to be euthanized.

FURTHER READING

Knottenbelt DC, Holdstock N, Madigan JE (2004) Equine neonatology, medicine and surgery. Saunders

McAuliffe SB, Slovis NM (2008) Color atlas of diseases and disorders of the foal. Elsevier

Paradis MR (2006) Equine neonatal medicine. A case-based approach. Elsevier

Chapter 21

Muscle disorders and performance problems

William V. Bernard

CONTENTS

21.1 Examination of the muscular system 451
Physical examination 451
Clinical pathology 451
Muscle biopsy 452
Electromyography 452
21.2 Congenital/familial disease 452
Myotonia 452
Hyperkalaemic periodic paralysis (HYPP) 452
21.3 Myopathy with physical causes 453
Postanaesthetic myoneuropathy 453
Exertional rhabdomyolysis 454
Fibrotic and ossifying myopathy 455
21.4 Myopathy of infectious origin 455
Clostridial myonecrosis 455
Corynebacterium pseudotuberculosis abscesses 455
21.5 Myopathy of metabolic origin 455
Hypocalcaemic tetany 455
Synchronous diaphragmatic flutter 455
Malignant hyperthermia 456
Polysaccharide storage myopathy 456
21.6 Myopathy of nutritional origin 457
Nutritional myodegeneration 457
21.7 Ionophore antibiotic toxicity 457
21.8 Atypical myopathy (atypical myoglobinuria) 457
21.9 Performance problems 458
Diagnostic evaluation 458
21.10 Causes of poor performance/exercise intolerance 458
21.11 Thermoregulatory problems 458
Pathophysiology 458
Heat stroke 458
Anhidrosis 458
21.12 Exhausted horse syndrome 458
Further reading 460

21.1 EXAMINATION OF THE MUSCULAR SYSTEM

Physical examination

- The horse should be evaluated initially from a distance. Symmetry, size and shape of the muscle groups should be assessed.
- The various muscle groups and pairs of muscles are palpated to give an impression of tone, sensitivity, asymmetry or atrophy. The horse should be given time to relax so that apprehension to palpation does not elicit a false impression.
- The horse is walked and examined for evidence of lameness, weakness or pain associated with movement. Differentiating between weakness caused by neurological disease and weakness caused by muscular disease can be difficult.

Clinical pathology

Three enzymes are routinely measured in the evaluation of muscular disease:

- Creatinine phosphokinase (CK or CPK).
- Lactate dehydrogenase (LDH).
- Aspartate aminotransferase (AST).

Creatinine phosphokinase

- CK is found predominantly in skeletal and cardiac muscle and in the brain.
- When the muscle cell wall is disrupted, CK is released from the cytoplasm and enters the extracellular fluid.
- Elevated serum CK is usually caused by myopathy rather than by neuropathy, since CK activity in nervous tissue is not sufficient to elevate serum concentrations.

© 2013 Elsevier Ltd
DOI: 10.1016/B978-0-7020-2801-4.00021-3

- Elevated serum concentrations of CK can be a sensitive indicator of muscle damage. Serum concentrations increase within hours of an insult to muscle.
- Training, transport and strenuous exercise can cause a mild elevation in serum CK, whereas diseases such as exertional rhabdomyolysis or nutritional muscular dystrophy may cause CK concentrations of thousands to hundreds of thousands of IU/L.

Lactate dehydrogenase

- Serum LDH activity is tissue non-specific, although muscle, liver and erythrocytes may be the major source of high activity.
- Elevations of serum LDH can be seen in horses with exertional rhabdomyolysis and other causes of myodegeneration, myocardial necrosis and in association with liver disease.
- To differentiate the source of LDH, it is helpful to separate it into its different isoenzyme forms by electrophoresis.

Aspartate aminotransferase

- Serum AST has high activity in skeletal muscle, liver, red blood cells and other tissues.
- AST is used to detect general cellular damage and is not muscle specific.

Interpretation of enzyme levels

- Concentrations of CK peak in 6 to 12 hours, and AST concentrations peak in 12 to 24 hours.
- The half-life of CK is 1 to 2 days, and for AST it is 7 days.
- Therefore, elevated CK levels indicate active muscle damage. If CK decreases or is normal and AST is high this indicates that myonecrosis is not continuing. If the injury is not progressive, CK will return to normal within 24 to 48 hours.
- Collection and storage of blood samples are important. Ideally, samples should be transported to the laboratory immediately. Separation of the serum or plasma should be done as soon as possible since RBC lysis can lead to release of AST and LDH.

Muscle biopsy

Percutaneous muscle biopsy allows examination of myofibres, neuromuscular junctions, nerve branches, connective tissue and blood vessels. Biopsies should be obtained from both normal and abnormal muscle tissue. Standardized sites from the gluteus medius and semitendinosus muscles are most common. Samples for biochemical analysis are frozen in liquid nitrogen. Samples for histological analysis are frozen in freon or isopentane suspended in liquid nitrogen. The clinician should check with the diagnostic laboratory as to how the samples should be handled prior to submission.

Electromyography

Electromyography and nerve conduction velocities are used most commonly to investigate neuromuscular diseases rather than primary muscle disorders.

21.2 CONGENITAL/FAMILIAL DISEASE

Myotonia

Myotonia is a skeletal muscle disorder primarily affecting the Quarter horse, characterized by a period of involuntary contraction of muscles following stimulation or voluntary motion.

Aetiology/pathogenesis

- The mode of inheritance is unknown.
- The pathophysiology is thought to involve abnormalities in sarcolemmal chloride conductance.

Clinical signs

- Primarily affects young horses less than 1 year of age.
- Primarily affects the extensor muscles of the pelvic limbs resulting in a stiff gait.
- Bilateral focal enlargement of the proximal caudal thigh and rump muscles gives the impression that the horse is overdeveloped or is 'double muscled'.
- Percussion of affected muscles induces a prolonged contraction resulting in raised lumps or 'dimpling' which lasts for a minute or more with eventual slow relaxation.
- Clinical signs usually do not progress beyond 6 to 12 months of age but can involve multiple organ systems.

Diagnosis

- Tentatively based on clinical signs, age and breed.
- Definitive diagnosis is based on an EMG examination. The high-frequency crescendodecrescendo repetitive electrical bursts produce a characteristic 'dive bomber' sound.

Treatment

- No treatment can be recommended because the pathophysiology is unknown.
- Administration of phenytoin, quinine and procainamide can provide symptomatic relief in humans but has been unsuccessful in horses.

Prognosis

- Variable, depending on the severity of the clinical signs.
- Euthanasia may be indicated in cases that progress to fibrosis and pseudohypertrophy.

Hyperkalaemic periodic paralysis (HYPP)

HYPP is a familial disorder which affects pure and crossbred Quarter horses. Affected horses experience intermittent episodes of muscle tremors and weakness which may result in collapse.

Aetiology and pathogenesis

- HYPP is inherited as an autosomal dominant trait.
- The gene responsible has been identified in descendants of the stallion Impressive.
- The pathogenesis involves an altered permeability of muscle membranes to sodium and potassium by affecting the sodium ion channel. An uncontrollable influx of sodium causes depolarization of muscle fibres resulting in uncontrollable muscle twitching and weakness.

Clinical signs

- Recurrent episodes of muscle weakness, tremors and collapse.
- Usually less than 4 years of age. HYPP has been recorded in horses as young as 2 months and as old as 15 years.
- Males are overrepresented.
- Signs often begin with stiffness, sweating and muscle fasciculations.
- Some horses 'dog-sit'; some become recumbent.
- Occasionally episodes are accompanied by respiratory noises due to paralysis of the larynx and pharynx.
- Horses remain bright and alert and respond to noise and painful stimuli.

- During episodes of clinical disease there is an increase in PCV/total protein and potassium [hyperkalaemia (serum potassium 5.0 to 12.3 mEq/L)].
- Affected horses are clinically normal between episodes.

Diagnosis

- DNA blood test is available at the University of California – Davis, and is the most reliable and effective way of identifying carriers of the HYPP gene.
- HYPP can be tentatively diagnosed on the basis of the clinical signs and signalment.
- Potassium chloride challenge test – involves administering 90–150 mg/kg of potassium chloride dissolved in water and given via a nasogastric tube following an overnight fast. A positive result is characterized by signs of HYPP in 2–4 hours. Known affected horses do not always test positive at the lower doses.

Note: It is recommended to start with the low dose and increase by 25% daily. Horses receiving the test should not be left alone because it can cause life-threatening hyperkalaemia. Once a horse demonstrates signs of HYPP, treatment should begin.

Treatment

Prior to treatment, blood samples are obtained to determine the serum concentrations of potassium and muscle enzymes.

Mild attacks (trembling but no recumbency):

- Exercise the horse by walking or lunging. This stimulates adrenaline, which aids in the replacement of intracellular potassium.
- Feed grain because the carbohydrates will supply glucose which will stimulate insulin release which promotes potassium uptake by cells.
- Acetazolamide orally. This carbonic anhydrase inhibitor will increase potassium excretion by the kidneys.

Severe attacks (horse is recumbent and unable to rise):

- Insert an intravenous catheter.
- Administer calcium gluconate 23%: 150 mL added to 1 to 2 L of 5% glucose per 500 kg body weight. The majority of affected horses will stand immediately following this treatment.
- If there is no response to calcium gluconate, administer 1 litre of 5% sodium bicarbonate IV (1 mEq/L).
- If there is no response, give 3 litres of 5% dextrose IV and monitor the serum potassium concentration.
- Once diagnosed, most cases can be managed with dietary and exercise modifications. Decrease or eliminate alfalfa to decrease potassium concentration and replace with oat or grass hay or pasture turnout.
- Pasture or paddock exercise is preferred to stall rest.
- If conservative treatment is ineffective, oral acetazolamide can be administered two to three times a day.
- Affected horses should not be bred.

21.3 MYOPATHY WITH PHYSICAL CAUSES

Postanaesthetic myoneuropathy

Pathophysiology

- Affected horses are usually large and well-muscled and have been exposed to prolonged or deep anaesthesia on a hard surface.
- Muscle compression and prolonged immobility result in muscle ischaemia and hypoperfusion.
- A history of gaseous anaesthesia, mechanical ventilation and mean arterial pressures of less than 65 mmHg for an extended period of time.
- A localized form ('compartmental syndrome') is associated with damage within osteofascial compartments.
- The generalized form is thought to be due to the combination of the compartmental syndrome, muscle sensitivity to anaesthesia, stress, lowered blood pressure and use of muscle relaxants during anaesthesia.

Clinical signs

This disorder primarily affects the triceps, quadriceps, hind limb extensors, longissimus muscles, masseter and gluteal muscles.

1. Localized form:
   - Lateral recumbency affects the triceps, deltoid and hind limb extensors.
   - Dorsal recumbency affects the longissimus and gluteals.
   - Affected muscles can be flaccid or hard, hot and painful on deep palpation and are swollen.
   - The horse is weak and unable to support its weight on the affected limb. It may have a dropped elbow appearance or knuckling of both hind fetlocks (Figure 21.1).
   - Signs may first appear 30 to 60 minutes after standing.
   - There is usually recovery within 24 hours if only a single muscle group is involved.
2. Generalized form:
   - The forelimbs, pectorals and/or hind limbs can be affected.
   - Generalized weakness or paresis (Figure 21.2).
   - Restlessness, anxiety, sweating or colic.

Figure 21.1 Dropped elbow due to post-anaesthetic neuromyopathy.

Figure 21.2 'Dog-sitting' due to post-anaesthetic myopathy affecting the hind limbs.

Diagnosis

1. History and clinical signs.
2. Elevated concentrations of CK, AST and LDH.
3. Sometimes there is myoglobinuria.

Treatment

1. Mild cases:
   - Phenylbutazone or flunixin meglumine.
   - Dimethyl sulphoxide (DMSO) IV or topically to affected muscles.
2. Severe generalized acute rhabdomyolysis:
   - Goals are to prevent more muscle damage and acute renal failure secondary to myoglobinuria, to provide analgesia, and to relieve stress.
   - NSAIDs.
   - Xylazine, detomidine or romifidine.
   - Butorphanol.
   - Meperidine.
   - Acepromazine.
   - Fluid therapy with a balanced electrolyte solution is indicated to prevent acute renal failure and to maintain normal electrolyte and acid/base balance.
   - Mannitol may be indicated in the severely affected horse.
   - Glucocorticoids may be indicated in the acute phase to reduce inflammation and muscle damage by stabilizing cell membranes and preventing lipid peroxidation of cellular membranes.

Prevention

- Proper padding and positioning under general anaesthesia; pull lower forelimb forward and separate hind limbs.
- Anaesthesia should be maintained on the lightest plane possible and mean arterial pressures maintained greater than 70 mmHg.
- Dantrolene sodium orally 1 to 2 mg/kg 1 to 2 hours prior to surgery may aid reduction of myopathy.

Exertional rhabdomyolysis

Other terms for this disorder are azoturia, 'Monday morning disease', 'tying up', paralytic myoglobinuria and myositis. Exertional rhabdomyolysis (ER) is a syndrome with many causes. The classical description is the draught horse in work that is rested for the weekend and maintained on full feed. Then when the horse returns to work it suffers an attack of the disease. Currently, the discussion of ER can be simplified if the condition is divided into two forms, sporadic and recurrent.

Aetiology

- Sporadic cases may be caused by dietary imbalances or overexertion (rapid increase in work intensity or duration).
- Chronic/recurrent cases may be related to defects in intracellular calcium regulation or glycogen metabolism.
- Highly strung mares and fillies are affected more than colts.
- Tends to be an increased incidence in certain breed lines and families of horses.

Pathogenesis of sporadic ER

- Increase exercise intensity above the foundation level of training.
- Electrolyte imbalances.
- Diets deficient in Vitamin E and/or selenium.
- Diets with a high non-structural carbohydrate.
- Heat exhaustion (see 21.11).

Pathogenesis of recurrent ER

- Abnormal intracellur calcium regulation.
- Usually seen after a horse reaches a level of fitness.
- Usually occurs during slower pace exercise.
- May occur before an event, rarely after racing.

Clinical signs

- Signs vary, depending on the extent of muscle degeneration.
- Commonly see a stiff or stilted gait and reluctance to move.
- Pain may be elicited by deep palpation of the back and hind limb musculature.
- Tachycardia, tachypnoea and pyrexia are sometimes seen.

Diagnosis

- Clinical signs.
- Increased serum concentrations of CK, AST and LDH.
- Pigmenturia/myoglobinuria.
- Fluid and electrolyte imbalances: dehydration, hypochloraemia, hypocalcaemia.
- Comparison of pre- and post-exercise serum concentrations of CK and AST (in mild cases).

Treatment

- Goals are to limit further muscle damage, to maintain fluid and electrolyte balance and to prevent acute renal failure.
- Further exercise or movement is contraindicated in most cases.

1. Mild cases:
   - Phenylbutazone or flunixin meglumine.
   - Acepromazine to increase peripheral blood flow.
   - Rest for 3 to 5 days.
   - Vitamin E and selenium orally or IM.
2. Severe cases:
   - Intravenous fluids to prevent renal failure.
   - Corticosteroids (dexamethasone).
   - Phenylbutazone or flunixin meglumine.
   - Xylazine if very painful.
   - Rest for 6 to 8 weeks before returning to training.
3. Chronic intermittent cases:
   - Dantrium sodium decreases the release of sodium from skeletal muscle fibres.
   - Phenytoin. Must be withdrawn 7 days prior to racing. Side effects may include drowsiness, ataxia and rarely seizures. Acts on ion channels including sodium and calcium.
   - Assessment of electrolyte metabolism using fractional excretion (clearance) of electrolytes (Chapter 8).

Prevention

- Address exercise management issues where appropriate, e.g. long warm up/cool down, use exercise sheets, avoid box rest days, encourage turn out to pasture.
- High-fat/low-starch diet.
- Dietary electrolyte supplementation based on FE results.
- Although without significant evidence base a wide variety of preventative ages have been recommended including: sodium bicarbonate, vitamin E and selenium, diemthylglycine, methyl sulfonyl methane, thiamine, acepromazine, phenytoin, thyroxine dantroline and diazepam.

Fibrotic and ossifying myopathy

See Chapter 17.

21.4 MYOPATHY OF INFECTIOUS ORIGIN

Clostridial myonecrosis

Aetiology and pathophysiology

- Most clostridial infections result from injections into the cervical, pectoral and hindlimb muscles.
- Local muscle necrosis, trauma or concurrent infection provide low oxygen tension which is optimal for growth of clostridial organisms.
- Introduction of spores or organisms is usually by hypodermic needles.
- Clinical signs are seen when the spores become vegetative exotoxin-producing organisms.
- *Clostridium chauvoei*, *septicum* and *perfringens* type A are most commonly isolated.

Clinical signs

- Muscle swelling, lameness, stiff gait or reluctance to lift the head, and head oedema, depending on the muscles affected.
- Affected muscles are warm to the touch with gas accumulation (crepitus).
- Laryngeal hemiplegia due to recurrent laryngeal nerve damage.
- Pyrexia, tachycardia, tachypnoea and decreased capillary perfusion.

Diagnosis

- History of intramuscular injection within the last 24 to 48 hours.
- Culture, cytology and Gram stain.

Treatment

- Antibiotics: potassium penicillin; procaine penicillin; chloramphenicol; metronidazole.
- Surgery involves fasciotomy and debridement of the affected area.
- Basic supportive care: intravenous fluids, analgesics, wound management.

Prognosis

- *Clostridium chauvoei*, septicum – poor prognosis; patients often die.
- *Clostridium perfringens* – highest survival rate.

Corynebacterium pseudotuberculosis abscesses

See Chapter 13.

This disease is also referred to as pigeonbreast, Colorado strangles and dry land distemper.

Aetiology

- The bacterium responsible for this disease is *Corynebacterium pseudotuberculosis*. It also causes ulcerative lymphangitis and contagious acne.
- Seen in the late summer and fall in Western USA and other hot, arid areas (Figure 21.3).
- There is a possible association with cutaneous habronemiasis and onchocerciasis.

Clinical signs

- Large abscesses of the pectoral muscles and less frequently of the axilla, ventral abdomen and limbs (Figure 21.3).
- Abscesses are thick walled and progressively increase in size.
- There may be ventral oedema.

Diagnosis

- Clinical signs and culture.

Treatment

- May spontaneously rupture or can be surgically incised and flushed daily.
- Antibiotics – penicillin.
- Applications of vaseline to the skin to prevent serum burn.

21.5 MYOPATHY OF METABOLIC ORIGIN

Hypocalcaemic tetany

See Chapter 11.

Synchronous diaphragmatic flutter

This condition is referred to as 'thumps'. It is seen in association with fluid and electrolyte abnormalities caused by endurance riding, exercise, transport and digestive disorders.

Figure 21.3 *Corynebacterium pseudotuberculosis* abscess in a working donkey in Egypt.
Courtesy of Zofia Lisowski MRCVS.

Aetiology and pathogenesis

- Diets high in calcium may predispose horses to synchronous diaphragmatic flutter.
- Several conditions may incite the condition: extensive exercise, transit or lactation tetany, trauma, digestive disorders and furosemide treatment.
- Metabolic acidosis with hypochloraemia, hypokalaemia and hypomagnesaemia.
- Fluid and electrolyte abnormalities may disrupt normal membrane potentials of the phrenic nerve. This allows the phrenic nerve to discharge in response to atrial depolarization.

Clinical signs

- Major clinical signs are the result of the synchronous contraction of the diaphragm with the heartbeat.
- Unilateral or bilateral twitch of the flank. This twitch can result in the characteristic 'thumping' noise.

Treatment

- Usually a transient disorder which resolves when acid–base and electrolyte abnormalities are corrected.
- Affected horses respond quickly to intravenously administered calcium solutions.
- See treatment for hypocalcaemia (Chapter 11).

Malignant hyperthermia

Malignant hyperthermia is a rare disease. It is a generalized condition in which there is progressive hyperthermia, hypercapnia and muscle rigidity associated with certain anaesthetic agents or muscle relaxants.

Aetiology

- Agents implicated are inhalation anaesthetics (e.g. halothane), suxamethonium (succinylcholine) and local anaesthetics with an amide structure.
- There may be a genetic predisposition.
- Associated anaesthetic factors are difficult induction, increased PaCO2 levels, anaesthesia time greater than 2 hours and metabolic acidosis.

Pathogenesis

- There may be a defect in the sarcoplasmic reticulum which allows high concentrations of calcium to remain in the sarcoplasm.
- This defect results in continuous muscle contraction with heat production due to the energy needed to sustain the contractions.

Clinical signs

- Muscle stiffness, fasciculations.
- Progressive rise in body temperature (42.2°C, 108°F).
- Tachycardia, sweating and variable breathing patterns.
- Severe cases may develop metabolic and respiratory acidosis.

Diagnosis

- Based on clinical signs, especially if the horse is receiving inhalational anaesthesia.

Treatment

- Discontinue anaesthesia as soon as possible.
- Decrease body temperature with the use of alcohol baths, ice baths and cool IV fluids.
- Support cardiac and respiratory function as needed.
- Dantrolene sodium 2–3.5 mg/kg IV can help alleviate signs.

Polysaccharide storage myopathy

Polysaccharide storage myopathy(PSSM) is a cause of exertional rhabdomyolysis, muscle soreness and weakness. The primary breeds affected are Quarter horses, draught horses and Warmblood breeds.

Aetiology

- Glycogen accumulates because of abnormalities in muscle-energy metabolism.
- This is a result of an inherited enzyme defect in glycogenlysis, glycolysis, or energy metabolism.
- It has a genetic basis in Quarter horses.

Pathogenesis

- Enzyme defect results in abnormal storage and utilization of glycogen.

Clinical signs

- The most common clinical sign in Warmbloods is the presence of firm, painful muscles of the back and hind quarters associated with reluctance to collect and engage the hind quarters.
- In draught breeds clinical signs are not consistently present with PSSM. When clinical signs are present they are variable and can be as severe as progressive muscle weakness and recumbency.

- Affected Quarter horses develop painful, fasciculating muscles, stiffness, sweating, weakness and reluctance to move.

Diagnosis

- Definitive diagnosis is made on the basis of muscle biopsy.

Treatment

- Management is the only current therapy available.
- Rest with turn-out, not stall confinement.
- Consistent exercise with gradual increases in training levels; duration of exercise may be more important than intensity of exercise. Days off from exercise should be limited.
- Dietary modification aimed at reducing dietary glucose intake and providing energy by adding fat to the diet.

21.6 MYOPATHY OF NUTRITIONAL ORIGIN

Nutritional myodegeneration

Termed white muscle disease or nutritional muscular dystrophy, this is a myodegenerative disease that can affect cardiac and skeletal muscle of foals and occasionally adults.

Pathogenesis

- Highly reactive oxygen-free radicals are by-products of normal cellular metabolism.
- Oxygen-free radicals can react with unsaturated fatty acids to form toxic lipid peroxidases which can disrupt cell membranes and proteins, leading to a loss of cell integrity.
- Vitamin E is an antioxidant which scavenges free radicals preventing cell disruption.
- Selenium is a component of glutathione peroxidase which converts hydrogen peroxide and lipoperoxidases into water and less harmful alcohols.
- Vitamin E and selenium deficiency allows these toxic events to occur leading to myodegeneration.

Clinical signs

1. Acute myocardial degeneration:
   - Foals display difficulty rising, tachycardia, tachypnoea, and dyspnoea.
   - Death usually occurs within 24 hours despite therapy.
   - Lesions are found in the heart, diaphragm and intercostal muscles.
2. Skeletal form:
   - Foals display muscle stiffness and weakness.
   - Tachycardia, tachypnoea, dyspnoea and failure to suckle.
   - Affected muscles may be swollen and painful on palpation.
   - Adults show signs of diffuse muscular involvement, stiff gait and weakness.
   - Depression, difficulty eating, head and neck swelling and myoglobinuria.

Diagnosis

- Cases usually occur in selenium deficient areas.
- Clinical signs.
- Laboratory findings:
  - increases in CK, AST and LDH.
  - hypochloraemia.
  - hyperkalaemia.
  - hyponatraemia.
  - myoglobinuria.
- Necropsy.
  - bilateral symmetrical myodegeneration.
  - affected muscles have pale streaks.
- Vitamin E and selenium concentrations.

Treatment

- Cardiac form: supportive treatment, but condition is usually fatal.
- Skeletal form: vitamin E/selenium supplementation. Selenium administered parenterally, vitamin E administered orally. Injectable combinations do not provide adequate concentrations of vitamin E, as vitamin E is poorly absorbed from IM sites.

21.7 IONOPHORE ANTIBIOTIC TOXICITY

These antibiotics include monensin, lasalocid and salinomycin. They are often used in the control of coccidiosis in poultry and ruminants, and as growth promoters in ruminants.

Pathophysiology

These antibiotics complex with various cations and are transported across cellular membranes. This disrupts normal ionic gradients, resulting in cellular dysfunction.

Clinical signs

Horses can suffer from an acute clinical disease or a delayed toxicity with cardiovascular effects.

1. Acute toxicity:
   - Anorexia, colic, hypovolaemic shock, ataxia, weakness, cardiovascular dysfunction.
   - Death can follow in 24 to 48 hours.
2. Chronic toxicity:
   - Exercise intolerance and cardiovascular dysfunction, usually atrial fibrillation.
   - Signs may not be seen for weeks or months.
   - Azotaemia, haematuria, haemoglobinuria, myoglobinuria.
   - Increases in CK, AST and LDH.
   - Increased hepatic enzymes and total serum bilirubin.

Diagnosis

- Clinical signs.
- History of exposure.
- Rule out other conditions.

Treatment

- Basic supportive therapy.
- Mineral oil or activated charcoal via stomach tube to decrease intestinal absorption.
- Vitamin E and selenium may be beneficial.

21.8 ATYPICAL MYOPATHY (ATYPICAL MYOGLOBINURIA)

Myopathy that resembles exertional rhabdomyolysis clinically and pathologically but that occurs in unworked or minimally worked horses. The disease has been recognized sporadically in Europe. Atypical myoglobinuria often occurs as an outbreak on the same property and primarily affects young horses kept outdoors. Adverse climatic conditions appear to predispose to the onset of disease. The clinical

course is short and often severe. Many affected horses become recumbent, and there is a high mortality rate. The clinical features, diagnostic features and treatment are similar to those of exertional rhabdomyolysis.

21.9 PERFORMANCE PROBLEMS

Poor performance can be defined as a suboptimal level of performance or the inability of a horse to perform at a previous level of exercise. Special attention must be given to the systems responsible for performance: the respiratory, cardiovascular, musculoskeletal and haematopoietic systems.

Diagnostic evaluation

History

An accurate history should include age, sex, breed, intended use and training programme. Performance history should include details of the previous level of exercise, the onset of problems (training, competition, racing), whether poor performance is consistent or intermittent, and whether there is any associated respiratory noise or lameness. Most horses either perform badly throughout exercise or develop exercise intolerance and finish poorly.

Clinical examination

A thorough physical examination is indicated with special attention to the respiratory, cardiovascular and musculoskeletal systems. The heart should be auscultated detect murmurs and arrhythmias. The lung fields should be auscultated with and without a rebreathing bag, and the chest should be percussed. The horse should be evaluated for any subtle lameness.

Diagnostic tests

- Haematology, plasma biochemistry.
- Electrocardiography.
- Endoscopy.
- Transtracheal wash/bronchoalveolar lavage.
- Muscle biopsy.
- Exercise testing.
- Radiology.

21.10 CAUSES OF POOR PERFORMANCE/ EXERCISE INTOLERANCE

See Table 21.1.

21.11 THERMOREGULATORY PROBLEMS

Pathophysiology

Working muscle requires a tremendous increase in metabolic rate. Since only a portion of the energy is used, the rest accumulates as heat which is removed by the vascular system. The vascular system removes the heat from the core to the skin where heat exchange can occur. Heat dissipation occurs via four mechanisms: radiation, convection, conduction and evaporation. The first three require a favourable heat gradient between the horse and the environment to be effective. Evaporation is the most important mechanism for the exercising horse to dissipate heat. A racehorse can lose 10 to 15 L of sweat in a race, and an endurance horse may lose 10 to 15 L per hour. Heat associated illnesses are usually seen in horses exercising in a hot, humid environment where there is a competing demand for heat dissipation.

Heat stroke

Aetiology

Heat stroke usually occurs in horses that are transported in poorly ventilated trailers or horses that are overworked during hot, humid weather. The major contributing factor to heat stroke is dehydration due to sweat loss which compromises the horse's ability to lose heat. Other contributing factors are long hair coat, obesity, heavy blanketing, extensive skin damage or anything that can affect sweat evaporation.

Clinical signs

- Tachycardia.
- Tachypnoea.
- Pyrexia (>40°C, 104°F).
- Restlessness, depression, disorientation, collapse or death.
- Sweating may be profuse but usually less than expected.
- The skin is hot to the touch.

Treatment

1. Control body temperature:
   - Cold water or alcohol bath.
   - Provide good ventilation – fans.
2. Restore hydration:
   - Intravenous lactated Ringer's solution.
   - 0.9% NaCl + 10–15 mEq KCl/L.
3. Antipyretics:
   - Dipyrone.
   - Phenylbutazone.
   - Flunixin meglumine.

Anhidrosis

See Chapter 9.

21.12 EXHAUSTED HORSE SYNDROME

Equine fatigue or exhaustion is the inability of a horse to maintain a given level of exercise.

Aetiology and pathophysiology

It can involve brief maximal exercise (racing) or prolonged submaximal exercise (endurance).

- Involves a combination of impairment of excitation–contraction coupling, loss of energy production, depletion of energy substrates (glycogen) and fluid and electrolyte imbalances.
- Exercise leads to an increased metabolic rate and increased use of energy which result in increased generated heat. In this case sweating is the primary way of thermoregulation. This can cause fluid losses of 20 to 40 L or more.
- Equine sweat is hypertonic and contains sodium, potassium, chloride and magnesium. These electrolytes tend to be slightly decreased in serum, but serum concentrations do not necessarily reflect the total body losses.
- The energy consumed can deplete glycogen stores but blood glucose concentration is usually within normal limits.

Table 21.1 Causes of poor performance

Respiratory causes	Non-respiratory causes
Obstructive upper airway diseases	**Metabolic/systemic causes**
Laryngeal hemiplegia (recurrent laryngeal neuropathy). Epiglottic entrapment. Arytenoid chondritis. Guttural pouch infection. Aryepiglottic fold entrapment. Tracheal stenosis, stricture, collapse. Arytenoid chondroma. Ethmoid haematoma. Nasal polyps. Pharyngeal and subepiglottic cysts. Rhinitis, nasal granuloma.	Anaemia. Fluid and electrolyte abnormalities. Anhidrosis. Heat exhaustion. Generalized granulomatous disease. Thyroid adenoma/adenocarcinoma. Hyperparathyroidism. Liver disease. Myeloproliferative disease. Diarrhoea. Neoplasia.
Lower airway diseases	**Musculoskeletal causes**
Exercise-induced pulmonary haemorrhage. Chronic obstructive pulmonary disease (COPD). Viral infections; EHV-1, rhinovirus, reovirus, adenovirus. Bacterial pneumonia. Pleuritis. Pulmonary abscesses. Interstitial pneumonia.	Exertional rhabdomyolysis. Degenerative joint disease. Fracture. Osteochondrosis, bone cysts. Tendinitis, desmitis. Hoof imbalance. Back problems. Sacroiliac problems. Chronic sarcocystosis. Other causes of pain or lameness.
Non-respiratory causes	**General causes**
Cardiovascular causes	Obesity. Unfit horse. Poorly trained horse. Genetically slow horse.
Atrial fibrillation. Heart block. Ventricular premature beats. Congestive cardiac failure. Mitral insufficiency. Tricuspid insufficiency. Ruptured chordae tendinae. Pulmonic stenosis. Endocarditis. Myocarditis. Pericarditis. Cor pulmonale. Aortoileofemoral arteriosclerosis or thrombosis.	**Neurological causes**
	Equine protozoal myelitis. Equine cervical vertebral malformation.

- The decrease in chloride, potassium and magnesium can alter membrane potentials and neuromuscular transmission, causing gastrointestinal stasis, cardiac arrhythmias, muscle spasms and cramps.
- Thirst is suppressed in the exhausted horse for two reasons. First the hypothalamus is only responsive to blood osmolarity which is hypotonic due to the loss of hypertonic sweat. Secondly intestinal motility is decreased in exhausted horses; this often leads to small intestinal distension causing pain and this decreases the desire to drink.

Clinical signs

- Pyrexia.
- Tachycardia, tachypnoea.
- Dehydration.
- Decreased or absent borborygmi.
- Concentrated or absent urine.
- Increased jugular filling.
- Muscle cramps/spasms.
- Cardiac abnormalities.
- Synchronous diaphragmatic flutter may be present.

Diagnosis

- History and clinical signs.
- Serum electrolytes: hypokalaemia, hypocalcaemia, hyponatraemia.
- Mixed respiratory and metabolic alkalosis, or purely metabolic alkalosis.
- Bicarbonate may be normal or increased.

Treatment

Goals are to restore circulating blood volume, correct electrolyte abnormalities, provide a metabolizable source of energy and to prevent acute renal failure. This can be accomplished by administering oral or intravenous fluids.

1. Intravenous fluids:
   - Polyionic solutions, e.g. lactated Ringer's solution.
   - 0.9% NaCl + 10–15 mEq KCl/L.

- Dextrose 5% or 50% can be added to lactated Ringer's solution to provide 50–100 g/h.
- If needed, calcium solutions (calcium borogluconate) 100–300 mL of 20% solution can be given slowly.

2. Oral fluids:
   - Should be isotonic or hypotonic.
   - 5 to 8 L at a time; can be repeated every 30–60 minutes.
   - Isotonic fluids: 1 tablespoon NaCl, 1 tablespoon Lite salt per gallon of water.
   - Hypertonic fluids should be avoided because they draw fluid in from the depleted intracellular fluid.
3. Analgesics if needed (xylazine or detomidine).
4. Non-steroidal anti-inflammatory drugs can be used but have the potential to produce toxicity in dehydrated horses.
5. Glucocorticoids are controversial due to the possibility of inducing laminitis.
6. Phenothiazine-derived tranquillizers should not be used, because they may cause collapse and death.

FURTHER READING

Aleman M (2008) A review of equine muscle disorders. Neuromuscul Disorders 18:277–287

Beech J (1997) Chronic exertional rhabdomyolysis. Veterinary Clinincs of North America Equine Practice 13:145–168

Brown CM, Kaneene JB, Walker RD (1988) Intramuscular injection techniques and the development of clostridial myositis or cellulitis in horses. Journal of the American Veterinary Medical Association 193:668

Cox JH, De Bowes RM (1990) Episodic weakness caused by hyperkalemic periodic paralysis in horses. Compendium on Continuing Education for the Practicing Veterinarian 12:83

Hodgson DR, Rose RJ (1994) The athletic horse. Principles and practice of equine sports medicine. W.B. Saunders, Philadelphia

McCue ME, Ribeiro WP, Valberg SJ (2006) Prevalence of polysaccharide storage myopathy in horses with neuromuscular disorders. Equine Veterinary Journal Suppl (36):340–344

Votion DM, Serteyn D (2008) Equine atypical myopathy: a review. Veterinary Journal 178:185–190

Wijnberg ID, Back W, de Jong M et al (2004) The role of electromyography in clinical diagnosis of neuromuscular locomotor problems in the horse. Equine Veterinary Journal 36:718–722

Chapter 22

Metabolic diseases and toxicology

Tim S. Mair, Sandy Love

CONTENTS

22.1 Calcium metabolism 462
Complexed calcium 462
Protein-bound calcium 462
Ionized calcium 462
22.2 Calcium homeostasis 462
Parathyroid hormone (PTH) 462
Calcitonin 462
1,25-Dihydroxycholecalciferol 462
22.3 Hypercalcaemia 462
Renal failure 462
Plant toxicity 462
Other causes of hypercalcaemia 462
22.4 Hypocalcaemia 462
Lactation tetany/transport tetany/idiopathic hypocalcaemia 462
Synchronous diaphragmatic flutter 463
Blister beetle (cantharidin) toxicosis 463
Other causes of hypocalcaemia 463
22.5 Phosphorus metabolism 463
22.6 Hyperphosphataemia 463
Secondary nutritional hyperparathyroidism 463
22.7 Hypophosphataemia 463
22.8 Sodium metabolism 463
22.9 Hypernatraemia 463
22.10 Hyponatraemia 463
22.11 Magnesium metabolism 463
22.12 Hypermagnesaemia 463
22.13 Hypomagnesaemia 463
22.14 Potassium metabolism 463
22.15 Hyperkalaemia 464
Hyperkalaemic periodic paralysis 464
22.16 Hypokalaemia 464
22.17 Free fatty acid metabolism 464
22.18 Acid–base balance 464
Collection of blood for blood gas analysis 464
Metabolic acidosis 464
Respiratory acidosis 464
Metabolic alkalosis 465
Respiratory alkalosis 465
22.19 Toxicology 465
22.20 Toxic plants 465
Plants containing pyrrolizidine alkaloids 465
Castor bean (*Ricinus communis*) 465
Plants producing CNS dysfunction 465
Plants containing cyanogenic glycosides 466
Plants containing cardiac glycosides 466
Oak (*Quercus* spp.) 466
Maple (*Acer* spp.) 466
Black walnut (*Juglans nigra*) 466
Wild jasmine (*Cestrum diurnum*) 466
Plants causing photosensitization 466
22.21 Insecticides 467
Chlorinated hydrocarbons 467
Organophosphorus and carbamate insecticides 467
22.22 Rodenticides 467
Warfarin and other anticoagulants 467
Strychnine 467
ANTU (alpha-naphthylthiourea) 467
Arsenic 467
22.23 Heavy metals 467
Lead 467
Selenium 467
Zinc 467

© 2013 Elsevier Ltd
DOI: 10.1016/B978-0-7020-2801-4.00022-5

Arsenic 467
Other heavy metals 467
22.24 Anthelmintics 467
Carbon tetrachloride 467
Phenothiazine 467
22.25 Petroleum products 467
22.26 Feed-associated poisonings 468
Ionophores 468
Antibiotics 468
Blister beetle (cantharidin) poisoning 468
Aflatoxin 468
Further reading 468

22.1 CALCIUM METABOLISM

Calcium is one of the most abundant minerals in the body. Ninety-nine per cent of calcium (Ca^{2+}) stores are in the bones and teeth; these serve as reserves.

Calcium is essential for neuromuscular activity, cell membrane permeability, muscle contraction, and blood clotting.

Calcium exists in three forms within the intravascular compartment: complexed, protein-bound and ionized.

Complexed calcium

Calcium complexes with phosphate, citrate and sulphate.

Protein-bound calcium

- Represents 40–50% of total calcium.
- Binds with albumin.
- Serum protein concentration (especially albumin concentration) should be considered when interpreting the results of blood calcium estimations.

Ionized calcium

- Ionized calcium is the biologically active form.
- This represents 40–60% of the total calcium stores.
- Alkalosis increases protein-binding of calcium, thereby reducing the concentration of ionized calcium.
- Acidosis decreases protein-binding thereby increasing the concentration of ionized calcium.

22.2 CALCIUM HOMEOSTASIS

Parathyroid hormone (PTH)

See Chapter 9.

- Produced by the parathyroid gland.
- Secretion is stimulated by hypocalcaemia.
- Causes an increase in the production of vitamin D[1,25-(OH)2D].

Calcitonin

- Produced by C-cells of the thyroid gland.
- Secreted in response to hypercalcaemia.
- Inhibits renal resorption of calcium and resorption of calcium from the bone.

1,25-Dihydroxycholecalciferol

- Vitamin D [1,25-(OH)2D].
- Stimulates absorption of calcium in the small intestine.
- Hypocalcaemia causes an increase in PTH and 1,25-dihydroxycholecalciferol which stimulates an increase in intestinal absorption of calcium and an increase in bone resorption.

22.3 HYPERCALCAEMIA

Serum calcium is more than 3.5 mmol/L in hypercalcaemia.

Renal failure

See Chapter 8.

- Both acute and chronic renal failure may cause hypercalcaemia, but hypercalcaemia is present in less than 50% of renal disease cases.
- Possibly due to a decrease in renal excretion of calcium due to a decrease in glomerular filtration rate, renal epithelial death, and decreased gastrointestinal absorption due to decreased 1,25-dihydroxycholecalciferol.
- Possibly due to a subnormal parathyroid hormone activity.

Plant toxicity

1. Wild jasmine, day-blooming jasmine (*Cestrum diurnum*):
   - Found in tropical and sub-tropical America and the Caribbean islands.
   - Toxic all year round – all parts of the plant are toxic.
   - Clinical signs of toxicity are weight loss, stiffness, progressive lameness and eventually recumbency.
   - Dystrophic calcinosis of the elastic tissues of the heart, arteries, tendons and ligaments.
   - Contains a steroidal glycoside that is hydrolysed in the digestive tract to vitamin D_3.
   - Causes an increase in intestinal absorption of calcium beyond that which can be physiologically accommodated.
2. Other plants that cause similar syndromes:
   - *Solanum malacoxylon* (South America).
   - *Solanum sodomauim* (Hawaii).
   - *Trisetum flavescens* (Germany).

Other causes of hypercalcaemia

1. Excessive or rapid administration of intravenous calcium.
2. Neoplasia (pseudohyperparathyroidism – see Chapter 9).

22.4 HYPOCALCAEMIA

See also Chapter 26.

Lactation tetany/transport tetany/idiopathic hypocalcaemia

See Chapter 11.

Synchronous diaphragmatic flutter

See Chapter 21.

Blister beetle (cantharidin) toxicosis

Poisoning occurs by horses eating alfalfa contaminated by blister beetles. The toxin (cantharidin) is contained in the lymph of the beetles.

Hypocalcaemia is frequently seen with this condition. The pathophysiology is unknown. Clinical signs vary with the dose of toxin and include anorexia, colic, depression, straining to urinate and diaphragmatic flutter. Therapy is symptomatic.

Other causes of hypocalcaemia

- Malignant hyperthermia (see Chapter 21).
- Pancreatic disease.
- Exertional rhabdomyolysis (see Chapter 21).
- Oxalate toxicity.
- Acute renal failure (see Chapter 8).
- Tetracycline therapy.
- Furosemide therapy.
- Excessive sodium bicarbonate administration.

22.5 PHOSPHORUS METABOLISM

Phosphorus is stored in the bones and teeth, in close association with calcium. Intracellular phosphorus is utilized as adenosine triphosphate (ATP), the primary form of energy in the cell. Phosphorus in the extracellular fluid is an essential part of acid–base balance. Phosphorus is regulated by dietary factors, vitamin D, parathyroid hormone and calcitonin. Young horses have a higher serum concentration of phosphorus than adults.

22.6 HYPERPHOSPHATAEMIA

Secondary nutritional hyperparathyroidism

See Chapter 9.

22.7 HYPOPHOSPHATAEMIA

1. Seen with chronic renal failure (see Chapter 8).
2. Seen with excessive ingestion of *Brassica* spp. (turnips, swedes, rape).
3. Also seen with chronic wasting states, starvation, and with deficient dietary sources.

22.8 SODIUM METABOLISM

Sodium represents 90% of the positive ions of the extracellular fluid. Rapid, major changes in sodium concentrations can lead to neurological signs. The normal serum range is 130–140 mEq/L.

Plasma sodium concentration provides information about the relative amounts of water and electrolytes in the extracellular fluid, and, by inference, on the state of intracellular hydration.

22.9 HYPERNATRAEMIA

See also Chapter 26.

Causes

- Dehydration.
- Aggressive administration of hypertonic fluids.

It may result in haemoglobinaemia and haemoglobinuria.

If hypernatraemia is longstanding, correction must be slow; rapid correction can lead to severe cerebral oedema.

22.10 HYPONATRAEMIA

See also Chapter 26.

In hyponatraemia the serum concentration is less than 110 mEq/L.

It can occur with colic, renal disease, iatrogenic/idiopathic water overloading, aggressive enema administration and uroperitoneum in foals.

22.11 MAGNESIUM METABOLISM

The normal serum concentration is 0.9–1.2 mmol/L. Approximately 80% of magnesium is diffusible, with 35% of serum magnesium bound to protein.

22.12 HYPERMAGNESAEMIA

This is rare. It may be associated with CNS depression and cardiac depression.

Causes

- Can occur as a result of overzealous oral administration of epsom salt as a cathartic.
- May also occur in renal disease, liver disease and after glucose ingestion.

22.13 HYPOMAGNESAEMIA

See also Chapter 26.

The serum magnesium is less than 0.6 mmol/L in hypomagnesaemia. It can cause central nervous system irritability (hyperresponsiveness), which can lead to nervousness, muscle tremors, ataxia and seizures.

Causes

- Grass tetany.
- Cantharidin toxicosis.
- Endurance exercise.
- Renal disease with high urine output.

22.14 POTASSIUM METABOLISM

The plasma concentration of potassium is largely dependent on renal function and does not necessarily reflect potassium state of the body cells. Normal serum potassium is 4.2 mmol/L.

22.15 HYPERKALAEMIA

See also Chapter 26.

Causes

- Frequently present with uroperitoneum.
- Transfer of potassium from intracellular to extracellular fluid:
  - Haemolysis.
  - Muscle damage with reduced renal excretion.
  - Acidosis.
- Reduced renal excretion.

Hyperkalaemia causes peaking or 'tenting' of the T waves of an ECG.

Hyperkalaemic periodic paralysis

See Chapter 21.

22.16 HYPOKALAEMIA

See also Chapter 26.

Hypokalaemia is caused by a decrease of oral intake, increased loss via gastrointestinal tract, or shifts between the intra-and extracellular compartments due to acid–base imbalance.

Specific causes

- Frequently seen in horses with gastrointestinal disturbances (colic, diarrhoea, equine monocytic erhlichiosis).
- Can be seen with renal tubular acidosis.
- Polyuria.
- Alkalosis.

22.17 FREE FATTY ACID METABOLISM

See Chapter 9.

22.18 ACID–BASE BALANCE

See also Chapter 26.

Protein metabolism results in the formation of hydrogen ions (H^+). This represents fixed acid. Carbohydrate and fat metabolism results in the formation of CO_2. This represents volatile acid.

The body maintains a homeostasis by buffering the acids produced via metabolism.

- A buffer can accept hydrogen ions to minimize change in pH.
- The bicarbonate–carbonic acid system is used to monitor acid–base in the horse.
- Bicarbonate is the primary buffer of the extracellular fluid.
- Proteins and phosphate are the primary buffers of the intracellular fluid.

Acid/base disturbances arise from either metabolic or respiratory imbalances. In decreasing order of frequency, acid–base disturbances include:

- Metabolic acidosis.
- Respiratory acidosis.
- Metabolic alkalosis.
- Respiratory alkalosis.

These disturbances are considered either primary or compensatory.

Collection of blood for blood gas analysis

A 2- to 5-mL syringe should be precoated with lithium heparin (1000 U/mL). All excess heparin should be removed from the syringe to avoid dilution of the sample. All air bubbles should be removed from the syringe, and the needle must be capped with a rubber stopper. If the sample is exposed to air, PCO_2 is decreased, and pH and PO_2 are increased.

Common sites for collection of arterial blood include carotid, facial, transverse facial, submandibular and greater metatarsal arteries. The greater metatarsal artery is used only in the anaesthetized horse to avoid self-injury.

Sites in the foal include greater metatarsal, brachial, radial, carotid, facial and femoral arteries.

The arterial pulse should be palpated, and the area disinfected. A 20-gauge needle (adults) or 25-gauge needle (foals) should be used for arterial puncture. After the artery has been punctured, the artery should be compressed for 5 minutes to avoid haematoma formation.

The sample should be analysed as soon as possible (within 30 minutes or less), otherwise the sample can be stored on ice for up to 2 hours until analysed.

Metabolic acidosis

Both pH and bicarbonate concentrations are reduced.

- Seen in normal horses in response to exercise (transient).
- Frequently seen in:
  - hypovolaemic shock.
  - surgical colic involving strangulated bowel (compartmentalization of fluid).
  - diarrhoea.
  - endotoxic shock.
- Sometimes seen in:
  - peritonitis.
  - ruptured bladder.
  - renal failure.
  - renal tubular acidosis.

The compensatory respiratory response is an increase in respiratory rate to lower PCO_2. This response starts immediately and is at maximal effect within hours.

Treatment

- Diagnosis and treatment of the underlying disease that has caused the metabolic acidosis is frequently all that is needed to restore normal acid–base balance.
- In severe acidosis, therapy with $NaHCO_3$ is necessary. A general rule of thumb is to provide half of the base deficit rapidly, and the other half slowly over 24 hours. The dose of $NaHCO_3$ is calculated as:

$$NaHCO_3 \text{ needed} = 0.4 \times \text{body weight (kg)} \times \text{base deficit}$$

Respiratory acidosis

This is uncommon. It causes a decreased pH with an increase in PCO_2.

It may be seen in association with a severe decrease in ventilatory efficiency:

- Upper airway obstruction.
- Pneumonia.
- Pleuritis.
- CNS depression (CNS disease or drugs).
- Respiratory distress syndrome.

The metabolic compensatory response is renal retention of bicarbonate. This response starts immediately but does not reach maximal effect for several days.

This condition is a result of poor ventilation, not deficient bicarbonate, therefore bicarbonate therapy is contraindicated.

Treatment

- Rapid diagnosis and treatment for any underlying cause of alveolar hypoventilation.
- Any obstruction of ventilation (i.e. airway obstruction, pleural fluid, pneumothorax) must be removed.
- Oxygen therapy is indicated in acute respiratory distress. O_2 therapy can lead to a decrease in ventilation as hypoxaemia becomes the key driving force of ventilation with long-standing hypercapnia.
- Mechanical ventilation may be necessary. Arterial blood gases should be monitored to properly adjust ventilation rate and depth.

Metabolic alkalosis

Metabolic alkalosis causes an increase in both pH and bicarbonate concentration. Decreased circulating fluid volume, chloride or potassium depletion all lead to the impairment of renal bicarbonate excretion.

1. Occurs in conditions that cause excessive amounts of gastric reflux:
   - duodenitis–proximal jejunitis.
   - ileus.
   - gastrointestinal obstruction (most of these cases are in fact associated with a metabolic acidosis).
2. Also seen in endurance horses after massive sweating.
3. Can be seen with excessive bicarbonate administration.

Treatment

- Diagnosis and treatment of the underlying disease.
- Renal bicarbonate excretion is linked to H^+, and loss of H^+ leads to renal inability to excrete bicarbonate. Administration of fluids containing chloride and potassium allows renal excretion of excess bicarbonate.

Respiratory alkalosis

- Rare. Caused by hyperventilation.
- Occasionally seen with hypoxaemia, fear, pain.
- Treatment is by removal of underlying cause.

22.19 TOXICOLOGY

Basic therapy of poisonings:

1. Prevent further exposure.
2. Establish a tentative diagnosis of the cause/ poison involved.
3. Delay further absorption.
4. Administer specific antidotes/treatments.
5. Hasten elimination of absorbed poison.
6. Administer supportive therapy.

Emergency treatment:

1. Begin treatment promptly.
2. Retain samples for analysis – blood, urine, faeces.
3. Keep the horse warm and comfortable during therapy.

In cases of ingested poisons, gastric lavage may be attempted (either in the conscious horse or under general anaesthesia) to prevent further absorption of toxin. Alternatively, a laxative e.g. mineral oil may be administered to speed emptying of the gastrointestinal tract. Activated charcoal should be administered by nasogastric tube (250–500 g in 2–4 L water) to adsorb toxin in the digestive tract; this dose may be repeated in 12 hours.

In cases of skin exposure, the coat should be washed with a mild detergent and plenty of water.

22.20 TOXIC PLANTS

Plants containing pyrrolizidine alkaloids

A large number of plants contain pyrrolizidine alkaloids. Most of these plants are unpalatable, and horses eat them only when other forage is scarce, or when the plant is baled in hay or pelleted, or when the plant has been killed by herbicides.

This group of plants include:

- Ragworts (*Senecio* spp.): *S. jacobaea* (stinking willie), *S. vulgaris* (common groundsel).
- Fiddleneck (*Amsinckia* spp.).
- Rattleweed (*Crotalaria* spp.).
- Hound's tongue (*Cynoglossum officinale*).
- Salivation Jane (*Echium lycopsis*).

Toxicity usually occurs from chronic ingestion of the plants over periods of weeks to months. The alkaloids damage the liver and result in weight loss and inappetence, followed by specific signs of liver damage and hepatic encephalopathy.

See Chapter 3 (liver disease).

Castor bean (*Ricinus communis*)

Castor bean is grown in southern USA. The seeds contain a phytotoxin, ricin. The seeds may be eaten when mixed with other feedstuffs.

Acute toxicity is characterized by anaphylaxis and shock. Signs include incoordination, sweating, muscle spasms, tachycardia, tachypnoea, diarrhoea (may be haemorrhagic), straining and colic. Serum liver enzymes may be elevated due to liver damage. Death occurs within 36 hours and is preceded by convulsions.

Plants producing CNS dysfunction

1. Locoweed and Darling pea (*Astragalus* spp., *Oxytropus* spp., *Swainsona* spp.) – see Chapter 11.
2. Yellow star thistle (*Centaurea soltitialis*) and Russian knapweed (*C. repens*) – see Chapter 11.
3. Birdsville (*Indigophera enneaphylla*) – see Chapter 11.
4. Water hemlock, cowbane (*Cicuta* spp.), water dropwort, and dead men's fingers (*Oenanthe crocata*). Grow along waterways and in marshy ground. All parts of the plants are poisonous. Signs occur 10 to 60 minutes after ingestion. Pupillary dilation, excitement, teeth grinding, salivation, muscle fasciculations, laboured breathing, ataxia, convulsions and death (often within 1–8 hours). Horses surviving longer than this may recover over the next 1–2 days.
5. Oleander (*Nerium oleander*). A native of the Mediterranean, it is cultivated in warmer areas of the world as a hedging and landscaping plant. It contains numerous cardenolides that have cardiotoxic effects similar to digoxin. Horses eat the plant only

when it is wilted. Toxicity causes depression, excessive salivation, profuse diarrhoea, colic, heart arrhythmias and murmurs, sweating, muscle tics, coma and death.

6. White snakeroot (*Eupatorium rugosum*). A woodland plant found in midwest USA. Contains a complex mixture of sterols and ketones collectively referred to as Tremetol. Signs of toxicity include excessive sweating, dysphagia, reluctance to move, sluggishness, stiffness, ataxia. Death occurs after about 2 days.
7. Yew (*Taxus* spp.). Contains an alkaloid, taxine. Toxicity usually occurs when horses are given access to trimmings or debris after storms. The horse is extremely susceptible to yew toxicity, and one mouthful can be fatal. Death occurs rapidly (within 5 minutes), and there are rarely any other clinical signs.
8. Bracken fern (*Pteridium aquilinum*). Horses usually consume bracken in late summer, when other forage is scarce, or when it is used as bedding. The toxic agent is thiaminase, which results in myelin degeneration in peripheral nerves. The plant is usually consumed over a period of several months prior to the onset of clinical disease. Signs include weight loss, weakness, progressive incoordination and staggering, muscle tremors, cardiac arrhythmias, recumbency, clonic convulsions and opisthotonus. Death occurs within 2 to 10 days. Treatment with thiamine hydrochloride IV or IM is often successful if begun early in the clinical disease.
9. Horsetail, marestail (*Equisetum arvense*). The plant is usually consumed in hay. Toxicity and clinical signs are similar to that of bracken fern.
10. Hemlock (*Conium maculatum*). Horses usually consume the plant in the spring when it is palatable. Toxicity is due to several pyrimidine alkaloids, especially the highly toxic gamma-coniceine. Signs occur about 2 hours after ingesting the plant and include posterior incoordination, trembling, colic, excessive salivation, frequent urination, recumbency and coma. Death occurs in severe cases.
11. Nightshades (*Solanum* spp.). Nightshades are not normally consumed by horses, unless it is present in hay. Clinical signs include depression, dyspnoea, muscle tremors, weakness, paralysis, colic and diarrhoea.
12. Caley pea (*Lathyrus hirsutis*), sweet pea (*L. odoatus*) and everlasting pea (*L. latifolius*). Contain toxic amino acids and nitrites, especially in the seeds, that cause a variety of neurologic, musculoskeletal and blood vessel abnormalities, collectively referred to as lathyrism. Toxicity usually occurs following ingestion of the plant in baled hay. The clinical disease is characterized by incoordination without paresis, stiffness and difficulty getting up. The disease is progressive as long as the horse continues to eat the plant resulting in neuromuscular degeneration and laryngeal paralysis. Residual signs may be seen for years, and the horse may develop a stringhalt-like gait (see Chapter 11).
13. Ryegrass, Bermuda grass and Dallas grass – see Chapter 11.

Plants containing cyanogenic glycosides

A large number of plants contain glycosides that release hydrogen cyanide and hydrocyanic acid on hydrolysis in the gastrointestinal tract. These include hydrangeas (*Hydrangea* spp.), flax (*Linum* spp.), various cherries (*Prunus* spp.), and Sudan grass (*Sorghum* spp.). Cyanide blocks cytochrome oxidase, which prevents oxygen uptake by tissues. Clinical signs are due to tissue anoxia despite adequate blood oxygenation. Signs include dyspnoea, trembling, ataxia and recumbency. Death occurs from respiratory arrest.

Treatment includes IV administration of sodium nitrite and sodium thiosulphate. Sodium thiosulphate can also be given orally to fix free cyanide in the stomach.

Sorghum grass ataxia is described in Chapter 11.

Plants containing cardiac glycosides

Ingestion of milkweeds (*Asclepias* spp.), azaleas and rhododendrons (*Rhododendron* spp.) and foxglove (*Digitalis* spp.) may result in sudden death. Clinical signs may be observed, including anorexia, colic and diarrhoea. Atropine may be helpful in countering the cardiovascular effects.

Oak (*Quercus* spp.)

Acorn poisoning is occasionally encountered in the UK at the end of long, dry summers. Acorns and oak contain tannic acid and other tannins. Toxicity results in renal failure and gastrointestinal damage. Clinical signs include depression, anorexia, weakness, colic, constipation followed by diarrhoea, incoordination, dysphagia and haematuria. Death from stomach rupture has been reported. There is no specific treatment, and the prognosis is poor once renal failure has developed.

Maple (*Acer* spp.)

Toxicity is most commonly caused by red maple (*Acer rubrum*). Horses usually ingest the plant in the form of wilted leaves. Clinical signs develop several days after eating wilted leaves and include anorexia, depression, methaemoglobinaemia and weakness. This is followed in 24 hours by haemolysis, anaemia, Heinz body formation, haemoglobinuria, icterus, cyanosis and respiratory distress. Severely affected horses become comatose after 4 to 5 days and often die. Mildly affected horses usually recover after 7 to 14 days.

Treatment for methaemoglobinaemia with methylene blue is not highly effective. Intravenous fluids and blood transfusions may be necessary to correct the anaemia and to support renal function. Ascorbic acid may be helpful in conjunction with anti-inflammatory drugs.

Black walnut (*Juglans nigra*)

Black walnut is used in the furniture industry. Poisoning occurs when horses are bedded on shavings containing as little as 5–20% of black walnut. Clinical signs are usually seen about 1–3 days after exposure. Signs include depression, pyrexia, laminitis, reluctance to move and severe oedema of the limbs. Treatment is directed towards treating the laminitis (see Chapter 16).

Wild jasmine (*Cestrum diurnum*)

See Section 22.3.

Plants causing photosensitization

Photosensitization results from the interaction of light with photodynamic chemicals, resulting in cellular damage in the skin. Primary photosensitization occurs following the ingestion of plants containing photodynamic substances; these substances reach the skin following absorption from the alimentary tract. Secondary photosensitization occurs in association with liver damage. Liver dysfunction can result in the accumulation of phylloerythrin, a photodynamic breakdown product of chlorophyll.

Primary photosensitivity has been associated with the ingestion of St Johnswort (*Hypericum perforatum*), clovers (*Trifolium* spp.), vetches (*Vicia* spp.) and buckwheat (*Fagopyrum* spp.). Clinical signs are seen several days after ingestion. Erythema and oedema of the unpigmented areas of skin occurs, followed by blistering and sloughing of the skin. The affected areas are painful, and the horse may seek out shade. Horses make a full recovery if kept out of the sun and are removed from the source of the plant.

22.21 INSECTICIDES

Chlorinated hydrocarbons

Toxicity is usually seen within an hour of exposure. Clinical signs include apprehension, hyperexcitability, muscle spasms, hyperaesthesia, and repeated tonic–clonic seizures. Seizures may last several hours, and the horse may die or recover slowly.

There is no specific therapy.

Organophosphorus and carbamate insecticides

See Chapter 11.

22.22 RODENTICIDES

Warfarin and other anticoagulants

Produce coagulation system defects by antagonizing vitamin K. Clinical signs include epistaxis, bloody diarrhoea, subcutaneous haematomas and lameness due to haemarthrosis. Occasionally, massive haemorrhage into the abdomen or thorax may result in sudden death.

Treatment involves replacing inhibited coagulation factors (blood transfusion) and administering vitamin K_1.

Strychnine

Strychnine antagonizes inhibitory spinal cord neurons, which results in convulsions and seizures. There is acute onset of seizures that may resemble tetanus. The seizures rapidly worsen ending in death in less than one hóur. Rigor mortis occurs rapidly. There is no specific therapy.

ANTU (alpha-naphthylthiourea)

ANTU increases the permeability of lung capillaries causing pulmonary oedema. Death occurs within hours.

Arsenic

Arsenic poisoning can occur by exposure to a number of different pesticides, rodenticides, defoliants and drugs. The toxicity of arsenic varies with the formulation.

Arsenic is a heavy metal that produces digestive tract irritation within hours of ingestion. Signs include colic with excessive peristaltic activity, diarrhoea (which becomes bloody), dehydration, cardiovascular collapse and death.

Dimercaprol (BAL) is the specific antidote. Sodium thiosulphate may be used as an alternative.

22.23 HEAVY METALS

Lead

See Chapter 11.

Selenium

Poisoning occurs in areas with a high selenium concentration in soil and may be acute, subacute or chronic. Acute toxicity causes depression, diarrhoea, laboured breathing, recumbency and death. Death may occur within several hours. Subacute toxicity results in liver and CNS dysfunction with signs of depression, blindness, weakness, paralysis and death. Chronic toxicity results in weight loss, hair-loss from the mane and tail, and cracking at the coronary bands.

There is no specific therapy. Approximately 50% of chronic cases recover if they are removed from the source of selenium.

Zinc

Toxicity usually occurs from environmental contamination of pastures around lead and zinc mines, zinc smelters, etc. Toxicity is usually seen in foals and yearlings. Signs include enlargement of the epiphyses of long bones, lameness or stiff gait, weight loss and ill-thrift. There may be an association with the development of osteochondritis dissecans.

Arsenic

See Section 22.22.

Other heavy metals

- Cadmium – nephrotoxic.
- Copper – hepatotoxic and haemolytic.
- Mercury – gastrointestinal tract irritation, CNS and kidney damage.

22.24 ANTHELMINTICS

Carbon tetrachloride

Carbon tetrachloride has a peculiar odour and is rarely ingested by horses. Ingestion of large quantities causes liver damage (centrilobular necrosis and fatty infiltration) and kidney damage. Clinical signs include depression, weakness, ataxia, diarrhoea, collapse and death.

Phenothiazine

Signs of toxicity are seen within hours of drug administration and include anorexia, depression, staggering hindleg gait, colic, jaundice and haemoglobinuria. Photosensitization may also occur.

22.25 PETROLEUM PRODUCTS

Ingestion of petroleum products (kerosene, fuel oils, crude oil and partially refined petroleum products) causes blistering of the mouth, salivation, colic and diarrhoea. Skin exposure causes hair-loss and local irritation. Inhalation of droplets of petroleum products results in coughing, sneezing and dyspnoea.

22.26 FEED-ASSOCIATED POISONINGS

Ionophores

See Section 22.4.

Antibiotics

Severe, and often fatal, colitis has occurred in horses after feeding lincomycin, tylosin, tetracycline or neomycin. The toxicity probably involves overgrowth by pathogenic bacteria, but the precise identity of these bacteria is uncertain. Clinical signs are usually observed 24 to 48 hours after exposure to contaminated feedstuffs.

Blister beetle (cantharidin) poisoning

See Section 22.4.

Aflatoxin

Aflatoxins are a group of mycotoxins produced in grains by *Aspergillus* spp. They cause acute hepatotoxicity, and the clinical disease is characterized by anorexia, fever, lethargy, tachycardia, tachypnoea and diarrhoea. Jaundice may also be seen. Coagulation defects and bleeding tendencies may occur if the horse survives the acute stage of toxicity.

FURTHER READING

Galey FD (1992) Current therapy in equine medicine 3, section 8. In: Robinson RE (ed) Toxicology. W.B. Saunders, Philadelphia

Gupta R (2007) Veterinary toxicology. Basic to clinical principles. Academic Press, New York

Knight AP (2005) A guide to poisonous house and garden plants. Teton New Media, Jackson, WY

Oehme FW (1987) Current therapy in equine medicine 2, section 16. In: Robinson RE (ed) Toxicology. W.B. Saunders, Philadelphia

Orsin J, Divers T (2007) Equine emergencies. Treatment and procedures, 3rd edn, part 4. Toxicology. Elsevier, St Louis, MO

Reed S, Bayly W, Sellon D (2010) Equine internal medicine, 3rd edn, ch 22. Toxicologic problems. Elsevier, St Louis, MO

Roder J (2001) Veterinary toxicology. Butterworth-Heinemann, Woburn, MA

Chapter 23

Principles of wound management

Patrick J. Pollock, Jim Schumacher

CONTENTS

23.1 Definition of a wound 469
23.2 Classification of wounds 469
Open wounds 469
Closed wounds 470
23.3 Assessment of the wounded horse 470
General assessment 470
Penetrating wounds over the abdomen or thorax 470
23.4 Detailed assessment of the wound 470
Initial assessment 470
Suspected synovial involvement 470
Blood supply 470
Factors that impair healing 470
23.5 Stages of wound healing 473
Inflammatory phase 473
Debridement phase 474
Repair phase 474
Maturation phase 474
23.6 Methods of wound management 474
23.7 Primary (first intention) healing 474
Definition 474
Criteria for primary closure 474
Preparation of a wound for primary closure 475
Surgical debridement 475
Debridement by lavage 475
Local anaesthesia 476
Options for wound closure 476
Drains 476
Skin tension 477
23.8 Delayed primary closure (third intention healing) 477
23.9 Delayed secondary closure 479
23.10 Second intention healing 479
Wound expansion 479
Contraction 479
Epithelialization 480
Granulation tissue 480
23.11 Skin grafting 481
Indications for grafting 481
Graft classification 481
Physiology of graft acceptance 482
Causes of graft failure 482
Preparing the wound for grafting 482
Preparing the donor site 482
Sheet grafts 482
Island grafting 485
Allografts and xenografts 487
Storage of grafts 487
Further reading 487

23.1 DEFINITION OF A WOUND

A wound is a disruption of the anatomical and cellular continuity of tissue. Wounds can be created accidentally, or they can be created intentionally, such as by surgical incision.

23.2 CLASSIFICATION OF WOUNDS

Open wounds

1. Incision. A wound produced by sharp objects, such as, intentionally with a scalpel or accidentally by glass, sheet metal, etc. The skin edges are cleanly cut with little tissue damage, and such injuries cause little pain.

© 2013 Elsevier Ltd
DOI: 10.1016/B978-0-7020-2801-4.00023-7

2. Laceration. This is the most common type of wound and is characterized by irregular skin edges and extensive damage to underlying tissue. Such injuries cause considerable pain from bruising of tissues. When tissue has been lost, the laceration is termed an avulsion, and a laceration that extends into tendons, ligaments, synovial structures, or body cavities is referred to as a complicated wound.
3. Puncture. A wound produced by a sharp object that perforates tissue. These injuries may be complicated by the presence of dirt, manure, and other debris carried into the depths of the wound. There is considerable risk that a cavity, such as a joint, tendon sheath, bursa, abdomen, or thorax, may be perforated. Such wounds are easily trivialized; however, size belies severity.

Closed wounds

The entire thickness of skin has not been separated.

1. Abrasion (also referred to as a "graze"). Abrasions are friction injuries to the superficial surface of skin or mucous membrane and are characterized by oozing of serum and minimal haemorrhage; due to exposure of nerve endings these injuries result in considerable pain. Example: the donor site of a split-thickness skin graft.
2. Contusion. The result of bleeding and tissue destruction within and under intact skin without actual division of skin.
   Types:
   (a) 1st degree – slight haemorrhage into and under the skin with little haematoma formation.
   (b) 2nd degree – produces a subcutaneous haematoma.
   (c) 3rd degree – haematoma is so extensive that the skin above it eventually sloughs.
3. Burn. Exposure of skin to excessive heat (or cold or corrosive substances). Examples: Thermal burn, friction burn, chemical burn, freeze burn, and sunburn. Burns result in the coagulation of tissue proteins.
   Types:
   (a) Superficial (1st degree) – damages epidermis.
   (b) Partial thickness (2nd degree) – damages entire epidermis and varying depths of dermis.
   (c) Full-thickness (3rd degree) – causes complete destruction of full-thickness of skin and variable damage to subcutaneous tissue.

23.3. ASSESSMENT OF THE WOUNDED HORSE

General assessment

- The general state of the horse should be assessed.
- A history should be obtained and a thorough physical examination performed.
- To assess the horse and evaluate the wound, the horse must be cooperative. Twitch, sedation, and regional, local or even general anaesthesia may be necessary.
- Tetanus vaccination status should be determined. Appropriate protection should be instituted.
  1. Administer tetanus toxoid if the horse is on a vaccination programme for tetanus and has not been vaccinated in the previous 6 months.
  2. Idiopathic acute hepatic disease in horses (i.e. Theiler's disease) (see Chapter 3) may be associated with administration of tetanus antitoxin. A lactating mare may be most at risk of developing Theiler's disease after administration of antitoxin.

Penetrating wounds over the abdomen or thorax

The horse should have serial haematocrits performed to determine if it is haemorrhaging internally. Serial sampling and examination of fluid from the body cavity may be indicated to determine if the gastrointestinal tract has been penetrated or if there is internal haemorrhage.

23.4 DETAILED ASSESSMENT OF THE WOUND

Initial assessment

Initial assessment and care of the wound have the single most decisive influence on healing. Knowledge of anatomy in the region of the wound is crucial in assessing the severity of injury. Consideration must be given to the location of the wound (Figure 23.1).

1. Injuries to the extremities frequently involve important underlying structures, such as nerves, major blood vessels, ligaments, bones, or joints.
2. To avoid contaminating an open body cavity or synovial structure, digital palpation of the wound should be delayed until the wound is cleansed and should never be performed without sterile gloves.
3. Nerves lie in close proximity to arteries and veins, and therefore, trying to clamp a bleeding vessel in a standing horse can be dangerous and is best avoided.
4. The wound should be evaluated for the degree of contamination and for the presence of foreign bodies.

Suspected synovial involvement

When invasion of a synovial structure is suspected:

- A sterile, balanced electrolyte solution or a radiocontrast medium can be injected into the joint at a site remote from the injury (so-called synovial pressure test) (Figure 23.2).
- Solution exiting the wound indicates that the joint has been penetrated. If possible, synovial fluid should be examined cytologically.

Blood supply

Blood supply should be assessed.

Factors that impair healing

Examine the horse for factors that prevent or impair healing. These include the following:

1. Infection.
2. Contamination/presence of foreign body/sequestrum (Figures 23.3, 23.4).
3. Necrotic tissue.
4. Movement (Figure 23.5).

Figure 23.1 Lacerating wounds to the thorax (left) – consideration must be given to involvement of ribs and the thoracic cavity – and the palmar/plantar aspect of the pastern (right) – consideration must be given to involvement of the digital flexor tendon sheath and tendons/ligaments of the palmar/plantar pastern region.

Figure 23.2 Synovial pressure test. Sterile saline solution being injected intra-articularly into the elbow joint is seen to exit from the wound (arrow).

Figure 23.3 Contaminated and infected leg wound.

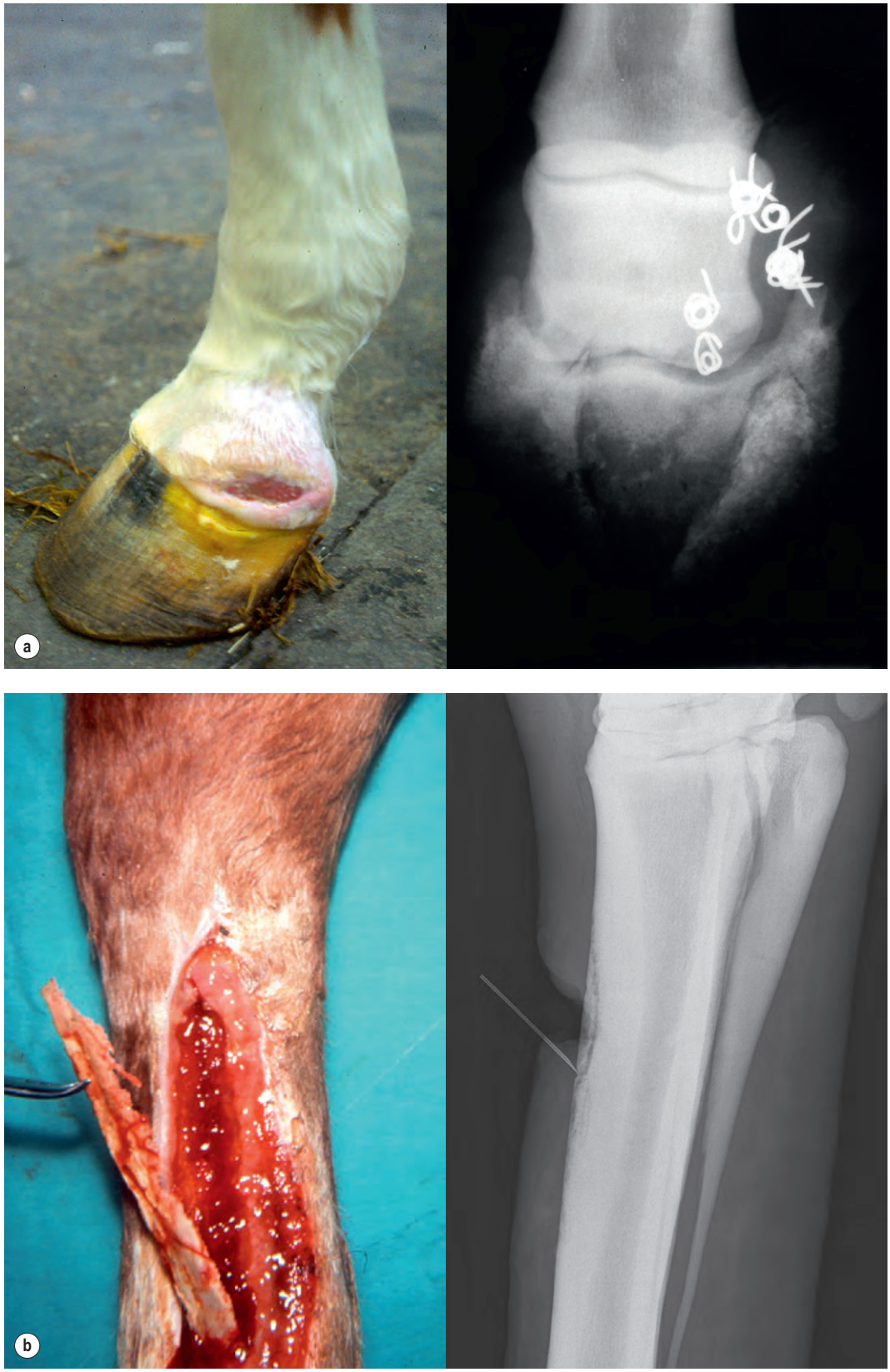


Figure 23.4 (a) The presence of foreign bodies (wire barbs) in a pastern wound. (b) Sequestrum of metatarsal bone 3 preventing healing of the overlying skin wound.

Figure 23.5 Non-healing wound over the dorsal tarsus – associated with excessive movement of the site.

5. Loss of blood supply.
6. Poor tissue oxygenation.
7. Loss of tissue (deficit).
8. General/systemic health disorder of the horse.
9. Tumour transformation (sarcoid) (Figure 23.6) (see Chapter 13).
   (a) Benign, locally aggressive fibroblastic tumour.
   (b) Wounds on horses with sarcoids at other sites are predisposed to developing sarcoid.
10. Cutaneous habronemiasis (see Chapter 13). Also known as 'summer sore' or 'granular dermatitis', a granulomatous, mildly pruritic disease caused by cutaneous migration and encystment of the larvae of the equine stomach worms (*Habronema musca*, *Habronema majus* and *Draschia megastoma*). Larvae passed in the faeces are ingested by fly maggots, and after the maggots pupate, the larvae are deposited on wounds from the feeding flies. The condition appears in spring and summer, when flies are prevalent, and usually disappears with onset of cold weather. Cutaneous habronemiasis is characterized by exuberant granulation tissue that contains numerous, small, yellow, hard, caseous granules composed of eosinophils, nuclear remnants, and larvae. Affected horses may have a circulating eosinophilia. Lesions are resolved by eliminating the larvae with ivermectin or an organophosphate or by reducing the horse's hypersensitivity to them with corticosteroids.
11. Pythiosis (see Chapter 13). This disease is caused by invasion of a protistal organism, *Pythium insidiosum* (formerly *Hyphomyces destruens*). The condition is characterized by a rapidly enlarging granuloma that contains sinuses filled with purulent, serosanguineous fluid and yellow granules, commonly referred to as 'kunkers' or 'leeches'. Affected horses can be successfully treated with a vaccine made from *P. insidiosum*. Ancillary treatment includes systemic administration of sodium iodide and then organic iodide (ethylenediamine dihydroiodide).
12. Ehlers–Danlos syndrome (hyperelastosis cutis). A heritable dysplasia of connective tissue. Affected skin is hyperextensible, fragile, and prone to wounding with slight trauma.
13. Burn carcinoma.
    Malignancy that develops long after the original injury is described as chronic, or latent.
    Malignancy that develops soon after the original injury is described as acute.
    Latent malignancy is more common than acute malignancy.
    The most common type of malignancy induced by injury is the carcinoma, and the most common type of injury producing the malignancy is a burn. In general, the older the patient at the time of injury, the shorter the interval from injury to the onset of neoplasia. Cause may be persistent irritation from chronic infection and trauma to the poorly vascularized wound. Healing after each insult becomes more difficult, and regenerated epithelium becomes increasingly inferior. The prognosis for survival of horses with burn-induced neoplasia is poor because of a marked propensity for the neoplasm to metastasize.

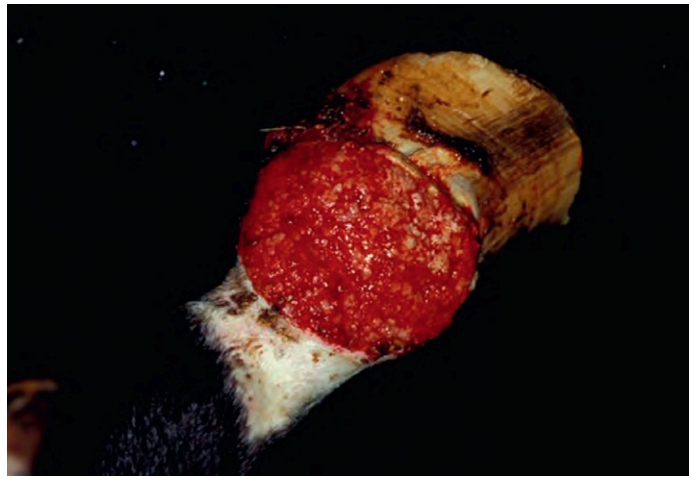


Figure 23.6 Growth of sarcoid tissue at a wound site.

23.5 STAGES OF WOUND HEALING

- Wounds elicit a consistent and orderly response by the body that restores continuity to injured tissue. Wounds move predictably down a common pathway of healing, and knowledge of the predictable events of healing can be used to decide steps in wound management. These phases are found in both sutured and unsutured wounds.
- The sequence of events progresses through four overlapping phases.

Inflammatory phase

Occurs during the first 2 to 3 days after wounding. This phase is essential to protect against infection and to initiate the repair. Initially,

a transient vascoconstriction occurs, followed by an increase in vascular permeability. The wound fills with fibrinogen which converts to fibrin. Blood and fibrin form a fibrocellular clot. The clot limits blood loss and provides a scaffold for the migration of cells. Leukocytes move into the wound. First to appear are neutrophils. Higher numbers of neutrophils move into the wounds of ponies than into the wounds of horses. The neutrophils die and release enzymes that digest cellular debris and contribute to inflammation. Neutrophils disappear more rapidly in wounds of ponies than in wounds of horses. Numbers remain persistently high in wounds of horses compared to wounds of ponies.

The inflammatory phase of wound healing occurs more quickly and to a greater degree and for a shorter time in ponies than in horses. The course of inflammatory response in ponies is apparently more efficient for wound healing, and this is why the wounds of ponies heal faster than do wounds of horses.

The inflammatory phase of wound healing occurs more quickly and to a greater degree and for a shorter time in trunk wounds than in limb wounds. This is one of the reasons why wounds of the trunk heal faster than do wounds of the limbs.

Debridement phase

Occurs within 6 to 8 hours of injury, and the duration depends on the amount of debris and contamination. Monocytes are the predominant leukocyte. They become macrophages when they move into the wound and are responsible for removal of debris. Furthermore, they have an inductive effect on fibroblasts and provide cytokines and growth factors necessary for the stimulation of fibroplasia and angiogenesis.

The inflammatory and debridement phases of wound healing have also been described as the lag phase. The lag phase involves preparation of the wound for repair.

Repair phase (sometimes called the fibroblastic or proliferative phase)

The repair phase proceeds after the wound is debrided. During this phase wound strength increases rapidly (until this phase, the wound has no strength).

Granulation tissue is produced during the repair phase. This is a complex of fibroblasts, vascular endothelial cells, and macrophages within a matrix of collagen and fibrin.

(a) Fibroblasts and capillaries appear in the wound by day 3.
(b) Fibroblasts use the fibrin clot as a matrix and replace it with new matrix.
(c) Fibroblasts lay down collagen and other components of connective tissue.
(d) Granulation tissue is necessary for wound contraction and migration of epithelium.
(e) Fibroblasts may proliferate in wounds of horses even after the wound has been filled with granulation tissue, but fibroblasts cease to proliferate in wounds of ponies when the wound is filled with granulation tissue.
(f) Epithelial cells move across the granulation tissue.

Maturation phase (sometimes called the remodeling phase)

The maturation phase begins about 2 weeks after wounding and continues for 6 to 12 months. The wound contracts, granulation tissue diminishes, and collagen production declines. Intra- and intermolecular cross-linking of collagen fibres occurs. It should be noted that the scar is never as strong as the tissue it replaced. It remains 15 to 20% weaker than the surrounding tissue.

23.6 METHODS OF WOUND MANAGEMENT

Methods of wound management include primary closure, second intention healing, delayed primary closure (or third intention healing), delayed secondary closure, and grafting.

23.7 PRIMARY (FIRST INTENTION) HEALING

Definition

Healing occurs primarily (or by first intention) when union or restoration of continuity of tissue occurs directly with minimal production of granulation tissue and minimal epithelial migration. Wounds are sutured with the intent that they will heal primarily.

Primarily closed wounds of ponies are less likely to dehisce or develop bone sequestrae than are primarily closed wounds of horses.

Criteria for primary closure

1. The wound must be vascular. Suturing a wound caused by a sharp object is more likely to be successful than is suturing of a wound caused by blunt trauma.
2. Sutured wounds must have minimal tension.
3. Wound must be clean and non-infected. Infection is the main cause of dehiscence.

Wounds should be closed during the "golden period", which is the time that elapses between wounding and infection. This period is generally considered to be approximately 6 to 8 hours although this is influenced by the site of the wound and the level of contamination.

The wound becomes infected when the concentration of bacteria in the tissue exceeds the ability of humoral and cellular defences to destroy the bacteria, and for most types of bacteria, this concentration is about 10^5 organisms per gram of tissue.

All open wounds are contaminated, because no amount of protection prevents airborne or skin bacteria from entering the wound.

Factors that affect the concentration required to cause infection:

- Foreign bodies, such as sutures and drains or bone sequestrae, in contaminated wounds dramatically lower the concentration of bacteria necessary to cause infection.
- Clean, insoluble material, such as glass, gravel, or metal cause less tissue reaction than do porous, contaminated, and organic material, such as grass, straw, and wood.
- Soil increases the risk of infection. Infection-potentiating factors (IPF) are found in soil. The IPF have a high negative charge that inactivates leukocytes, which are positively charged. Clay and organic soil contain the most IPF, whereas sand has few.
- Necrotic tissue and dead space dramatically lower the concentration of bacteria necessary to cause infection.
- The concentration of some bacteria, most notably β-haemolytic streptococci and pseudomonads, required to infect a wound is much less than 10^5 per gram of tissue.
  - β-haemolytic streptococci produce proteolytic enzymes, including streptokinase and staphylokinase, which are destructive to the wound.
  - *Pseudomonas* spp. produce elastase, which specifically degrades elastin in the wound to which fibrin attaches.

- *Streptococcus* spp. are nearly always susceptible to a topically applied penicillin or cephalosporin, and pseudomonads are susceptible to aminoglycides, polymixin B, and sulfamylon.

When deciding whether to close a wound, the time that has elapsed between wounding and treatment should be given only modest consideration. For example, wounds of the head have a long grace period because the blood supply to the head is excellent, causing bacteria in a wound of the head to multiply slowly to the concentration of 10^5 organisms per gram of tissue. Soft-tissue wounds of the head can often be closed safely often 24 hours or longer after wounding (Figure 23.7). Bacterial quantitative analysis can be used to determine the concentration of bacteria in a wound. If a wound contains more than 10^5 bacteria per gram of tissue, the wound should not be sutured. Quantitative analysis is usually impractical because:

- It is time-consuming.
- The type of bacteria is more important.

Qualitative analysis is usually used to determine readiness for closure.

Any wound that shows signs of inflammation, such as redness, swelling of the surrounding area, and formation of exudate, should be assumed to be infected. A wound showing signs of inflammation should not be closed, and a wound managed by primary closure that develops signs of inflammation should be opened or partially opened. Unfortunately, even a wound that has no signs of inflammation and appears to be healthy might have more than 10^5 bacteria per gram of tissue because there is a lag phase between microscopic and clinical signs of infection.

Figure 23.7 Many wounds of the head can be treated by primary closure often 24 hours or longer after injury because of the rich blood supply to the area.

Preparation of a wound for primary closure

The objective in care of all open wounds is to convert the open contaminated wound into a surgically clean wound that can be closed.

Because hair in a closed wound acts as a foreign body, care should be exercised to avoid contaminating the wound when removing hair. Hair surrounding the wound should be dampened with water or coated with K-Y water soluble jelly, and the wound should be packed with sterile saline-dampened sterile gauze. Sterile gauze dampened with a local anaesthetic agent can also be used.

The skin surrounding the wound should be cleansed with antiseptic soap, but soap applied to the wound may interfere with healing.

- Soap interferes with defense mechanisms of the wound and when applied to contaminated tissue increases the wound's susceptibility to infection. The detergent may drive bacteria deeper into the tissue.
- Soap applied to uncontaminated fresh wounds irritates tissue but does not significantly increase the wound's susceptibility to infection.

The wound should be debrided, i.e. removal of foreign material and devitalized tissue. Devitalized tissue prolongs healing and provides an environment conducive to bacterial proliferation

Surgical debridement

1. The guiding principle in surgical debridement is that, when excised, living tissue bleeds, and devitalized tissue does not.
2. The simplest method of debridement is to create a surgically clean wound by totally excising the wound (i.e. en bloc debridement). This can rarely be done in horses because horses do not have abundant soft tissue.
3. Usual method is to excise all grossly nonviable or damaged tissue with a scalpel. Superficial fascia is often a edematous making it easy to excise with a scalpel or Metzenbaum scissors. Surgically debriding wounds overlying critical structures, such as nerves, tendon sheaths, and joint capsules, is difficult.
4. If the wound contains much devitalized tissue or debris, the wound should be left open for delayed primary closure or to heal by second intention.
5. Rather than excising a flap of skin that appears to have an inadequate blood supply to survive, subcutaneous tissue can be sharply excised from the dermis, and the flap sutured in place to act as a full-thickness "skin graft".
6. Curettage of exposed cortical bone (i.e. bone without periosteum) may be helpful in preventing formation of an osseous sequestrum.

Debridement by lavage

The aim is to reduce the concentration of contaminating bacteria on wounds to non-infective concentrations.

1. Low-pressure lavage – pressure below 8 psi (lb/sq in).
2. High-pressure lavage – pressure ≥8 psi (lb/sq in).
   - Mechanically disrupts bacterial adherence in addition to removing foreign material.
   - Can be achieved with a 19-gauge needle and a 35-mL syringe or with a spray bottle.
   - Common concern during lavage is that high-pressure lavage may disseminate foreign material and bacteria from the

surface of the wound into the depths of the tissue, but this concern is not justified.

Solutions for lavage

- Tap or distilled water.
- Hypotonicity causes swelling and destruction of some cells; however, the benefits outweigh problems caused by lack of isotonicity.
- Physiological saline solution (PSS), which can be made by adding a flat teaspoon of table salt to 1 pint (0.6 L) of boiled tap water.
- Lactated Ringer's solution (LRS).
- Hydrogen peroxide. Gives impressive appearance as it foams on the wound, but it has little value as an antiseptic. The solution is more effective as a sporocide than as a bactericide and has a narrow antimicrobial spectrum of action. A 3% concentration of hydrogen peroxide is toxic to fibroblasts and may cause thrombosis in the microvasculature adjacent to the wound's margin. Hydrogen peroxide should be reserved for initial treatment of dirty wounds in which clostridial spores may be present.

Antiseptics added to lavage solutions

An antiseptic is added to the lavage to reduce bacterial concentration. Antiseptics commonly added:

Povidone iodine (PI)

Antimicrobial activity is a property of 'free iodine' in the solution. Complexing iodine with polyvinylpyrrolidone eliminates the adverse properties of iodine, such as irritation and staining, but decreases iodine's bactericidal properties because, if the concentration of PI is high, the iodine is more tightly bound to the polyvinylpyrrolidone, resulting in less free iodine and thus, lower bactericidal activity. The usual concentration is 0.1% to 0.2% (10 to 20 mL of 10% stock solution/1 L). Some advise a 0.001% solution (0.1 mL of 10% stock /1L diluent).

Advantages:

- Wide spectrum of antimicrobial activity.
- Nearly instantaneous action.
- No known bacterial resistance.

Disadvantages:

- Toxicosis could occur with repeated use.
- Inactivated by organic material.
- Poor residual activity.

Chlorhexidine

Advised concentration is 0.05%. Use 1 mL of 2% stock solution (i.e. Nolvosan) per 40 mL water (or 25 mL per 975 mL solution).

Advantages:

- Wide spectrum of antimicrobial activity.
- Is not absorbed systemically and hence does not cause toxicosis.
- Longer residual antibacterial activity than PI. It binds to protein in the stratum corneum.

Disadvantages:

- Is unstable in PSS or LRS, but only after 12 to 24 hours. Therefore solutions made with PSS or LRS should not be stored.
- Cannot be used in synovial cavities.
- Is toxic to the cornea when in direct contact with it.
- *Proteus* and *Pseudomonas* spp. are resistant to it.

Local anaesthesia

During debridement, to prevent pain to the horse, the wound should be anaesthetized with local or regional anaesthesia, before it is cleansed, debrided, and sutured. Horses with extensive wounds may require general anaesthesia.

Local anaesthetic solution adversely affects healing by reducing adherence of leukocytes to endothelium, which reduces their migration into the wound. However, the effect is minimal.

The addition of epinephrine (adrenaline) to the local anaesthetic solution is usually unnecessary but may be helpful because it slows absorption of the local anaesthetic solution and thereby extends the time of anaesthesia. The addition of epinephrine to the solution may be helpful in highly vascular wounds.

Options for wound closure

- Tissue adhesive (e.g. n-isobutyl cyanomethacrylate). Can be used only when there is no tension on the wound margin. Maintains adhesion for only 4 to 6 days, and closure cannot be adjusted after adhesive is applied.
- Sutures. Monofilament nylon and polypropylene are most appropriate. For deeper layers, synthetic, monofilament absorbable sutures are most appropriate. Interrupted patterns provide more security because failure of one suture does not jeopardize the entire closure. When compared to simple continuous patterns, surgery time is longer and holding power is less. Slightly everting suture patterns are ideal because skin edges tend to invert during healing. Excessive tension on simple interrupted sutures may cause inversion of skin edges.

1. Interrupted horizontal mattress.
   - Everting or appositional, depending on tension applied.
   - Tendency to strangulate tissue.
2. Interrupted vertical mattress.
   - Everting or appositional depending on tension applied.
   - Withstands tension better than a horizontal mattress.
   - Less likely than the horizontal mattress to strangulate tissue.
3. Staples.
   - Produce a slightly everted skin closure.
   - Reduce tissue handling.
   - Decrease surgery time.

Epithelium spans the gap on a sutured or stapled wound at about 48 hours. The wound is susceptible to infection until then.

Drains

Drains are placed to rid the wound of serum, exudate and haemorrhage, which can disrupt fascial planes and harbour bacteria. They should be considered for contaminated or infected wounds and in areas where an excessive volume of fluid may accumulate.

Types of drain:

1. Passive drain tubes. Example: latex drains, such as Penrose drains. These provide no intraluminal drainage. All fluid is drained by forces of surface tension, and therefore, creating holes in the tubing reduces effectiveness.
2. Active drain tubes. Example: vacuum or suction drains. These drains rely on a fenestrated tube and a persistent mild vacuum created with a large syringe or a commercial concertina pack system; they minimize the risk of ascending infection.

With all drains, the dependent portion of drain should exit skin at a site remote from the suture line to minimize the risk of dehiscence. Skin incision at the exit point should be large and oriented perpendicular to the drain so that the drain can lie flat to achieve maximum drainage (Figure 23.8).

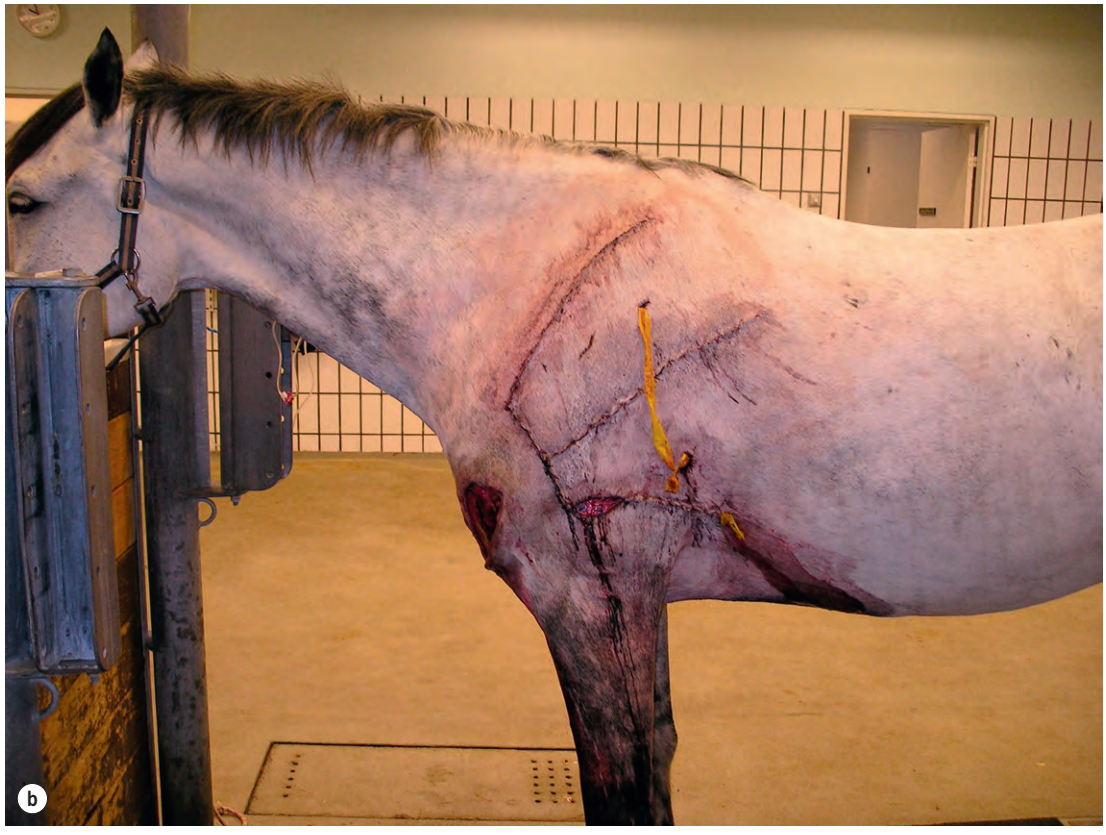


Figure 23.8 (a) Large skin flap at wound to the chest wall. (b) After primary closure with Penrose drains exiting via large skin incisions remote from the wound edges.

Disadvantages of drains

1. They act as a foreign body to decrease resistance to infection. Consider systemic administration of an antibiotic while the drain is in place.
2. They act as a wick for infection. Wound and drain should be covered with a bandage, if possible.

In all cases, the potential benefits and harmful effects of a drain should be weighed before a drain is placed.

Skin tension

Wounds closed under tension occasionally develop necrosis of the wound edge. Techniques used to avoid excessive tension on the suture line include the following:

1. Undermining the skin. This is the simplest method, but care should be taken because if the skin is too widely undermined and advanced too far, the blood supply to the skin edge may be compromised.
2. Various tension suture patterns:
   - Presuturing. Sutures are placed before a wound is debrided or a lesion is excised to ease tension on the margin of the wound before the wound is sutured. Presuturing decreases the force required to close the wound by 40%. Performed using local or regional anaesthesia between 2 and 8 hours prior to surgery. Resembles a Lembert suture, with a bite on each side of the lesion. The technique relies on stress relaxation, which is the phenomenon whereby when skin is stretched to a certain length, the force required to keep it at that length decreases over time. (Collagen fibres in the relaxed state are arranged in a randomly oriented, convoluted pattern. Load applied to skin straightens the collagen fibres so that they become parallel.)
   - Walking sutures permit coverage of the wound, they obliterate dead space, and they evenly distribute tension rather than concentrating tension at the wound's edge. Technique. Skin at the margin of the wound is undermined, and at the junction of the undermined skin and underlying subcutaneous tissue, a suture bite is placed into the dermis. With the margin of the wound stretched, a second suture bite is placed through the subcutaneous tissue underlying the suture bite in the dermis. Tightening the suture moves the skin toward the centre of the wound. Rows of these sutures are placed in the stretched, undermined skin on each side of the wound.
   - Horizontal and vertical mattress tension sutures:
     1. Placed well away from the skin edges.
     2. Skin edges are approximated with simple-interrupted sutures.
     3. Can interfere with blood supply. Vertical mattress sutures are less likely than horizontal mattress sutures to interfere with the blood supply. Rubber stents (effectively made from IV line tubing) may prevent interruption of blood supply by re-distributing pressure.
     4. Removed in 5 to 7 days. The skin edges remain apposed with the simple-interrupted sutures.
   - Far-near or pulley sutures. These serve as both a tension and an approximating suture. Far component relieves tension, and near component approximates. Excessive tightening leads to inversion of skin edges.
   - Stent bandage. These are useful where conventional bandages cannot be applied and provide support to the wound's margin. Stents maintain an even pressure, thus reducing dead space and accumulation of fluid. They may prevent self-mutilation.
   - Relaxing incisions. Indicated when the wound edges are close to being apposed, but apposition with tension sutures could produce enough tension to impair circulation. A single incision made parallel to the long axis of the wound allows the skin between this incision and the wound to be undermined and advanced over the wound. Creating a relaxing incision to close a wound may be justified if the relaxing incision allows a wound exposing ligament or tendon to be closed. The wound created by the relaxing incision heals by second intention. The width of the skin between the relaxing incision and the wound should be equal to the width of the wound. Creating a relaxing incision on each side of the wound, results in two smaller wounds. Multiple, staggered, parallel punctuate relaxing incisions heal faster than one or two large relaxing incision but don't provide as much relaxation.
   - Skin expanders. Silicone balloons are implanted subcutaneously, and the wound is allowed to heal. The implanted balloon is then filled with saline solution injected through a remote port slowly, over a period of days to weeks. After the expander has filled, it is removed, and the expanded skin is advanced or rotated to cover both the donor site and the defect. During expansion, the epidermis remains the same thickness, but the dermis is thinned. Skin flaps generated from expanded skin have increased survivability.

23.8 DELAYED PRIMARY CLOSURE (THIRD INTENTION HEALING)

Apposition of a wound after the 'golden period' (i.e. after it is considered infected rather than contaminated) but before the appearance of granulation tissue (usually within 4 to 5 days after injury). The technique can be applied to a wound that might perhaps heal if it were sutured immediately but appears to have a higher than normal vulnerability of developing an abscess, haematoma, or seroma.

Technique:

- Loose debris and foreign material are removed, and the wound is irrigated with copious amounts of physiological saline solution.
- The wound is packed with absorbent gauze, and sterile dressings are applied.
- The wound is sutured when the condition of the wound shows it to be ready for primary closure.

Quantitative bacterial analysis can be performed to determine when the wound is ready to suture, but this is not usually practical. Prior to closure, the wound should appear clean and healthy, secretions should be clear and non-odiferous and swelling should be minimal.

Remember that delaying suture closure does not interfere with healing because healing begins as an inflammatory response the instant trauma occurs and progresses whether the wound is sutured or not.

Problems associated with delayed closure include retraction, fibrosis, and decreased pliability of the wound margin. To avoid these problems, the technique of **augmented wound contraction** can be used.

- Local or regional anaesthesia is used to desensitize the wound and surrounding tissue.
- A continuous horizontal intradermal mattress suture pattern is placed intradermally using heavy suture material. Each bite is advanced slightly, so that the suture passes at an angle across the wound, allowing it to slide easily through the skin.

- Each end of the suture is anchored to a human umbilical clamp or split-shot.
- Tension is applied to the suture as an assistant pushes the wound edges together.
- The suture can be tightened daily without placing new sutures.
- Skin edges are eventually apposed, if possible, or the wound is left as it is to heal by second intention.

The most gain occurs within the first 3 days. This technique not only augments contraction but avoids expansion of acute wounds.

23.9 DELAYED SECONDARY CLOSURE

This is an uncommonly performed technique. Closure is performed after granulation tissue has started to form (i.e. after 4 to 5 days). When granulation tissue forms, the skin edges of the wound become adherent to the underlying tissue, and approximation is possible only by dissecting the edges free. Owing to the immobility of tissues, it is often easier to excise the granulation tissue and a thin rim of skin at the wound margin and perform a primary closure.

23.10 SECOND INTENTION HEALING

Wounds left unsutured heal by second intention. Second intention healing is used for wounds that have lost tissue or for those wounds that are infected or are susceptible to infection.

The two mechanisms by which wounds left unsutured heal are contraction and epithelialization. Both mechanisms depend on the formation of granulation tissue. Skin does not regenerate. Skin is not a simple tissue such as epithelium, fascia, or fat. It is a highly complex organ containing multiple structures derived from multiple germ layers. Other organs do not regenerate, and neither does skin. Second intention healing of wounds of ponies occurs faster than does second intention healing of wounds of horses. Second intention healing of wounds proximal to the carpus or tarsus occurs faster than does second intention heal of wounds distal to the carpus or tarsus.

Wound expansion

- Immediately after creation of a cutaneous defect the wound enlarges from retraction of the surrounding skin.
- Wounds on the distal limbs of horses may expand for 11 to 13 days before contraction begins.

Contraction

- Contraction is the process by which tissue surrounding a defect is pulled centripetally to close the defect.
  - **i.** Contraction begins after the lag phase.
  - **ii.** Wounds distal to the carpus and hock are not capable of significant contraction.
  - **iii.** Contraction is responsible for 95% or more of healing of wounds of the trunk and neck.
  - **iv.** The speed of wound contraction largely determines the speed of second intention wound healing.
- Contraction of a wound over the joint of a human being, or in some small animal species, sometimes leads to contracture. Contracture is the deformity of a joint caused by contraction of an overlying wound. This complication has not been reported in the horse.
- Mechanisms of contraction. Collagen fibres were once thought to be responsible for contraction of wounds. Collagen fibres, however, do not shorten. Wounds of scorbutic animals (i.e. those that lack vitamin C) have decreased collagen, yet they contract normally. Contraction of wounds is brought about by myofibroblasts, which are fibroblasts that have developed characteristics typical of smooth muscle. Smooth muscle actin is incorporated into the fibroblasts, and this is brought about by transforming growth factor beta (TGF-β). TGF-β is the most important instigator of wound contraction. Contraction starts sooner in wounds of ponies than in wounds of horses due to the fact that myofibroblasts in wounds of ponies are more organized than are myofibroblasts in wounds of horses, and granulation tissue of ponies contains more TGF-β than does granulation tissue of horses. The difference in wound contraction between ponies and horses is not caused by differences in the contractile capacity of the myofibroblasts but by mediators, such as TGF-β, in the granulation tissue. Contraction starts sooner in wounds of the trunk than in wounds of limbs.

Cessation of contraction – three mechanisms are responsible for cessation of contraction:

1. As the wound edges meet, contact inhibition of the cells stops further movement.
2. If the tension of the surrounding skin equals or exceeds the force of contraction, further contraction ceases.
3. Wound contraction stops when myofibroblasts disappear. Example: application of a skin graft causes the number of myofibroblasts to decrease.

Unfortunately there are currently no known factors that stimulate contraction, but factors that inhibit contraction should be controlled to allow contraction to proceed at a normal rate.

Factors that inhibit wound contraction:

1. Hypovolaemic anaemia. Causes hypoxia of tissues and decreases fibroblastic activity.
2. Hypoalbuminaemia (i.e. serum albumin below 3.0 g/dL). Wound itself can cause hypoalbuminaemia.
3. Corticosteroids administered systemically. These have to be administered in large doses and early in the inflammatory stage. (Horses affected with equine Cushing's disease heal poorly because of high circulating concentration of cortisone.) Corticosteroids delay development of granulation tissue by:
   - Inhibiting accumulation of inflammatory cells.
   - Depressing of formation of capillaries (i.e. angiogenesis).
   - Inhibiting proliferation of fibroblasts, thereby decreasing collagen production.
4. Non-steroidal anti-inflammatory drugs (NSAIDs). NSAIDs are prostaglandin inhibitors, and prostaglandins are necessary for the inflammatory stage of healing. These drugs may have adverse effects on migration of leukocytes, increasing the likelihood of infection, and can inhibit formation of granulation tissue and contraction but probably not in ordinary, therapeutic doses.
5. Full-thickness skin grafts. Application of grafts to wounds causes a reduction in the number of myofibroblasts, and therefore if applied to wounds before contraction commences, a full-thickness skin graft inhibits contraction. A full-thickness skin graft applied to a wound after contraction has commenced has little effect on inhibiting contraction. Split-thickness grafts are less effective than full-grafts in inhibiting contraction.
6. Shape of the wound. When contraction is measured kinetically, the shape of the wound has no influence on

contraction. Circular wounds contract similarly to wounds of other shapes.

7. Necrotic tissue. Formation of granulation tissue is retarded by the presence of necrotic tissue. Methods of debriding a wound of necrotic tissue are the same as for debridement for primary closure, but additional methods include:
   1. Enzymatic debridement. This technique should not be used on fresh wounds and is only appropriate when it is difficult to ascertain the extent of devitalized tissue. Common enzyme preparations contain pancreatic trypsin, or streptodornase/streptokinase of streptococcal origin, or a protease derived from *Bacillus subtilis*, or papain.
   2. Dakin's solution. A 0.5% solution of sodium hypochlorite. Prepared at one-tenth the strength of commercial laundry bleaches. It kills bacteria by releasing free chlorine and oxygen into the tissues and liquifies necrotic tissue. [One-half strength (0.25%) or one-fourth strength (0.125%) Dakin's solution causes less discomfort to the horse.]
8. Exuberant granulation tissue. Exuberant granulation tissue acts as a barrier to centripetal movement of the margin of the wound.
9. Infection. Infection is a serious detriment to contraction; bacteria secrete toxins and enzymes that alter the ability of haemoglobin to carry oxygen. Bacteria block the migration of fibroblasts and the formation of granulation tissue. The most detrimental bacteria are β-haemolytic streptococci and *Pseudomonas* spp.

Epithelialization

Epithelialization is the centripetal advancement of epithelium across a wound. In partial-thickness wounds (i.e. abrasions) epithelialization proceeds from the skin appendages, especially the hair follicles, and in full-thickness wounds, proceeds from the wound's edge. Migrating epithelium moves beneath the clot that covers the wound at a rate of about 1 to 1.5 mm/10 days in wounds on the limb. It can be first seen at about 2 weeks after wounding.

The epithelial cover is termed an **epithelial scar**. When epithelialization is the primary mode of wound healing, the scar is extensive.

Factors that increase the rate of epithelialization:

1. Heat. Bandaging increases heat at the wound.
2. Increased tissue oxygenation. Bandaging and casting increase tissue oxygenation by lowering the pH at the wound's surface. Live yeast-cell derivative, or skin respiratory factor, a water-soluble extract of yeast found in a haemorrhoid medication, may increase oxygenation.
3. Hyperbaric oxygen therapy (HBOT) increases the concentration of O_2 dissolved in plasma (which is normally about zero).
4. Moisture. Wet dressings promote more rapid epithelialization than does exposure to air, which desiccates the wound's surface, inhibiting epithelialization. An occlusive dressing may provide too much moisture, causing production of exudate, overgrowth of bacteria, and exuberant granulation tissue.
5. Some topically applied medications:
   - Silver sulfadiazine (1%). Produced a 28% increase rate of epithelialization in one study.
   - Neosporin. Produced a 25% increase in rate of epithelialization in one study (contains polymyxin B, bacitracin-zinc, and neomycin).
   - Zinc oxide ointment. Produced a 33% increase in rate of epithelialization in one study.
   - Live yeast cell derivative ointment (skin respiratory factor).
   - Protamine zinc insulin. More effective on chronic and infected wounds than on fresh, non-infected wounds.

Factors that decrease the rate of epithelialization:

1. Infection.
2. Necrotic tissue. Granulation tissue forms poorly if necrotic tissue is present, and granulation tissue is necessary for epithelialization.
3. Exuberant granulation tissue. Epithelium cannot migrate over protruding granulation tissue.
4. Some topically applied medications, including:
   - Glucocorticosteroids.
   - Nitrofurazone. Produced a 30% decrease in the rate of epithelialization in one study (polyethylene glycol, the base for nitrofurazone ointment, may be responsible for the decreased rate of healing).
   - Gentamicin cream (not gentamicin solution).
5. Changing a bandage too frequently may destroy new epithelium.

Granulation tissue

Granulation tissue begins to appear in the wound about 3 to 6 days after injury and provides a surface for migration of epithelial cells. It provides a barrier to infection (phagocytes in the tissue limit invasion of organisms), myofibroblasts necessary for wound contraction, and the fibroblasts responsible for collagen formation.

Factors that promote the formation of granulation tissue:

1. Body size. Horses (i.e. >140 cm), particularly draught horses, develop greater amounts of granulation tissue than do ponies. Ponies are less apt to develop exuberant granulation tissue. The wounds of ponies have a stronger initial inflammatory response and, therefore, develop granulation tissue more rapidly. Horses have a prolonged inflammatory response, which leads to production of exuberant granulation tissue.
2. Location of the wound. Wounds on the distal portion of the limbs develop more granulation tissue and wounds in mobile areas develop more granulation tissue. (Figure 23.9)
3. Environment of the wound. The following conditions at the surface of the wound, all provided by a bandage, encourage the development of granulation tissue:
   - Moisture.
   - Warmth.
   - Low oxygen tension.
   - Low pH (enhances oxygenation at the wound). Ideally, the wound should have a normal or very slightly acidic pH.
4. Topical medications that enhance formation of granulation include:
   - Scarlet oil. Usually used to stimulate granulation tissue in large wounds of the trunk.
   - Sugar or honey. Irritates granulation tissue and, therefore, stimulates its formation. Lowers pH, which increases oxygenation, Acts as a debriding agent and attracts macrophages.

Excessive production of granulation tissue (i.e. proud flesh) is detrimental to healing. It inhibits both contraction and epithelialization. The horse has a propensity to develop greater amounts of granulation tissue than do other species. Causes of persistent formation of exuberant granulation tissue include:

- Bone sequestrae and other foreign bodies.
- Irritation, such as from infection.

Exuberant granulation tissue can be prevented or controlled using the following methods:

1. Counter-pressure. Bandages and casts. Although bandages and casts provide counter-pressure, they may also promote exuberant granulation tissue. When excess proliferation is persistent, removing the bandage may resolve the formation of exuberant granulation tissue.

Figure 23.9 Excessive granulation tissue on a limb wound.

2. Immobilization. Excess movement, such as at the fetlock or hock, encourages deposition of collagen; however, total lack of movement can result in a weak scar because some stress is required to induce proper arrangement of collagen.
3. Antimicrobial drugs. An infected wound tends to produce excess granulation tissue because bacteria on the surface of wounds are strongly chemo-attractive to leukocytes, which increase the inflammatory response. Topical treatment for bacterial infection of granulating wounds is more effective than systemic treatment because systemically administered antimicrobial drugs are often incapable of achieving therapeutic concentrations within granulation tissue. Fibrin within the granulation tissue appears to prevent systemically administered antimicrobial drugs from reaching bacteria.
4. Various topical agents (any agent that retards granulation tissue, however, may also impede the epithelialization and contraction):
   - Corticosteroids inhibit the production of fibroblasts by decreasing the release of TGF-β1 and β2 from monocytes and macrophages. Without fibroblasts, there is no collagen. Generally one application when granulation is first noted to be exuberant is all that is necessary. Continued use may have a negative effect on contraction and epithelialization.
   - Cyanoacrylate spray. Completely occlusive, and so can result in maceration of the wound.
   - Cauterizing agents induce necrosis and slough of excess granulation; the most commonly used cauterizing agent is copper sulphate.
5. Cryogens. Cryogenic destruction of granulation tissue, however, impedes contraction.
6. Silicon dressings. These are used to prevent and treat hypertrophic scars in people and have been shown to prevent formation of exuberant granulation tissue in horses.
7. Grafts and biological dressings. Collagen, allografts and amnion are examples of biological dressings.
8. Excision. One of the best methods of removing excess granulation tissue. Granulation tissue contains no nerves and so, it can be trimmed without general or local anaesthesia. During excision, surface bacteria and excess leukocytes, which stimulate inflammation and hence granulation tissue, are removed.

23.11 SKIN GRAFTING

Indications for grafting

Grafting is indicated for any wound so large that it cannot heal by any other means and any open wound that cannot be sutured. It allows for rapid healing and as a result may be the most economical choice for managing any wound that cannot be sutured. In addition, a grafted wound may be more cosmetic and resilient than a wound healed primarily by epithelialization.

Graft classification

The two basic types of skin grafts are:

1. Pedicle grafts. A pedicle graft retains a vascular connection to the donor site, at least temporarily, that nourishes the graft, ensuring its viability. Pedicle grafts are useful for covering a poorly vascularized wound because the viability of the graft is not dependent on the vascularity of wound. A wound healed by pedicle grafting has a good cosmetic appearance because all components of skin are transferred. Unfortunately wounds of horses are rarely covered with a pedicle graft because the equine skin is difficult to mobilize because of its inelastic nature.
2. Free grafts. A free graft is a portion of skin that has been separated from its blood supply and transferred to a wound at another site.

Free grafts can be classified according to their source:

- Autograft (or isograft) – a graft transferred from one site to another on the same individual.
- Allograft (or homograft) – a graft transferred between different members of the same species. An allograft or xenograft is rejected eventually because the recipient mounts an immune response against it. Such a graft can still be used as a biological bandage.
- Xenograft (or heterograft) – a graft transferred from one species to another.

Free grafts can also be classified according to their thickness:

1. Full-thickness grafts – composed of epidermis and the entire dermis.
2. Split-thickness (or partial-thickness) grafts – composed of epidermis and only a portion of the dermis.

The percentage of dermis in a graft influences the acceptance, durability, and appearance; the thicker the dermis, the better is the graft's durability and cosmesis, and the thinner the dermis the more likely is the graft to survive.

Free grafts can be classified according to how they are applied:

1. Sheet grafts:
   - Meshed.
   - Non-meshed.

2. Island grafts are embedded in granulation tissue.
   - Pinch.
   - Punch.
   - Tunnel grafts.

The method of grafting selected depends on:

- Characteristics of the wound, such its age, location, and size.
- Necessity for cosmesis.
- Financial constraints imposed by the owner.
- Equipment available.
- Expertise of the surgeon.

Physiology of graft acceptance

1. Recipient bed characteristics. The recipient wound must be vascular and free of infection. A free graft is accepted on any tissue except bone devoid of periosteum, tendon devoid of paratenon, and cartilage devoid of perichondrium. Fresh wounds accept a graft more readily than do granulating wounds, and new granulation tissue accepts a graft more readily than does mature granulation tissue.
2. Sequence of events of graft acceptance:
   - The graft is adhered initially to the wound by fibrin.
   - Vessels and fibroblasts invade the fibrin by the 4th or 5th day.
   - The graft is firmly adhered to the wound by the 10th day.
   - The newly applied graft is initially nourished by plasma-like fluid imbibed, by capillary action, into the graft's vessels.
   - After 48 hours, some new capillaries anastomose with those in the graft, a process called inosculation. Some invade pre-existing vessels within the graft, and others cut new vascular channels into the dermis.
   - The graft is re-vascularized by the 5th day.
   - Some areas of epidermis may die and slough, exposing pale dermis beneath it.
   - Exposed dermis becomes covered with epithelial cells that migrate from the adnexa.
   - Re-innervation returns more rapidly, but to a lesser extent, in split-thickness grafts.

Horses may sometimes suffer from hyperaesthesia of the grafted wound resulting in self-mutilation of the wound.

Causes of graft failure

Fluid accumulation

A haematoma, seroma, or exudate beneath the graft prevents fibrin from attaching the graft to the wound and is a barrier to revascularization. A haemorrhagic wound should not be grafted until haemorrhage has ceased.

Infection

Grafts applied to horses fail most frequently because of infection; any wound that shows signs of inflammation, such as redness, swelling of the surrounding area, and formation of exudate, should be assumed to be infected. Bacteria most detrimental to a graft are β-haemolytic streptococci and *Pseudomonas* species. They destroy a graft's fibrinous attachments.

Motion

Forces that disrupt the graft are shearing forces. Shearing forces are most apt to disrupt the attachment of a graft when the wound is located in a highly mobile area, such as over a joint.

Preparing the wound for grafting

The wound should be vascular and free of infection. Fibrous or infected granulation tissue should be excised to a level below the margin of the wound to allow the wound to form fresh granulation tissue free of infection.

Streptococci can be removed by applying penicillin or another β-lactam antibiotic to the wound. β-lactamase, an enzyme produced by other resident bacteria, inactivates β-lactam antibiotics, but the efficacy of a β-lactam antibiotic against streptococci can be preserved by also applying clavulanic acid, which inhibits β-lactamase. Pseudomonads are usually susceptible to topically applied aminoglycosides or polymyxin B sulphate.

The wound should be evaluated for:

1. Damage to underlying structures such as joints, ligaments, tendons or bone.
2. Foreign bodies or sequestered bone.
3. Cutaneous habronemiasis.
4. Neoplasia.
   - Sarcoids.
   - Carcinomas.

To prepare the wound immediately before grafting:

1. Clip or shave the skin around the wound.
2. Scrub the skin around the wound. Cleanse the wound itself with physiological saline solution and avoid contact between the wound and soap. Soap increases the wound's susceptibility to infection.

Preparing the donor site

The location selected as the donor site depends on the technique of grafting selected, but the donor site should be at an inconspicuous location.

1. Hair is removed from the donor site by clipping, by shaving, or by applying a chemical depilatory agent.
2. Donor site is scrubbed and should be rinsed thoroughly.

Sheet grafts

Full-thickness sheet grafts

A full-thickness graft is usually harvested from the cranial pectoral region because the graft can be excised and the donor site sutured with the horse standing and sedated after desensitizing the donor site with local anaesthetic solution. If the wound should dehisce, healing occurs rapidly by contraction with little scarring.

Following harvesting, the subcutaneous tissue attached to the dermis is removed by sharp dissection to expose the dermis and its vasculature (Figure 23.10).

Advantages of full-thickness sheet grafting:

1. Requires no sophisticated equipment for harvesting.
2. Is performed with the horse standing.
3. Full-thickness skin grafts resist trauma better than do split-thickness skin grafts.
4. Full-thickness skin grafts provide the best cosmetic appearance of any type of free graft.

Disadvantages of full-thickness grafting:

1. Full-thickness grafts are not accepted as readily as split-thickness grafts.
2. Usually reserved for fresh wounds.
3. Practical for covering only small wounds, even when the graft is meshed (see meshing).

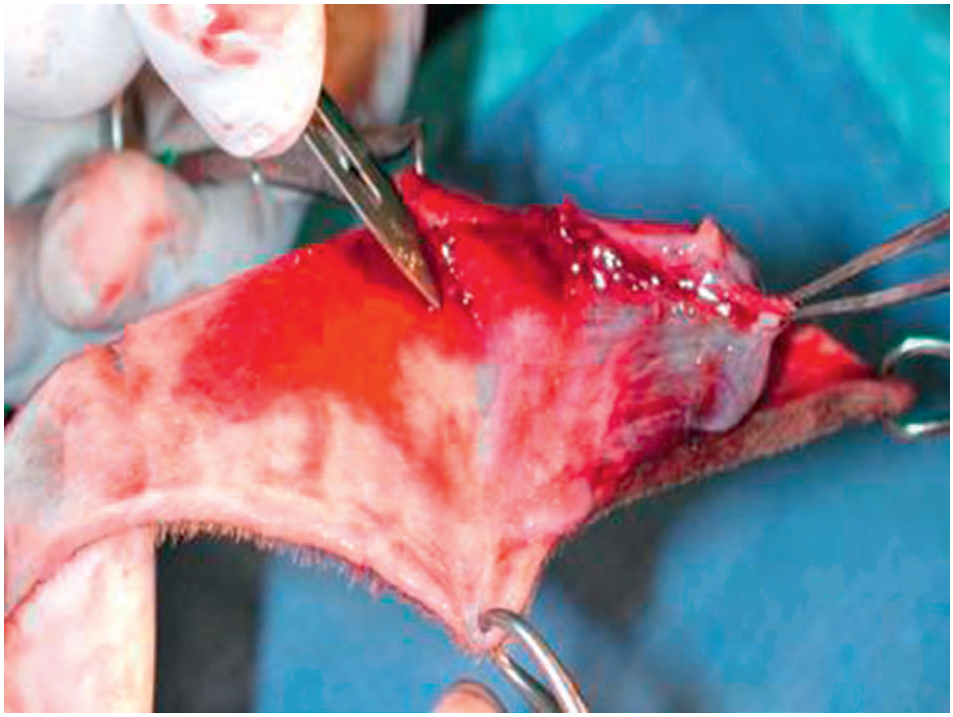


Figure 23.10 Full-thickness skin graft. Following harvesting, the subcutaneous tissue attached to the dermis is removed by sharp dissection to expose the dermis and its vasculature.

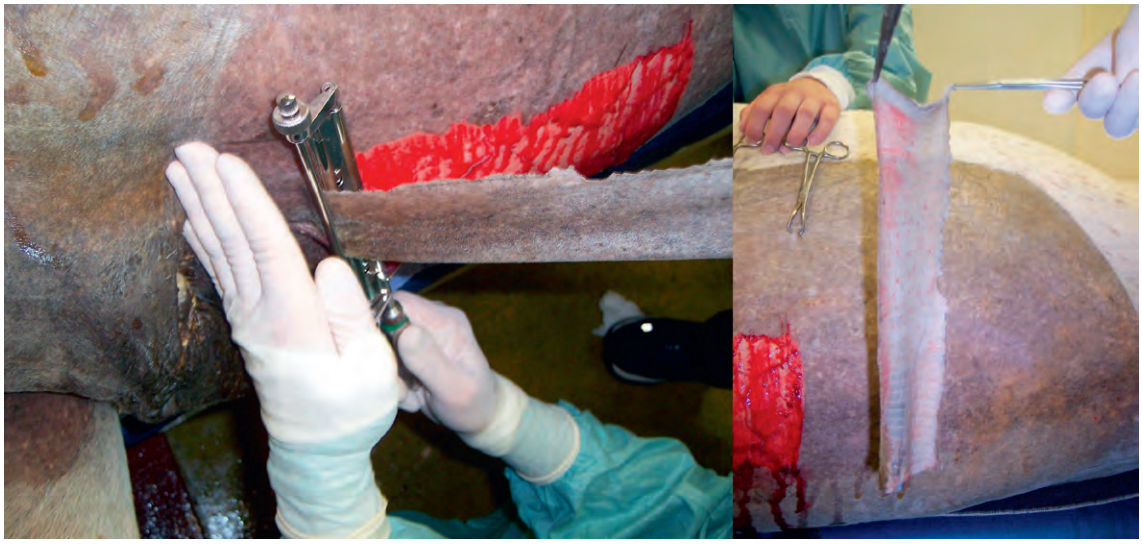


Figure 23.11 Split-thickness graft being harvested with a free-hand knife from the ventrolateral aspect of the thorax.

Split-thickness sheet grafts

A split-thickness graft is composed of epidermis and a portion of the dermis. Split-thickness grafts are harvested with a free-hand knife, a drum dermatome, or a power-driven dermatome usually from the ventrolateral aspect of the thorax or the ventral aspect of the abdomen (Figure 23.11). A free-hand knife should use disposable blades and should have an adjustable roller in front of the blade to control the depth of the cut. The thickness of the graft depends on the position of the roller, the angle of incidence at which the skin is cut, and the pressure applied to the knife. A drum dermatome has a knife that moves back and forth on a piston to split the dermis of a section of skin glued to the drum of the dermatome. It uses no external power source. The dermis can be split more precisely with the drum dermatome than with the free-hand knife, and a graft of the exact dimensions to fit the wound can be harvested. It is more expensive than a free-hand knife and is capable of harvesting a graft only as long as the circumference of the drum.

A power-driven dermatome uses an oscillating blade, driven by a gas turbine or an electric motor. The depth and width of cut can be adjusted precisely, and operating a power-driven dermatome requires

little experience. A power-driven dermatome is the most expensive type of dermatome.

Harvesting a split-thickness skin graft creates a large epithelial scar at the donor site and generally requires the horse to be anaesthetized, usually positioned in lateral recumbency.

Technique:

1. The donor site is lubricated with physiological saline solution.
2. Slight tension is applied to the cut end of the graft as the graft is harvested.
3. To determine if the graft is being harvested at the proper thickness, the first few centimetres of the graft and its donor site should be examined. A cut is too shallow if the graft is translucent, and cut is too deep if subcutaneous fat is exposed and few but large bleeding vessels are seen at the donor site.

Advantages of split-thickness grafting over full-thickness grafting:

1. Split-thickness graft can cover defects too large to be covered by a full-thickness graft.
2. Split-thickness grafts survive better than do full-thickness grafts.

Disadvantages of split-thickness grafting over full-thickness grafting:

1. The appearance of a wound healed with a split-thickness graft is inferior to that of a wound healed by a full-thickness graft.
2. To harvest a split-thickness graft, the horse must be anaesthetized.

Meshing sheet grafts (Figure 23.12)

Advantages of meshing a graft:

1. Allows a sheet graft to uniformly cover a wound larger than the graft itself.
2. Prevents mechanical disruption of the graft by allowing escape of serum, blood, or exudate from beneath the graft.
3. Enables a topically applied antimicrobial agent to contact a large portion of the wound.
4. A meshed graft conforms to an irregular surface better than does a non-meshed graft.
5. A meshed graft tolerates motion better than does a non-meshed graft.

A sheet graft is meshed by fenestrating it with staggered, parallel rows of incisions made with a scalpel blade or with a commercial meshgraft dermatome. The longer and the more numerous are the fenestrations, the greater is the expansion. Expanding a meshed graft exposes portions of the wound within the fenestrations, and these portions must heal by contraction and epithelialization, imparting diamond-shaped epithelial scars within the grafted wound.

Techniques of applying sheet grafts

Excising exuberant granulation tissue 24 hours before grafting provides time for haemostasis and for the development of budding capillaries capable of rapidly vascularizing the graft. If granulation tissue is not exuberant, the wound should be rubbed with a gauze sponge or gently scraped with a scalpel blade until the wound exudes serum.

The direction of hair growth in the graft should conform to that of the surrounding skin. It is useful to mark the direction of growth prior to clipping (a staple works well).

A graft can be sutured or stapled to the margin of the wound immediately after it is harvested (Figure 23.13), before the horse is

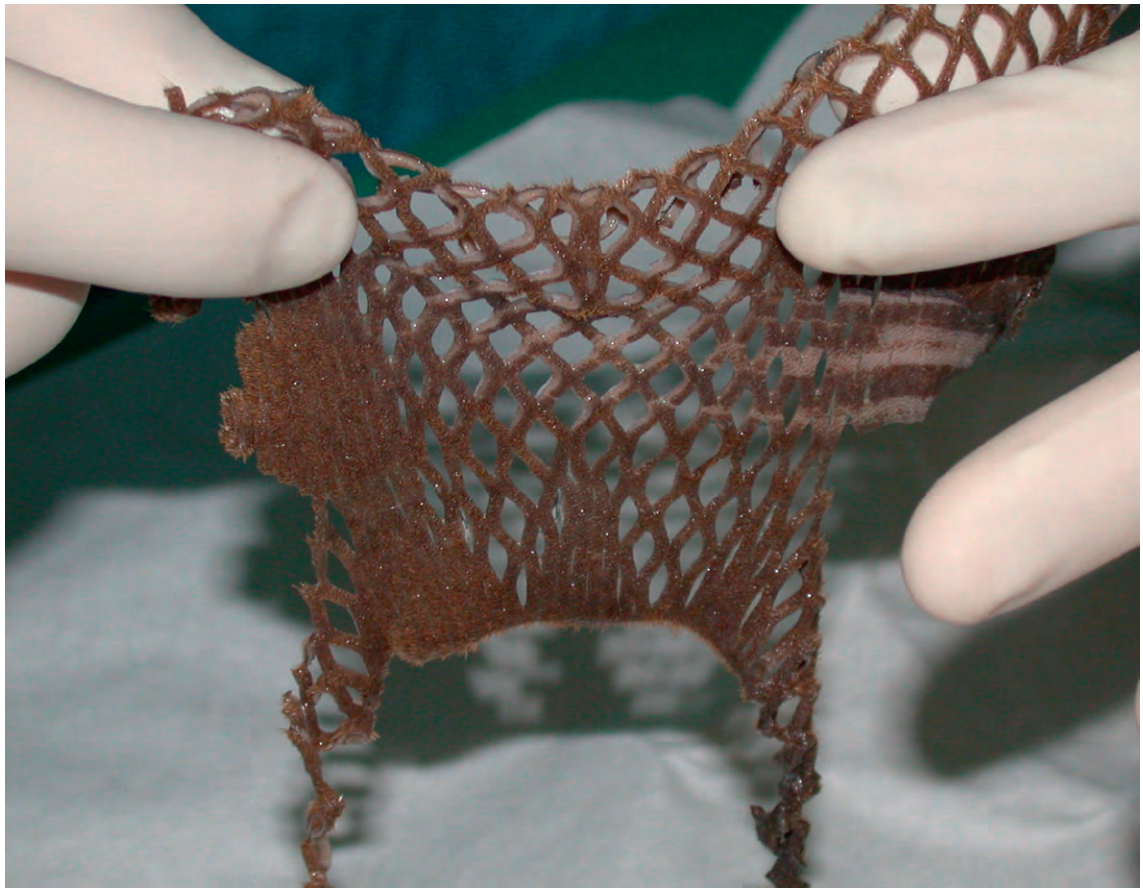


Figure 23.12 A meshed sheet graft.

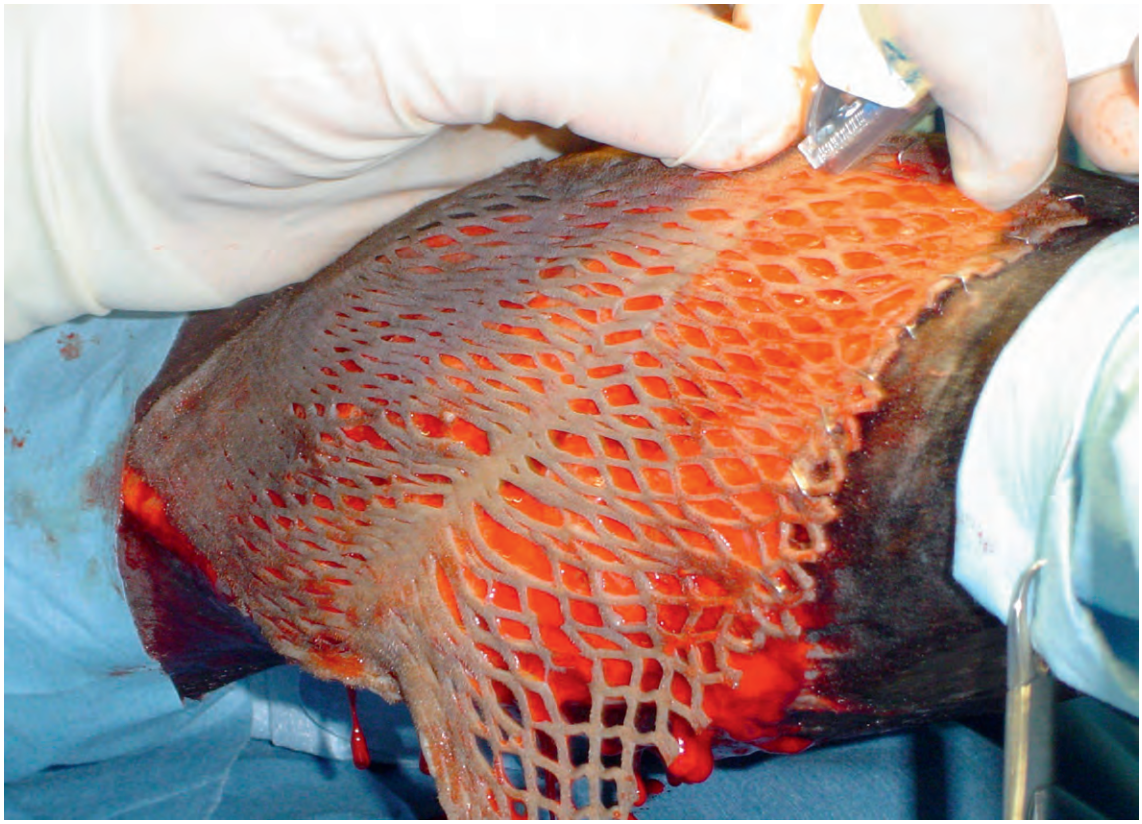


Figure 23.13 A graft being stapled to the margin of the wound immediately after harvest with the horse still under general anaesthesia.

allowed to recover from anaesthesia; however, applying the graft after the horse has recovered from general anaesthesia reduces the time of general anesthesia and removes the opportunity of the graft being damaged.

To apply a graft to a wound with the horse standing:

1. The graft is sutured or stapled to the margin of the wound after desensitizing the margins of the wound using regional anaesthesia.
2. The graft is glued to the margin of the wound with cyanoacrylate glue. A graft applied to an area that is difficult to immobilize can be further secured with simple-interrupted sutures placed through the fenestrations in the graft. Sutures can be removed in 6 or 7 days.

Acceptance and cosmesis

A thin, split-thickness skin graft is more readily accepted than is a thick, split-thickness skin graft because a thin graft has less metabolic demands and has greater number of exposed vessels for plasmatic imbibition and inosculation. The cosmetic appearance imparted by a thin, split-thickness skin graft, is poor. Split-thickness grafts between 0.63 mm (0.025 inch) and 0.76 mm (0.030 inch) thick generally provide a good cosmetic appearance at the recipient site.

Island grafting

Island grafting entails placing a small piece of split-thickness or full-thickness skin either into or onto a granulating wound. Types of island grafts are the punch graft, the pinch graft, and the tunnel graft.

1. Punch grafting

Punch grafting is the harvesting and implanting of full-thickness discs of skin into granulation tissue using skin biopsy punches. (Figure 23.14) Punch grafts can be harvested directly from the horse, usually from the neck or perineum, and the small wounds created by the biopsy punch can be sutured or stapled or left open to heal by second intention (Figure 23.15). Removing punch grafts directly from the horse creates small blemishes at the donor site.

Following harvesting subcutaneous fascia and fat are sharply excised from each individual graft.

Alternatively, punch grafts can be harvested from a full-thickness sheet of skin, usually excised from the pectoral region. A 12- to 10-cm-long by 5- to 4-cm-wide section of skin provides sufficient grafts for most wounds, and the donor site is closed in one or two layers. Subcutaneous tissue is sharply excised from the graft, and discs are cut from the graft using a 7- to 6-mm-diameter skin biopsy punch.

Recipient holes are created, about 6 to 10 mm apart, with a punch about 2 mm less in diameter to allow for contraction of the graft. Creation of the recipient holes should begin distally and proceed proximally so that the wound is not obscured by haemorrhage. The grafts are inserted into the holes, and the grafted wound is covered with a non-adherent dressing and a bandage.

The pigmented layer of the grafts frequently sloughs, exposing deeper, non-pigmented layers.

At about 3 weeks, each graft is surrounded by a red ring of migrating epithelial cells.

Advantages of punch grafting:

1. It can be performed with the horse standing.
2. It requires no expensive equipment.

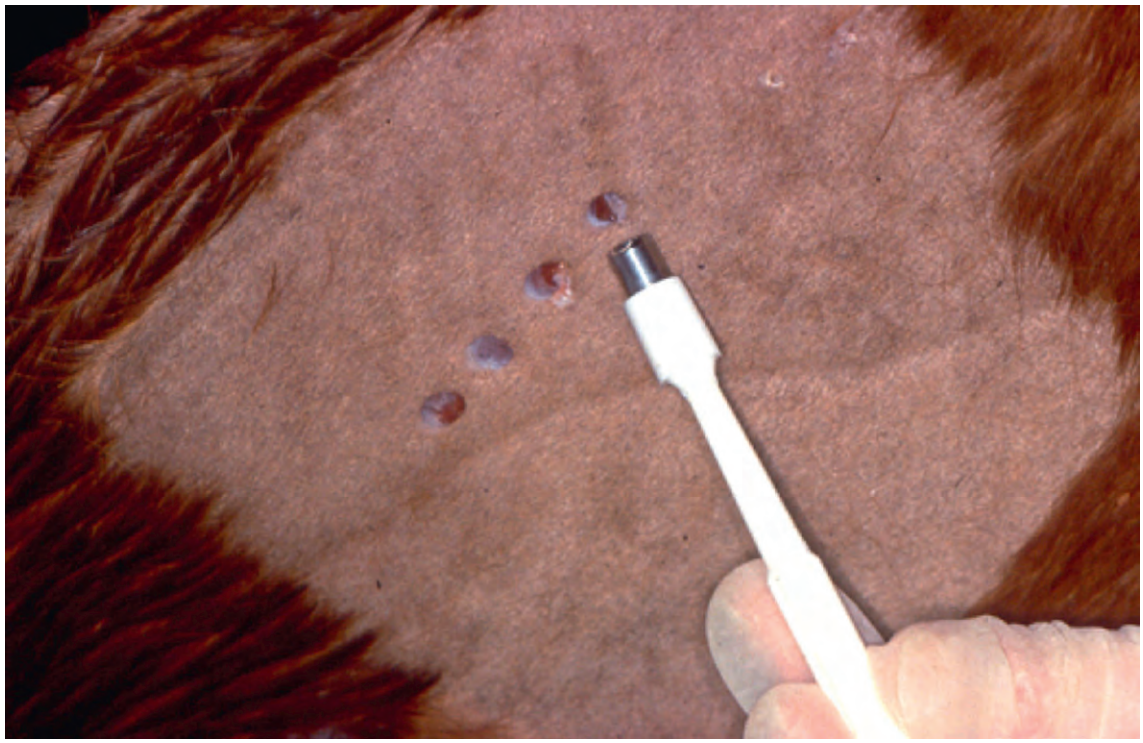


Figure 23.14 Punch grafts being harvested from the neck using skin biopsy punches.

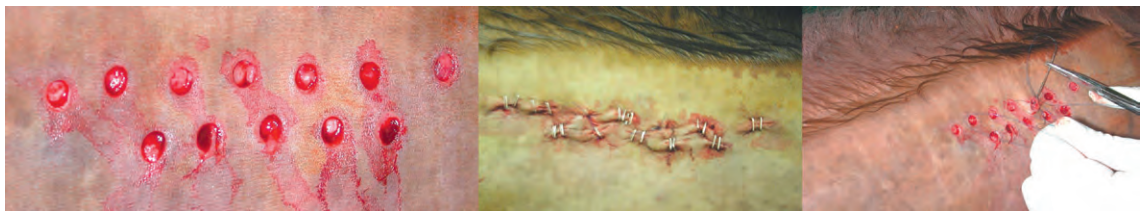


Figure 23.15 The small wounds created by the biopsy punch can be left open to heal by second intention or sutured or stapled.

3. It requires little expertise.
4. Grafts are accepted into granulating wounds that may be unreceptive to a sheet graft.
5. The grafts are not subjected to shearing forces between the wound and the bandage.

Disadvantages:

1. It is usually reserved for small wounds.
2. It is reserved for granulating wounds.
3. The cosmetic appearance of wounds healed by punch grafting is poor. The wound is covered primarily by an epithelial scar from which grow sparse tufts of divergent hair.

2. Pinch grafts

Pinch grafting is the harvesting and implanting of split-thickness discs of skin onto or into granulation tissue. The donor sites for harvesting pinch grafts are the same as those for harvesting punch grafts.

A cone of skin is elevated with a hypodermic needle with a bent point or a tissue forceps and transected with a scalpel blade. Wounds created at the donor site are left to heal by second intention or closed with a single suture or staple. The graft should be about 3 mm in diameter resulting in a graft which is nearly full-thickness at its centre, but its thickness diminishes toward its periphery.

To implant the disc of skin, a #15 scalpel blade is stabbed into the wound at an acute angle to create a shallow pocket into which the graft is inserted. Similarly to punch grafting, creation of the recipient holes should begin distally and proceed proximally so that the wound is not obscured by haemorrhage. Each graft is inserted into a pocket using the scalpel blade used to create the pocket. The grafted wound is covered with a nonadherent dressing and a bandage.

The thin layer of granulation tissue covering each graft sloughs at about a week after grafting, and the pigmented layer of the grafts frequently sloughs, exposing deeper, non-pigmented layers.

The grafts become surrounded by a red ring of migrating epithelium in about 3 weeks.

The advantages and disadvantages of pinch grafting are the same as for punch grafting.

3. Tunnel grafting

Tunnel grafting is the implantation of strips of split-thickness or full-thickness skin, about 3 mm wide, into tunnels created in granulation tissue. Tunnel grafts can be harvested directly from the horse, usually the neck or flank, or from a section of full-thickness or split-thickness skin harvested from the pectoral region or abdomen. If the strip of skin is full-thickness, its subcutaneous tissue should be removed to

expose the dermis. Such grafts can be procured and implanted with the horse anaesthetized or sedated.

To implant a strip of skin in a granulating wound:

1. The jaws of a thin, alligator forceps are inserted into the margin of the wound, at right angles to the convex surface of the wound, and advanced through the granulation tissue at a depth of about 2 to 4 mm until they emerge at the opposite margin of the wound.
2. An end of the graft is grasped in the jaws of the forceps, and the graft is pulled through the tunnel.
3. The graft is implanted in two steps if the wound is wider than the forceps or if the wound is so convex that it cannot be spanned by the forceps.
4. The strips should be embedded about 2 cm apart.
5. If the grafts were implanted deep within the granulation tissue (i.e. >4 mm), the granulation tissue covering each graft should be excised, with the horse sedated or anaesthetized, 6 to 10 days after grafting, to expose the graft. If the graft is buried shallowly (i.e. <2 mm), the granulation tissue may slough at about a week to expose the graft.

Advantages of tunnel grafting:

1. It does not require expensive equipment and can be performed with the horse standing.
2. Tunnel grafts are accepted in wounds located in highly mobile areas because the grafts are not affected by movement.

Disadvantages of tunnel grafting:

1. It can be used only on granulating wounds.
2. The cosmetic appearance is poor.
3. Removing granulation tissue covering the grafts can be tedious and difficult.

4. Aftercare of the donor site

1. The horse should receive an analgesic drug, such as phenylbutazone before a split-thickness graft is harvested and for several days thereafter.
2. A scab forms over the dermis of the donor site of a split-thickness graft, and beneath this scab, epithelial cells from the margin of the wound and from adnexa migrate to cover the abrasion.
3. Pink epithelium covers the donor site usually within 3 weeks.
4. The pink epithelial scar begins to become pigmented within a month causing the scar to darken.

5. Aftercare of the recipient site

1. The grafted wound should be covered with a sterile, non-adherent dressing, which is secured to the wound with sterile, elastic rolled gauze.
2. The secondary layer of the bandage should be bulky, to decrease motion, and absorbent, to wick exudate from the wound.
3. If the grafted wound is in a highly mobile area, immobilizing the limb with a cast or a splint may be necessary.
4. If nosocomial infections with streptococci or pseudomonads are a problem in the hospital, the bandage should be changed daily for at least 5 or 6 days. Otherwise, the bandage should not be changed for 4 or 5 days to avoid disturbing delicate vascular attachments to the graft.
5. A broad-spectrum antimicrobial drug effective against both *Pseudomonas* and β-haemolytic *Streptococcus* should be applied topically to the wound at each bandage change.
6. Complete loss of a sheet graft later than 5 days after grafting is uncommon.
7. A grafted wound on a limb should be protected by a bandage until the wound is completely epithelialized.
8. A grafted wound in an area that is difficult to bandage, such as the abdomen or thorax, can be protected with a Stent bandage or left unbandaged.
9. A horse may mutilate the healed, grafted wound if it develops hyperaesthesia of the graft. For this reason, grafted areas should be kept covered for an extended period after graft acceptance (a stable bandage or rug may suffice).

Allografts and xenografts

Cutaneous allografts and xenografts can be used to dress wounds of horses if autografting is physically unfeasible or economically impractical. Acceptance of an allograft or xenograft to a wound is a useful indicator that the wound is capable of accepting an autograft.

An allograft or xenograft encourages healing by promoting epithelialization and angiogenesis, by retarding formation of exuberant granulation tissue, and by acting as a barrier against bacterial infection. A graft decreases surface infection within several hours. Viable, cutaneous allografts can be obtained from refrigerated cadavers 24 hours or longer after the horse has died. A vascularized cutaneous allograft can survive for 2 to 3 weeks. A cutaneous xenograft provokes a more intense immunologic rejection than does a cutaneous allograft.

Storage of grafts

Cutaneous grafts can be accepted by wounds after being stored in a refrigerator for several weeks in physiological saline solution or lactated Ringer's solution. Skin can be stored in a refrigerator for at least 3 weeks when placed in a tissue-culture medium, such as McCoy's 5A Medium, to which serum has been added. The serum can be a commercially available antibody free serum, the horse's own serum, or pooled homologous serum. The concentration of serum in the storage medium should be between 10% and 33%.

Indications for storing a graft:

1. To avoid a second anaesthetic period. A split-thickness skin graft can be harvested while the horse is anaesthetized for treatment of a wound, such as lavage of a joint that communicates with the wound, and stored until the wound is healthy enough to permit grafting.
2. To enhance re-vascularization of the graft. A stored graft is often more readily accepted than a fresh graft, because grafts stored for 24 hours or more release anaerobic metabolites that stimulate angiogenesis.
3. To avoid a second surgery to procure more graft if a portion of a primary graft sloughs. For this reason, harvesting more skin than is required to cover a wound is often prudent.
4. To bank skin for allografting.

FURTHER READING

Stashak TS, Theoret CL (eds) (2008) Equine wound management, 2nd edn. Wiley-Blackwell, Oxford

Hendrickson DA (2005) Wound care management. Made easy series. Teton New Media, Jackson, WY

Chapter 24

Sedation and anaesthesia

Thomas J. Doherty, Alexander Valverde

CONTENTS

24.1 Risks associated with equine anaesthesia 489
24.2 Patient preparation 489
24.3 Drugs used for sedation 490
Phenothiazines 490
Alpha$_2$ adrenergic agonists 490
Opioids 490
Benzodiazepines 490
24.4 Sedation of adult horses 490
Sedation using constant rate infusions (CRI) 491
24.5 Sedation of foals 491
24.6 Induction of general anaesthesia in adults 491
Induction regimens 491
24.7 Prolonging anaesthesia with injectable drugs 492
24.8 Total intravenous anaesthesia 492
24.9 Inhalational anaesthesia 493
Advantages of inhalational anaesthetics 493
Disadvantages of inhalational anaesthetics 493
Recovery from inhalational anaesthesia 493
24.10 Anaesthesia of foals 493
Induction with an inhalational anaesthetic 493
Induction with injectable anaesthetics 493
Maintenance of anaesthesia 493
24.11 Analgesia 494
Non-steroidal anti-inflammatory drugs (NSAIDs) 494
Opioids 494
Alpha$_2$ agonists 494
24.12 Epidural anaesthesia and analgesia 494
Indications for epidural injections 495
Caudal epidural technique 495
Anterior (cranial) epidural technique 496
Drugs administered by the caudal epidural route 496
24.13 Complications of general anaesthesia 496
Complications at induction 496
Intraoperative complications 497
Postoperative complications 497
Further reading 497

24.1 RISKS ASSOCIATED WITH EQUINE ANAESTHESIA

It is important to keep in mind that, when compared to anaesthesia of small animals, anaesthesia of the horse carries a relatively high risk of morbidity and mortality. The risk of anaesthesia-related accidents can be greatly reduced by proper planning and attention to detail.

The temperament of the horse increases the risk of injury at induction and recovery, and its large body mass contributes to the development of myopathy and neuropathy while it is recumbent. Injuries during the recovery period may result from myopathy, neuropathy, or muscle weakness subsequent to intraoperative hypotension.

- The risks of anaesthesia increases with age of the horse, type of surgery (highest risk with fracture repair), emergency procedures, and duration of surgery.
- Anaesthesia maintained with inhalational drugs is thought to impart a higher risk than anaesthesia maintained with injectable anaesthetics, and this is probably due to greater cardiovascular depression induced by inhalational drugs.

24.2 PATIENT PREPARATION

- A physical examination should be performed, and the horse's medical history obtained. The cardiovascular and respiratory systems should be the focus of the physical examination. Conditions that may affect recovery or induction, such as musculoskeletal injury or neurological disease, should also be considered and, if necessary, plans should be implemented to assist the horse during induction and recovery.

© 2013 Elsevier Ltd
DOI: 10.1016/B978-0-7020-2801-4.00024-9

- Laboratory tests are not generally necessary for healthy horses but are warranted for some horses undergoing general anaesthesia, especially for an emergency procedure.
- Whether horses should be fasted is controversial. Fasting may not be necessary in most cases, but the mouth should be flushed with water to remove debris. This is especially important if tracheal intubation is intended.
- A jugular catheter should be placed if general anaesthesia is intended or when prolonged sedation, using a constant infusion of drugs, is planned.
- Preoperative stabilization of the cardiovascular system with intravenously administered fluids is necessary for some horses undergoing emergency surgery (e.g. intestinal accident).

24.3 DRUGS USED FOR SEDATION

Sedation is commonly necessary to facilitate a variety of surgical and non-surgical procedures in the standing horse. Sedating horses prior to the induction of anaesthesia is also common practice. The most commonly used sedatives include drugs in the phenothiazine and alpha$_2$ agonist classes. Opioids are not used as the sole agents for sedation but may be used to augment sedation and analgesia in a suitably sedated horse.

Phenothiazines

- Phenothiazines are purported to lower the seizure threshold, but this property is not proven.
- The most commonly used member of the group is acepromazine.
- *Acepromazine* has a number of physiological effects:
  Produces a mild tranquillizing effect. *Acepromazine* alone, however, does not give reliable restraint.
  Decreases the minimum alveolar concentration (MAC) of volatile anaesthetics.
  Decreases release of histamine from mast cells.
  Has cardiovascular effects, including an anti-arhythmogenic effect and vasodilation. Because of its vasodilating actions, *acepromazine* is not recommended for use in dehydrated horses or for use in those with cardiovascular compromise, such as may occur during endotoxaemia.
  Analgesia is not considered to be significant.
  Penile paralysis and priapism are potential, adverse side-effects. Keeping the dose to a minimum may reduce the likelihood of either of these complications occurring.

Clinical use of acepromazine

Acepromazine may be used alone to produce a mild tranquillizing effect, but more often it is used in combination with an alpha$_2$ agonist (e.g. xylazine).

- The dose for acepromazine is 0.02–0.05 mg/kg, i.v. or i.m. There is no benefit in increasing the dose beyond this range in an attempt to induce more profound tranquillization.
- The onset of action is approximately 15 min after i.v. administration and 30 min after i.m. administration.
- Acepromazine can be combined with an alpha$_2$ agonist (e.g. xylazine) and an opioid (e.g. butorphanol) for more profound sedation and analgesia.

Alpha$_2$ adrenergic agonists

Drugs in this group include *xylazine*, *detomidine*, *medetomidine*, *dexmedetomidine*, and *romifidine*. Alpha$_2$ agonists have profound, dose-dependent, sedating, and analgesic effects. Alpha$_2$ receptors are found throughout the central and peripheral nervous system and in many organs.

- A variety of physiological effects result from the administration of alpha$_2$ agonists and include:
  Sedation, the degree and duration of which is dose-dependent.
  Analgesia (somatic and visceral).
  A decrease in the MAC of volatile anaesthetics.
  Cardiovascular changes, including bradycardia (often accompanied by a second-degree heart block), hypertension followed by hypotension, and a decrease in cardiac output.
  Hyperglycaemia, resulting from an inhibition of insulin release.
  Contraction of the gravid uterus, but this does not seem to create a problem. Nevertheless, advising the owner of the risk of sedation of a mare in late pregnancy is prudent.

Clinical use of alpha$_2$ agonists

Alpha$_2$ agonists are the most commonly used drugs for sedation of horses for standing procedures. They can be used alone or combined with other drugs, such as opioids (e.g. *butorphanol* or *morphine*) or *acepromazine*. Although alpha$_2$ agonists can cause profound sedation, it is important to remember that the horse may still react to noise or noxious stimuli, and therefore, the handler should remain with the sedated horse.

- The degree of sedation, and hence the duration of effect, can be controlled by titrating these drugs to effect.
- When administered i.v., the onset of peak effect is about 5 min. The i.m dose is two- to three-fold that of the i.v. dose, and the onset of peak effect is a minimum of 15 min.

Opioids

Drugs (e.g. *butorphanol, morphine, buprenorphine, meperidine* [*pethidine*]) in this category are not used alone for sedation, because they are likely to cause dysphoria or excitement, especially if given as a bolus i.v. to the unsedated, healthy, adult horse.

- Release of histamine can result from opioid administration, and is more likely following i.v. administration of meperidine. Severe hypotension may result.

Benzodiazepines

Drugs of this category are not used for sedating adult horses due to their muscle-relaxing and ataxia-inducing properties; however, benzodiazepines can be used to sedate very young foals (see 24.5 'Sedation of foals').

24.4 SEDATION OF ADULT HORSES

Short-term sedation is generally achieved by administering a bolus of an alpha$_2$ agonist, with or without an opioid.

- Depending on the degree of sedation required, i.v. bolus injections in the following dose ranges can be used:
  Xylazine (0.25–1.1 mg/kg)
  Detomidine (0.005–0.02 mg/kg)
  Romifidine (0.03–0.1 mg/kg)
  Dexmedetomidine (0.0015–0.0025 mg/kg)
  Medetomidine (0.0035–0.007 mg/kg).
- When used in combination with an opioid, the horse **must be** sedated with the alpha$_2$ agonist prior to opioid administration,

to avoid opioid-induced excitement. An opioid is combined with an alpha$_2$ agonist only when heavy sedation or additional analgesia is required. Doses of the individual drugs should be tailored to achieve the desired effect. Bolus injections (i.v.) of the combinations, in the following dose ranges, can be used:
Xylazine (0.5–1.0 mg/kg) plus *butorphanol* (0.02–0.05 mg/kg)
Xylazine (0.5–1.0 mg/kg) plus *morphine* (0.15 mg/kg)
Detomidine (0.005–0.015 mg/kg) plus *morphine* (0.15 mg/kg)
Romifidine (0.03–0.08 mg/kg) plus *butorphanol* (0.02–0.05 mg/kg)
Medetomidine (0.0035–0.005 mg/kg) plus *butorphanol* (0.02–0.05 mg/kg)
Medetomidine (0.0035–0.005 mg/kg) plus *morphine* (0.15 mg/kg).

Sedation using constant rate infusions (CRI)

Infusions are used when prolonged standing sedation is necessary, such as when surgery is performed with the horse standing. A CRI has the advantage of providing a more steady-state of sedation.

- An i.v. catheter is placed, and the CRI of drugs is generally administered by adding the drugs to a 1 L bag of fluids (e.g. 0.9% NaCl).
- A loading dose (Ld) of the drug(s) is given before the CRI is started. Doses in the following ranges can be used:
  Detomidine: Ld = 0.005–0.01 mg/kg i.v. and a CRI = 0.02 mg/kg/hour.
  Medetomidine: Ld = 0.005 mg/kg i.v. and a CRI = 0.0035–0.005 mg/kg/hour.
  Dexmedetomidine: Ld = 0.0025 mg/kg i.v. and a CRI = 0.0017–0.0025 mg/kg/hour.
  Romifidine: Ld = 0.03–0.1 mg/kg and a CRI = 0.018 mg/kg/hour.
  Xylazine: Ld = 0.5–1 mg/kg i.v. and a CRI = 0.5–1.0 mg/kg/hour.
- An alpha$_2$ agonist can be combined with an opioid for CRI. In such cases, the dose of each drug may have to be reduced to avoid over sedation.
  Detomidine: Ld = 0.005 mg/kg i.v. and a CRI = 0.02 mg/kg/hour, *Butorphanol*: Ld = 0.02 mg/kg, i.v. and a CRI = 0.012 mg/kg/hour.
  Detomidine: Ld = 0.005 mg/kg, i.v. and a CRI = 0.02 mg/kg/hour, *Morphine*: Ld = 0.15 mg/kg, i.v. and a CRI = 0.15 mg/kg/hour.
  Medetomidine: Ld = 0.005 mg/kg, i.v. and a CRI = 0.0035–0.005 mg/kg/hour, *Morphine*: Ld = 0.15 mg/kg, i.v. and a CRI = 0.15 mg/kg/hour.
- If necessary, sedation or analgesia may be augmented during the CRI by giving a bolus of approximately ¼ of the loading dose of the alpha$_2$ agonist.

24.5 SEDATION OF FOALS

Neonatal foals become recumbent once sedated, and the foal should be supported until it assumes recumbency. Administration of alpha$_2$ agonists should be avoided in sick neonates and should be used cautiously in foals with respiratory compromise. In healthy neonates, alpha$_2$ agonists can be given safely, but the dose should be kept to a minimum.

- Older foals are sedated in a manner similar to that of adults but may require higher doses to achieve the equivalent effect.

Benzodiazepines (e.g. diazepam or midazolam [0.05–0.1 mg/kg i.v.])

Benzodiazepines should be administered slowly. Ataxia may be prolonged in some cases, and thus, high or repeat dosing is not recommended. These drugs are not analgesic and, alone, are not recommended for painful procedures.

Benzodiazepine and ketamine

- These can be combined for painful procedures (e.g. joint lavage).
- Ketamine (2–3 mg/kg) can be mixed with diazepam or midazolam (0.05–0.1 ml/kg), and the mixture can be dosed to effect to produce a light plane of anaesthesia. The advantages of this regimen over a benzodiazepine alone are the provision of analgesia and faster recovery and reduced ataxia from using a lower dose of the benzodiazepine.
- Premedication with a low dose of an alpha$_2$ agonist (e.g. xylazine 0.25–0.5 mg/kg, i.v.) augments the aforementioned drugs and may be necessary for stronger foals.

24.6 INDUCTION OF GENERAL ANAESTHESIA IN ADULTS

Most periods of general anaesthesia in horses are of short duration (i.e. <1 hour), and the effects of the drugs used for sedation and induction may be of sufficient duration to allow completion of the surgical procedure (e.g. castration). Under hospital conditions, it is common practice to maintain anaesthesia with inhalational anaesthetics or combinations of inhalational and injectable drugs. Under field conditions, injectable drugs are normally used to extend the period of anaesthesia.

- Horses should be sedated before induction of anaesthesia, regardless of the method of induction.
- Sedation improves the ease of handling, thus making induction safer for the horse and its handler(s), and decreases the dose of induction drugs.
- In most cases, sedating agents (e.g. acepromazine and alpha$_2$ agonists) also decrease the dose of maintenance drugs and provide analgesia (e.g. alpha$_2$ agonists and opioids) and may improve the quality of recovery by prolonging the time until the horse's first attempt to stand (e.g. acepromazine and long-acting alpha$_2$ agonists [e.g. detomidine and romifidine]).
- Alpha$_2$ agonists are the most commonly used group of drugs for pre-anaesthetic sedation. They can be used alone or in combination with opioids (e.g. butorphanol) or acepromazine.
- Acepromazine improves the quality and speed of induction when used to augment sedation with alpha$_2$ agonists.

Induction regimens

Only a few drugs are available for induction of anaesthesia; however, there are a variety of drug combinations and permutations in use. The most common drug regimens are discussed here.

Ketamine and an alpha$_2$ agonist

- Ketamine, an antagonist at N-methyl-D-aspartate (NMDA) receptors, is the most widely used induction drug.
- It is imperative that ketamine be given only after the horse is heavily sedated. If given to the non-sedated or lightly sedated horse, extreme excitement or seizures may occur.
- Ketamine (2.2–2.5 mg/kg, i.v.) is administered after heavy sedation with an alpha$_2$ agonist (e.g. xylazine [1.1–1.4 mg/kg, i.v.]; detomidine [0.01–0.02 mg/kg, i.v.]).
- The higher doses of ketamine and alpha$_2$ agonists are recommended for young horses and for ponies.

- The duration of surgical anaesthesia is short (5–10 min), but the horse may remain recumbent for 40 min, if not stimulated.
- Ketamine does not produce skeletal muscle relaxation.

Ketamine/benzodiazepine and an alpha$_2$ agonist

- Benzodiazepines improve muscle relaxation and sedation.
- Ketamine can be combined in the same syringe with the benzodiazepine (e.g. diazepam [0.02–0.1 mg/kg]) for injection.
- The benzodiazepine allows for a reduction in the dose of the alpha$_2$ agonist, such as might be indicated for horses with a compromised cardiovascular system.
- The duration of anaesthesia is not significantly prolonged by the benzodiazepine.

Thiopental and an alpha$_2$ agonist

- Thiopental is a barbiturate with activity at GABA receptors. Cardiovascular and respiratory depression is dose-dependent.
- Thiopental (10–12 mg/kg, i.v.) can be used for induction of anaesthesia in the non-sedated horse, but induction and recovery are poor. Sedation with an alpha$_2$ agonist (e.g. xylazine 1.1 mg/kg, i.v.) allows the use of lower doses of thiopental (3–5 mg/kg, i.v.) and results in acceptable induction and recovery.
- Thiopental provides good muscle relaxation but has no analgesic properties.
- The duration of surgical anaesthesia is relatively short (5–10 min).

Tiletamine-zolazepam (TZ) and an alpha$_2$ agonist

Tiletamine is an NMDA antagonist, and *zolazepam* is a benzodiazepine. This combination may be used for induction, and the effects of the combination resemble the effects of the combination of *ketamine* and *diazepam/midazolam* in that it provides analgesia (when used with an alpha$_2$ agonist) and muscle relaxation.

- The TZ combination is administered after the horse is heavily sedated with an alpha$_2$ agonist (e.g. xylazine 1.1 mg/kg, i.v.).
- At a dose of 1.5 mg/kg, i.v., TZ induces a duration of anesthesia approximately twice that resulting from *ketamine/xylazine* administration.

Guaiphenesin with thiopental or ketamine

Guaiphenesin is a centrally acting muscle relaxant. It has minimal anaesthetic or analgesic effects and, at clinical doses, has minimal cardiorespiratory depressant effects. Due to the irritant nature of *guaiphenesin* and because large volumes are needed (e.g. 0.5–1.0 L of a 5% solution to a 450 kg horse), a catheter should be placed to avoid perivascular administration.

- It can be combined with an induction agent (e.g. ketamine [2–2.5 mg/kg] or thiopental [3–5 mg/kg]). The induction drug can be mixed with guaiphenesin, and the mixture can be delivered under pressure to speed administration. If not administered rapidly, the horse becomes ataxic before becoming recumbent.
- Alternatively, guaiphenesin can be infused rapidly until the sedated (e.g. with xylazine [0.5–1.0 mg/kg, i.v.]) horse begins to become ataxic, and then a bolus of the induction drug (e.g. ketamine [2–2.5 mg/kg] or thiopental [3–5 mg/kg]) can be administered.

Immobilon

This combination of *etorphine* and *acepromazine* is available in some countries. The mixture is lethal to human beings if inadvertently injected or splashed onto the mucosa, so extreme care is necessary, and an opioid antagonist (naloxone) should be available.

- Anaesthesia is characterized by poor muscle relaxation, tachycardia, hypertension, respiratory depression, and occasionally hyperthermia. Priapism occurs commonly. The eyes remain open and should be protected from injury.
- Analgesia is excellent, and the duration of anaesthesia is sufficiently long for most field procedures.
- Anaesthesia is terminated by injecting the antagonist, *diprenorphine*.

24.7 PROLONGING ANAESTHESIA WITH INJECTABLE DRUGS

Because common induction regimens are short-acting (5–15 min), anaesthesia must frequently be prolonged to allow humane and safe completion of the surgical procedure. The simplest and most common method of prolonging anaesthesia for short periods is to give a bolus injection of an alpha$_2$ agonist and ketamine.

- If duration of anaesthesia beyond the effect of the induction drugs is anticipated, it is prudent to have a 'top up' dose ready to administer prior to the horse becoming too light. Waiting until the horse moves spontaneously or in response to surgical stimulus requires a higher dose of induction drug to deepen anaesthesia.
- Xylazine and ketamine, at approximately one-quarter to one half of the initial doses, administered together as a bolus, prolong anaesthesia for 5–10 min. Anaesthesia can be extended safely using repeated injections, but maintaining a stable plane of anaesthesia for a prolonged period, using this method, is difficult.
- Thiopental (1–3 mg/kg, i.v.) is very effective at deepening anaesthesia and is more rapid in onset than are xylazine and ketamine in combination. Repeatedly using thiopental to deepen anaesthesia is not wise because thiopental accumulates in the tissues, adversely affecting recovery.

24.8 TOTAL INTRAVENOUS ANAESTHESIA

If the duration of anaesthesia is expected to exceed 30 min, a CRI should be used to maintain a stable plane of anaesthesia.

- The most commonly used drug regimen is the combination of xylazine, romifidine, or detomidine combined with ketamine and guaiphenesin. For adult horses, ketamine (1–2 g) and xylazine (500 mg) or romifidine (50 mg) or detomidine (20 mg) are added to 1 L of guaiphenesin (5%), and the mixture is infused at 1–3 mL/kg/h.
- Thiopental (2–3 g) can be mixed with 1 L of guaiphenesin (5%) and infused at 2–3 mL/kg/h. Thiopental is not the best choice for maintenance if anaesthesia was induced with thiopental or for a long procedure, because thiopental accumulates in the tissues resulting in a rough recovery.

24.9 INHALATIONAL ANAESTHESIA

Inhalational anaesthesia (e.g. *halothane*, *isoflurane*, *sevoflurane*) is the most common method of maintaining anaesthesia in the hospital setting. Halothane is less commonly used nowadays.

- The potency of inhalational anaesthetics is defined in terms of the MAC, which is the minimum alveolar concentration needed to prevent purposeful movement in 50% of subjects.
- The approximate MAC values for inhalational anaesthetics in adult horses are: isoflurane 1.3%, sevoflurane 2.3%, desflurane 7.6%, and halothane 0.85%.
- The MAC values in neonates are usually 30% lower than those in adult horses.
- For surgical procedures, the end-tidal concentration should be 30–50% greater than the MAC.
- MAC is reduced by sedatives (e.g. acepromazine, benzodiazepines, and alpha$_2$ agonists), anaesthetics (e.g. ketamine, thiopental, and propofol), or miscellaneous drugs (e.g. lidocaine). Opioids alone do not significantly reduce MAC in horses.

Advantages of inhalational anaesthetics

- Drug accumulation is minimal with modern, less-soluble drugs (e.g. isoflurane, sevoflurane).
- The end-tidal concentrations can be monitored, thus allowing an accurate determination of dosing; monitoring, however, requires a relatively expensive apparatus.
- Relatively rapid changes in the depth of anaesthesia can be made with less-soluble inhalational anaesthetics; using an injectable anaesthetic (e.g. ketamine, thiopental) for this purpose, however, is more effective.
- Inhalational anaesthetics can be used in combination with injectable agents, and this combination can decrease MAC, thereby reducing cardiovascular depression, and in some cases, can provide analgesia.

Disadvantages of inhalational anaesthetics

- Dose-dependent cardiovascular depression.
- Minimal analgesic effects.
- A relatively narrow therapeutic index (LD_{50}/ED_{50}).
- Pollution of surgical suite – a possibility.
- Rapid recoveries that are generally not as good as with injectable drugs.

Recovery from inhalational anaesthesia

For all inhalational anaesthetics, the quality of recovery is negatively correlated to the duration of anaesthesia. Following a short period of anaesthesia, recovery from isoflurane and sevoflurane is relatively fast and sometimes uncontrolled, and for this reason, some clinicians prefer halothane. Sedating the horse decreases the speed of recovery.

- An alpha$_2$ agonist (e.g. xylazine 0.2–0.4 mg/kg, i.v.) prolongs recovery.
- A mixture of an alpha$_2$ agonist (e.g. xylazine 0.2 mg/kg) and ketamine (0.2–0.4 mg/kg) in the recovery stage is a more reliable method of prolonging recovery than is administration of an alpha$_2$ agonist alone. The total volume may be given in two or three increments over a 5-minute period.
- Administration of acepromazine prior to induction may improve the quality of recovery. Alternatively, a low dose of acepromazine (i.e. 0.01–0.02 mg/kg, i.v.) can be given towards the end of surgery.

24.10 ANAESTHESIA OF FOALS

Anaesthetic management of foals poses special problems. Newborn foals are in transition to adult cardiovascular and pulmonary systems. Although vascular shunts close physiologically during the first few days of life, anatomical closure takes 2–4 weeks, and shunts may re-open in response to hypoxia, hypercarbia, or acidaemia. Supplementary oxygen should be provided to the sedated, recumbent foal. Many neonatal foals presented for anaesthesia have systemic illness, and therefore, adequate assessment of their health is important.

- Determining colostrum intake (by checking IgG concentration in plasma) and monitoring the concentration of blood glucose are important, especially in foals suffering from bacteraemia.
- Every effort should be made to prevent heat loss.
- Fasting is not imposed on neonates, but foals eating solid foods may have food withheld for a few hours, if deemed necessary.

Induction with an inhalational anaesthetic

Induction of anaesthesia using inhalational drugs has been widely recommended on the premise that neonates have limited ability to metabolize and clear drugs and, thus, may have prolonged recoveries from injectable drugs. There is no scientific reason to believe that neonatal foals have a problem with metabolizing or clearing drugs and, based on the authors' clinical impressions, recovery from injectable drugs is rapid. It is important to understand that inhalational anaesthetics have a lower therapeutic index than do injectable drugs, and an increase in mortality has been demonstrated when an inhalational anaesthetic is used as the sole drug for induction. This method of induction is not recommended by the authors.

Induction with injectable anaesthetics

- Induction is readily and safely achieved in neonates with combination of a benzodiazepine (e.g. diazepam [0.05–0.1 mg/kg, i.v.]) and ketamine [2.0–3.0 mg/kg, i.v.]. The drugs can be combined in one syringe and given to effect. Slow administration avoids apnoea and is especially important if the neonate is weak. Oxygen should be provided (e.g. using a mask) during induction to prevent haemoglobin desaturation.
- Older, stronger foals can be premedicated with an alpha$_2$ agonist (e.g. xylazine [0.5–1.0 mg/kg]) prior to induction with ketamine and a benzodiazepine.

Maintenance of anaesthesia

Inhalational anaesthetics can be used as the sole drug for maintenance. Alternatively, a lower dose of inhalational drug can be combined with injectable drugs, such as ketamine (3 mg/kg/h) and/or lidocaine (3–5 mg/kg/h), to decrease cardiovascular depression and to provide analgesia.

24.11 ANALGESIA

Provision of analgesia is an important part of anaesthetic management. Analgesics are also indicated for many non-surgical conditions and for the management of chronic pain.

Non-steroidal anti-inflammatory drugs (NSAIDs)

This is the most commonly used class of analgesic drugs and is suitable for treating horses with moderate to severe pain. NSAIDs can be used to treat horses with acute and chronic pain and are most effective in treating horses with pain of inflammatory origin. Adverse effects include gastric ulceration, right dorsal colitis, and renal damage, but these effects are more likely to occur with chronic use. Platelet inhibition, leading to increased bleeding in the surgical patient, does not seem to be a problem in the horse. Examples of NSAIDs and their dosages include:

Flunixin meglumine (0.25–1.1 mg/kg, i.v., q8–12 h)

- Should not be given i.m., as it is a tissue irritant. Clostridial infections can develop after i.m. administration.
- Can be administered orally for the treatment of horses with chronic pain.
- Commonly used for treating horses with postoperative pain and with acute abdominal pain.

Phenylbutazone (2.2–4.4 mg/kg, i.v., or *per os*, q12–24 h)

- Primarily administered orally to treat horses with chronic orthopaedic pain (1–2 mg/kg/day).
- Also used as a perioperative analgesic (2–4 mg/kg, i.v.), especially for orthopaedic pain.
- Must not be administered i.m. because of tissue irritation.

Ketoprofen (1–2 mg/kg, i.v.; q24 h)

- May have a lower incidence of adverse effects than aforementioned NSAIDs.

Opioids

Opioids are not used routinely in the horse because of their tendency to cause adverse effects, such as excitement and ileus.

Morphine (0.1–0.3 mg/kg, i.m.; q4–6 h)

- Excitement is likely if given i.v. to the unsedated horse, and for this reason, i.m. administration is recommended,
- Ileus is a risk with repeated administration; use of morphine, therefore, should only be considered for short-term analgesia.

Butorphanol (0.02–0.04 mg/kg, i.v., i.m.; q2–4 h)

- Excitement and ileus are potential complications.
- A CRI of butorphanol has been recommended as an analgesic for horses after colic surgery; the initial loading dose is 0.02 mg/kg, i.v. followed by a CRI of 0.024 mg/kg/h.

Meperidine (1–2 mg/kg, i.m.; q2–4 h)

- It should not be administered as a bolus i.v. due to the likelihood of histamine release and subsequent hypotension.

Alpha$_2$ agonists

- Because of their sedating effects, drugs in this class are generally used to treat horses for pain only if the pain is severe and acute, such as pain associated with severe colic. Continued administration increases the likelihood of inducing clinically significant ileus.
- Alpha$_2$ agonists can be combined with opioids for control of severe pain.
- Alpha$_2$ agonists are used to treat horses with acute pain within the dose ranges for sedation.

Tramadol

Tramadol is a relatively novel analgesic compound, but it has not been studied extensively in the horse. Tramadol's analgesic actions in other species are reportedly due to its activity, albeit weak, at opioid receptors and its inhibition of the re-uptake of norepinephrine and 5-hydroxytryptamine. Although it is used as an analgesic in other species, tramadol has not been shown to have analgesic efficacy in the horse.

- Sedation has been reported after its administration i.v. to horses; clinicians should be aware, however, that excitement has also been reported following its i.v. administration. At this time, the authors recommend that tramadol not be administered i.v. to horses.
- Oral administration (2.5 mg/kg, q12 h) does not seem to cause adverse effects, but there is no report of the analgesic efficacy of this treatment.

Lidocaine infusions

Intravenous infusion of lidocaine has been used to treat horses with mild to moderate pain.

- A bolus (1.5 mg/kg, i.v.) is infused over 5–10 min and is followed by a CRI (3 mg/kg/h).

Ketamine infusions

Subanaesthetic doses of ketamine are sometimes administered i.v. or i.m. to provide analgesia, but an analgesic effect has not been demonstrated in the horse.

- Infusion rates of 0.4–0.8 mg/kg/h are not associated with adverse behavioural effects.
- In the authors' experience, combining ketamine with lidocaine (at the dose listed above) is also free of adverse effects. Using this combination, ketamine is first administered as a bolus (1 mg/kg, i.v., over 30 min) and then by CRI (0.4 mg/kg/h).

24.12 EPIDURAL ANAESTHESIA AND ANALGESIA

Some drugs can induce analgesia and/or anaesthesia when administered epidurally (see Table 24.1). Drugs administered epidurally are primarily intended for analgesia or anaesthesia of the perineal area.

Table 24.1 Dose recommendations for administering epidural drugs

Drug		Dose (mg/kg)	Onset (min)	Duration (h)	Notes
Local anaesthetics	Lidocaine 2%	0.2–0.25	6–10	1	If combined with an alpha$_2$-agonist, reduce lidocaine dose to <0.2 mg/kg
	Mepivacaine 2%	0.2–0.25	6–10	1	Up to 4 mg/kg in neonates to include abdominal dermatomes
	Ropivacaine 0.5%	0.8	10	3	
Opioids	Morphine	0.1–0.2	30–60	6–16	Diluted to 20–30 mL with normal saline solution. Combined with detomidine (0.03 mg/kg) and diluted to 5–20 mL with normal saline solution
	Methadone	0.1	15	5	Diluted to 20–30 mL with normal saline solution
	Hydromorphone	0.04	20–30	4	Diluted to 20–30 mL with normal saline solution
Alpha$_2$-agonists	Xylazine	0.05–0.2	15–30	3	Diluted to 5–10 mL with normal saline solution. If combined with local anaesthetic, use low end of dose range.
	Detomidine	0.01–0.06	15–30	2–3	Diluted to 5–10 mL with normal saline solution
Miscellaneous	Ketamine	0.5–2	10	1–1.25	Diluted to 5–10 mL with normal saline solution
	Tramadol	1	30	4–5	Diluted to 20–30 mL with normal saline solution

The epidural space is readily accessible caudal to the sacrum between the first and second coccygeal spaces (Co_1–Co_2), (caudal epidural); or at the lumbosacral space (cranial epidural).

- The lumbosacral space is rarely used for drug injection, because cranial epidural drug administration is more likely than caudal epidural drug administration to lead to motor weakness.
- Regardless of whether drugs are administered at the lumbosacral or coccygeal spaces, severe ataxia, motor block, and recumbency can develop if drugs spread cranially in the epidural space.
- In the case of foals, it is reasonable to consider cranial epidural anaesthesia for surgical procedures of the abdomen and pelvic limbs; it is, however, a rarely performed procedure.

Indications for epidural injections

- Anaesthesia of the perineum.
- Rectovestibular laceration repair.
- Miscellaneous surgeries involving rectum, anus, perineum, tail, urethra, bladder, vulva, vestibule, or vagina.
- Relaxation of abdominal contractions during dystocia.
- Analgesia of the perineal area and hind limbs of adult horses.
- Analgesia or anaesthesia of the perineal area, hind limbs, and abdomen of neonates.

Caudal epidural technique (See Figure 24.1)

- The caudal epidural space is located by moving the tail up and down, with one hand, while applying digital pressure with the free hand to the appropriate intervertebral space caudal to the sacrum.
- Most texts claim that the sacrococcygeal joint is fused in adults and that the anatomical site where the tail actually hinges is the first intercoccygeal space (Co_1–Co_2).
- The area should be clipped and prepared for aseptic injection.

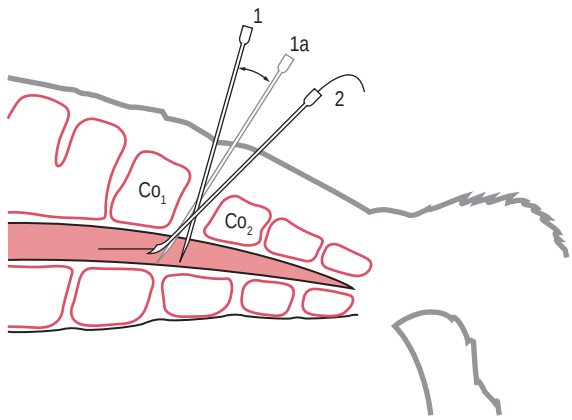


Figure 24.1 Insertion of needle into the first intercoccygeal (Co_1–Co_2) space. The needle is inserted approximately in the middle of the intercoccygeal space at an acute (1a) or obtuse (1) angle and advanced until it meets the floor of the canal. The needle is then withdrawn slightly. When placing a catheter using a Tuohy needle (2) or when attempting to slide a spinal needle into the epidural space, the needle is inserted closer to Co_2.

- Regular hypodermic needles, spinal needles, or epidural catheters can be used.
- Use a 22-, 20- or 18-gauge, 1.5- to 3.5-inch (2.54- to 8.89-cm) hypodermic needle or spinal needle, depending on the horse's size.
- Use a 17- or 18-gauge, 3–3.5 inch (7.62- to 8.89-cm) Tuohy needle for insertion of a 19- or 20-gauge epidural catheter.
- In unsedated horses, superficial infiltration with local anaesthesia (1–2 mL of 2% lidocaine using a 23- or 25-gauge needle) facilitates placement of the needle and catheter.
- The needle is introduced at 30–90° to the plane of the skin, and the angle is adjusted, as required, to facilitate placement in the epidural space. Proper placement can be verified by:
  Negative pressure observed when fluid (e.g. normal saline solution) placed in the hub of the needle is aspirated into the space (i.e. hanging drop technique).
  Lack of resistance during injection of air or normal saline solution.
  Lack of resistance during advancement of the epidural catheter.
- Catheters can be left in place for prolonged periods (weeks) for repeated administration of drugs when circumstances dictate such need.
- Catheters are normally advanced 10–30 cm into the epidural space and secured to the skin with adhesive or suture material.

Anterior (cranial) epidural technique

- This method is rarely used clinically.
- Cranial epidural injections can be performed at the lumbosacral space.
  More difficult to perform than caudal epidural.
  High likelihood of puncture of the dura and arachnoid membrane resulting in leakage of CSF or intrathecal injection.
- It has a higher risk of motor blockade and ataxia, regardless of drugs injected.
- It is important to realize that a cranial epidural can also result from the injection of a large volume of drug at the caudal epidural space.

Drugs administered by the caudal epidural route

The ideal drug for epidural administration should cause analgesia and anaesthesia, minimal motor blockade, and minimal systemic effects.

Local anaesthetics (e.g. lidocaine, mepivacaine, ropivacaine)

- Effects include analgesia, anaesthesia, motor blockade, and some sympathetic blockade.

Alpha$_2$ agonists (e.g. xylazine, detomidine)

- Effects include analgesia and anaesthesia (e.g. with xylazine) of the perineal region, some motor blockade, and systemic effects. Sedation and haemodynamic changes (e.g. bradycardia) may occur.

Opioids (e.g. morphine, methadone, hydromorphone)

- Effects include analgesia and, occasionally, mild sedation.

Tramadol

- Has been studied at 1.0 mg/kg (total volume of 20 mL in a 500 kg horse) epidurally.
- Analgesia was evident after 20 min and lasted for about 6 hours.

Ketamine

- Its effects include analgesia and some systemic effects (sedation, ataxia).
- Effects are of short duration (30–75 min).

Doses and recommendations (see Table 24.1)

- Because most epidural injections are performed in the standing horse, doses of anaesthetic drugs should be adjusted to prevent excessive ataxia and recumbency.
- Similarly, in the anaesthetized horse, epidural injections should not interfere with muscle strength and control during the recovery period.
- In the neonate, drugs in doses that also induced motor blockade and recumbency can be safely used because handling and restraint of foals under such circumstances is feasible.

24.13 COMPLICATIONS OF GENERAL ANAESTHESIA

Given the size and temperament of the horse, even the simplest complication can have a serious outcome for the horse. Complications, such as myopathy and neuropathy, are much more likely to occur during prolonged recumbency. For short procedures performed under field conditions, complications are more likely at induction.

Complications at induction

A number of complications are possible at induction, but most are avoidable.

Intracarotid injection

- This can have serious consequences, depending on the drug and volume injected, from agitation and restlessness to seizures and death. The horse may rear and fall over backwards causing a fracture of the base of the skull. Seizures should be controlled (e.g. with di*azepam*), and supplemental oxygen supplied.

Administration of the incorrect drug

- This can have serious consequences if, for example, *ketamine* is administered unintentionally to the unsedated horse. This results in excitement and staggering or collapse.

Haematoma formation

- Haematoma formation may result from failed jugular puncture, in which case the haematoma is small, or from inadvertent carotid puncture, in which case, the haematoma can become very large, compromising the airway.

Perivascular injections

- If significant volumes of an irritant substance are deposited perivascularly, the area should be lavaged with an appropriate volume of isotonic saline solution.

Injury resulting from poor inductions

- Injury to the horse or its handler(s) can result if the horse falls awkwardly.

Intraoperative complications

Clinically significant intraoperative complications related to anaesthesia are more likely during prolonged procedures.

Hypoxaemia

- Hypoxaemia is to be expected in all recumbent adult, full-sized horses and is generally of little consequence in healthy horses anaesthetized for short periods. Supplemental oxygen increases arterial oxygen partial pressure when the ventilation/perfusion ratio is low, but is not beneficial if blood is shunted in the lungs. Shunting occurs when there is no ventilation in a perfused segment of the lung.

Hypotension

- This is more common when anaesthesia is maintained with inhalational agents. The incidence and severity of hypotension is much less when injectable anaesthetics are used. There is a strong correlation between intraoperative hypotension and the incidence of ischaemic myopathy, and so, to reduce the likelihood of myopathy, mean arterial pressure in adult horses should be greater than 70 mmHg.
- Treatment for hypotension includes:
  Checking that the horse is not too deeply anaesthetized.
  Checking that the horse is not volume deplete. If so, administer a bolus of a balanced electrolyte solution rapidly (e.g. 20 mL/kg of lactated Ringer's solution).
  Administration of an inotrope, because drug-induced myocardial depression is usually the primary cause of hypotension. *Dobutamine*, a cardio-selective inotrope, is the drug of choice. It is administered to effect (e.g. 0.25–5 µg/kg/min).

Hypercapnia

- Hypercapnia is a common occurrence in anaesthetized horses due to the depressant effects of anaesthetic drugs on respiratory function. A $PaCO_2$ >45 mmHg is generally regarded as hypercapnia, but some anaesthetists regard a $PaCO_2$ up to 70 mmHg to be tolerable. Long-term hypercapnia of this magnitude results in a significant decrease in pH.
- Treatment, if indicated, includes:
  Ensuring that the horse is not too deeply anaesthetized.
  Instituting controlled ventilation.

Postoperative complications

Serious complications, such as a long bone fracture, occur occasionally during recovery. Other, generally less serious problems include *myopathy* and *neuropathy*.

Myopathy (see Chapter 21)

Signs are generally noticed when the horse attempts to stand, but mild myopathy may not be evident until the horse attempts to walk. The condition arises due to muscle hypoxia resulting from ischaemic damage during recumbency. Ischaemia is more likely to develop in the dependent muscles of large horses anaesthetized for prolonged periods. Ischaemia is also more likely to occur if the horse is on a hard surface and if it is hypotensive. Signs include:

- Inability to bear full weight on one or more limbs,
- Localized swelling of muscles (e.g. the gluteals),
- Myoglobinuria, in severe cases.

Treatment includes assisting with recovery, maintaining hydration, and providing analgesia.

Neuropathy

- Most commonly, peripheral nerves (e.g. the radial nerve) are involved, but on rare occasions, the spinal cord may be injured. Spinal cord injury is thought to be ischaemic in origin and is most commonly reported in young horses of heavy breeds.
- Contributing factors for peripheral nerve damage are somewhat similar to those involved in myopathy and include: large body mass, poor positioning of the horse, prolonged recumbency, and inadequate padding.
- Signs vary with the nerve involved. In the case of radial nerve damage, the horse has difficulty standing and bearing weight on the affected limb. The elbow is 'dropped', and the horse is unable to extend the affected limb. Most horses recover fully within a few days, but supportive care may be necessary until the horse recovers.

FURTHER READING

Doherty T, Valverde A (2006) Manual of equine anesthesia and analgesia. Blackwell Publishing, Oxford, UK

Grosenbaugh DA, Muir WW (1998) Cardiorespiratory effects of sevoflurane, isoflurane, and halothane anesthesia in horses. American Journal of Veterinary Research 59:101–106

Johnston GM, Eastman JK, Wood JNI (2002) The confidential enquiry into perioperative equine fatalities (CEPEF): mortality results in phases 1 and 2. Veterinary Anaesthesia and Analgesia 29:159–170

Young SS, Taylor PM (1993) Factors influencing the outcome of equine anaesthesia: a review of 1314 cases. Equine Veterinary Journal 25:147–152

Chapter 25

Equine diagnostic imaging

Renate Weller, Charlotte Sinclair

CONTENTS

25.1 Radiography 499
Introduction 499
Underlying physics 499
Indications for radiography in the horse 502
Interpretation of radiographs 502
Radiographic changes associated with musculoskeletal problems 509
Advantages/disadvantages of radiography 516
Radiography and radiographic anatomy 516
Radiography of the different anatomical regions 519
25.2 Ultrasonography 552
Introduction 552
Indications in the horse 552
Advantages/disadvantages of ultrasonography 554
25.3 Scintigraphy 554
Introduction 554
Indications in the horse 554
Advantages/disadvantages of scintigraphy 556
25.4 Computed tomography (CT) and magnetic resonance imaging (MRI) 556
Computed tomography (CT) 556
Magnetic resonance imaging (MRI) 558
Further reading 559

Equine diagnostic imaging has developed rapidly over the last few years and the following modalities are routinely used in equine medicine:

- Radiography.
- Ultrasonography.
- Scintigraphy.
- Magnetic resonance imaging (MRI).
- Computed tomography (CT).

25.1 RADIOGRAPHY

Introduction

Radiography is the oldest and still the most commonly used imaging modality in equine practice.

Underlying physics

How are X-rays produced?

- X-rays are electromagnetic waves from the high-energy end of the electromagnetic spectrum (visible light or radiowaves are part of the same spectrum, but of lower energy and different wave length).
- X-rays are produced when fast moving electrons collide with matter. This happens in an X-ray tube that houses a cathode and an anode. By heating up the cathode, a cloud of electrons (negatively charged particles) is produced, which are accelerated till they hit the positive anode at high speed.
- The higher the temperature of the cathode the more electrons are produced; this is related to the mAs (milliAmpere seconds) settings of the X-ray machine.
- The higher the speed of the electrons the higher the penetrating power of the resulting X-rays. This is controlled by the kV settings of the X-ray machine.
- X-rays radiate from the source in straight lines in all directions. For medical purposes only a small cone of the X-rays, the primary beam, is used.
- The size of the primary beam is set by adjusting the window through which X-rays can leave their housing. This is called

© 2013 Elsevier Ltd
DOI: 10.1016/B978-0-7020-2801-4.00025-0

collimation and is an essential radiation protection mechanism, but also optimizes image quality by reducing scatter.
- The intensity of the X-ray beam is inversely proportional to the square of the distance from the source. This is obviously important for radiation safety consideration and has to be taken into account when adjusting exposure settings:

$$\frac{I_1}{I_2} = \left(\frac{r_2}{r_1}\right)^2$$

where I = intensity, and r = distance.

How do X-rays interact with matter?

- When X-rays hit matter, they can either penetrate the material or get absorbed by it. The main underlying principles on an atomic level are called the Compton and the Photoelectric effect. The Compton effect is less desirable because it is responsible for scatter radiation that degrades image quality.
- The degree of absorption is determined by the thickness and the radiodensity of the absorber.
- The radiodensity depends on the physical density and the atomic number of the material, e.g. lead has a very high atomic number which allows complete absorption of X-rays with only a few millimetres of material. This is the reason why lead is used for shielding purposes, e.g. in protective clothing. The same effect can be achieved with material of lower atomic number, by increasing its thickness, e.g. a 20 cm brick wall.
- The body is composed of materials of different radiodensities and thickness, hence X-rays are absorbed differentially. For example bone has a higher radiodensity and hence absorbs more X-rays than does soft tissue. This provides the basis for the image contrast that allows differentiation between structures on radiographs.

How are X-rays registered and how is that transformed into an image?

- While the way X-rays are generated has not changed much, the way they are detected has undergone considerable changes in the last few years.
- Conventionally, X-rays are detected using photographic film in combination with intensifying screens. After exposure the film needs developing in a similar process to film-based photography, to produce the final radiograph. More and more practices, however, now use computerized radiography (CR) or digital radiography (DR).
- CR systems still require the use of cassettes and a processor. A phosphor-coated plate in the cassette absorbs X-rays and stores them as energy. The stored energy is released as visible light after stimulation of the atoms on the phosphor plate with a laser beam in the processor. The light is registered and converted into a digital signal. After erasing the imaging plate, it can then be re-used.
- In DR systems the image is displayed directly on a screen without the necessity of processing a plate.

What image parameters characterize a radiograph?

- Image resolution quantifies how close structures can be to each other and still be visibly resolved and provides a measure of detail. In radiography it is usually expressed as line pairs/mm.
- Image contrast is the difference in radiodensity that makes an object distinguishable from another structure.
- Exposure latitude is the extent to which a radiograph can be over- or underexposed and still achieve an acceptable result.
- Decreasing the kV settings increases the image contrast, but decreases the latitude. For each 10 kV change the mAs setting has to be doubled or halved to maintain the same opacity. A kV setting under 70 is desirable for good bone radiographs.

Figure 25.1 Example of infectious arthritis and osetomyelitis. Lateromedial (left) and dorsopalmar (right) radiograph of a fetlock showing severe soft-tissue swelling around the affected area, aggressive periarticular new bone production and bone lysis characterized by irregular, poorly defined lytic areas.

- Image sharpness describes how well the edges of a structure can be distinguished from other structures or the background. It is influenced by the.
  - size of the focal spot: the smaller the sharper.
  - focus–film distance (FFD): the bigger the sharper.
  - distance between the object and the X-ray plate: the smaller the sharper.
  - movement blur.
  - poor screen–film contact in conventional radiography.

What can radiographs show?

- Radiographs can show changes in tissue density, shape, size, outline and position of structures.
- Radiographs in the horse are primarily used to assess bones, but can also provide information about soft tissues.

When do we see changes in bone on radiographs?

- Bone is a dynamic tissue and undergoes constant changes in response to the stress it is put under (Wolff's law). This results in changes in bone density and bone shape, which is a physiological process but changes with pathology.
- A 30–50% change in mineralization is required before it can be visualized on radiographs.
- Compared to other species (e.g. humans and dogs) the horse has a very slow bone metabolism, hence radiographic changes are slow to develop.
- Once radiographic abnormalities have developed they can persist for a long time without being significant.

How does an increase in bone production appear on radiographs?

New bone production appears as opacity on radiographs and can be classified according to the location:

- Periosteal new bone: often caused by trauma, but can also be caused by infection (Figures 25.1, 25.2, 25.3).
- Endosteal new bone: most commonly associated with trauma, e.g. fractures, but can also be caused by infection or inflammation.
- Cortical thickening: in response to stress.
- Callus formation: fracture repair (Figure 25.3).
- Enthesiophytes: focal, distinct new bone formation at attachment site of ligaments, tendons and joint capsule, usually associated with chronic strain at this site (Figures 25.4, 25.5, 25.6).
- Osteophytes: periarticular new bone usually associated with osteoarthritis (Figures 25.4, 25.6, 25.7).
- Sclerosis is a term used for localized new bone formation, usually in response to stress (e.g. subchondral bone sclerosis in osteoarthritis) or when the body is walling off areas, e.g. a sequestrum or bone cyst) (Figures 25.1, 25.7, 25.8, 25.9).

How does a decrease in bone production appear on radiographs?

Bone resorption results in lysis of bone and appears radiographically as radiolucency. This is most commonly focal in the horse, but can also be diffuse.

- Focal lucencies:
  - Changes in bone contour, e.g. flattening of trochlear ridges in cases of osteochrondrosis (Figures 25.10, 25.11).
  - Well-defined lucencies within bone, e.g. osseous-cyst-like lesions (Figure 25.12).
  - Subchondral bone lucencies, e.g. in osteoarthritis (Figures 25.7, 25.9).
- Diffuse lesions:
  - Diffuse bone resorption affecting whole bones is seen in association with disuse osteopenia.
  - This is often best seen at the proximal sesamoid bones which have a honeycomb appearance in affected horses.

Figure 25.2 Example of a sequestrum DMPLO radiograph of the metatarsus. There is a bony opacity (sequestrum) surrounded by a lucent halo with a rim of opaque bone (involucrum) on the plantaromedial aspect of the third metatarsal bone (black arrows) and periosteal new bone extending distal from it (white arrows). There are gas shadows in the soft-tissue swelling overlying it.

What can radiographs tell us about soft tissues?

- Radiographs are not very sensitive when it comes to the assessment of different soft-tissue densities: e.g. fluids such as blood or urine have the same radiographic appearance as most soft tissues (tendons, cartilage etc.).
- The exception is fat, which appears more radiolucent than other soft tissues, which can, for example, be appreciated in the case of the triangular radiolucency consistent with the patellar fat pad in the stifle (Figures 25.11, 25.12).

- Abdominal radiography.
  - The sheer size of an adult horse makes it impossible to get detailed radiographs of the abdomen. One exception where abdominal radiographs may be useful is to visualize the presence of sand or enteroliths in the gut.
  - Abdominal radiographs in foals are commonly performed to evaluate the gastrointestinal tract and render diagnostic results similar to small animals.
- Thoracic radiography.
  - Can be performed only with high-output X-ray machine and a grid for scatter reduction, hence is usually only done in hospital settings.
  - It is not very sensitive for most thoracic disease and its usefulness should be considered very carefully in each case especially since it involves high radiation exposures!

Indications for radiography in the horse

Radiographs are indicated to:

- Confirm a clinically suspected diagnosis.
- Assess the severity of a disease.
- Exclude other pathological conditions.
- Assist in surgery planning.
- Monitor the progress of disease.

No imaging technique can replace the clinical examination! Radiographic findings do not indicate pain!

The clinical findings need to guide radiography, e.g. a wound, swelling or positive regional analgesia needs to point to an area.

Using imaging as a 'fishing exercise' is not a good idea because most changes observed on images may or may not be of clinical significance and their clinical meaning can be appreciated only in conjunction with the clinical findings.

An exception is prepurchase and presales radiographs, where an attempt is made to use radiography as a predictor for future soundness.

Interpretation of radiographs

Pitfalls in radiographic interpretation

- Over-interpretation: detecting pathology where none is present:
  - Normal anatomical variation is mistaken for pathology.
  - Artefacts that mimic lesions are mistaken for pathology.
- Under-interpretation: missing lesions.
  - Detection of obvious lesions distracts from other more subtle lesions.
  - Pathological changes are mistaken for normal anatomical variation or artefact.
  - Lesions are obscured due to poor image quality.
- Mis-interpretation:
  - Recognizing radiographic changes as abnormal but coming to the wrong radiographic diagnosis; this is often linked to clinical bias: matching a radiographic lesion to the clinical picture.

Figure 25.3 Sagittal P1 fracture. These dorsopalmar radiographs show an articular, complete, non-displaced, closed fracture of the proximal phalanx on day 0 (left image) and six weeks later (right image). The well demarcate fracture lines from day 0 (black arrows) are filled with callus six weeks later (white arrows). There is also peristeal new bone where the fracture line exits the cortex.

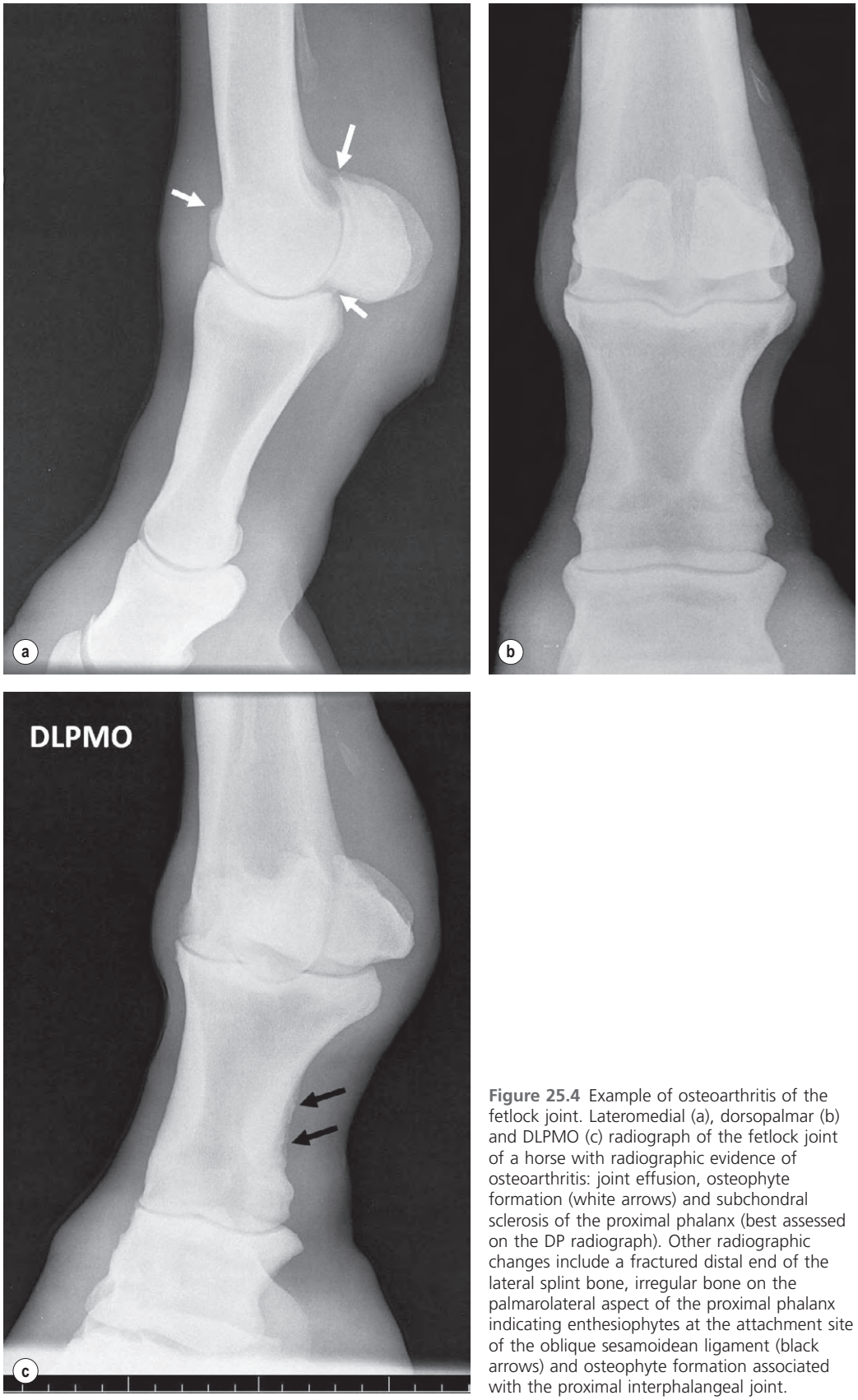


Figure 25.4 Example of osteoarthritis of the fetlock joint. Lateromedial (a), dorsopalmar (b) and DLPMO (c) radiograph of the fetlock joint of a horse with radiographic evidence of osteoarthritis: joint effusion, osteophyte formation (white arrows) and subchondral sclerosis of the proximal phalanx (best assessed on the DP radiograph). Other radiographic changes include a fractured distal end of the lateral splint bone, irregular bone on the palmarolateral aspect of the proximal phalanx indicating enthesiophytes at the attachment site of the oblique sesamoidean ligament (black arrows) and osteophyte formation associated with the proximal interphalangeal joint.

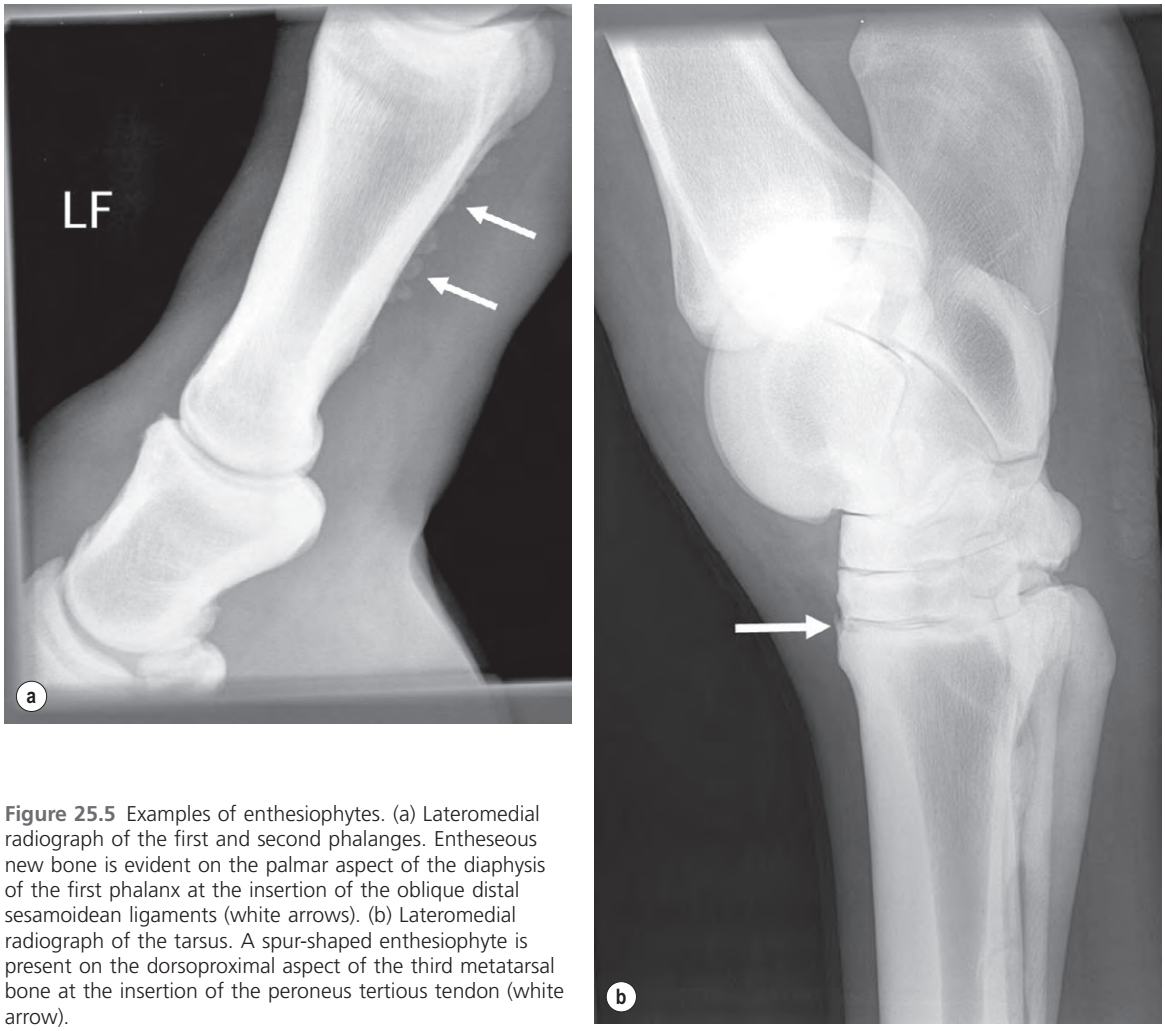


Figure 25.5 Examples of enthesiophytes. (a) Lateromedial radiograph of the first and second phalanges. Entheseous new bone is evident on the palmar aspect of the diaphysis of the first phalanx at the insertion of the oblique distal sesamoidean ligaments (white arrows). (b) Lateromedial radiograph of the tarsus. A spur-shaped enthesiophyte is present on the dorsoproximal aspect of the third metatarsal bone at the insertion of the peroneus tertious tendon (white arrow).

Figure 25.6 Lateromedial radiograph of the foot with radiographic evidence of laminitis. The dorsal hoof wall and the dorsal surface of the distal phalanx are not parallel, indicating rotation of the distal phalanx away from the hoof capsule. There is also new bone formation on the attachment site of the oblique sesamoidean ligament and osteophyte formation associated with the proximal interphalangeal joint.

How to view radiographs?

- Radiographs should be displayed on a viewing box in the case of films and a hot light should be available to highlight areas. Holding radiographs against the sun is not acceptable!
- In the case of digital images, a high-resolution screen should be used and one should ensure that the image format does not involve compression, and hence loss of quality.

How to assess radiographs?

A standard approach should incorporate the assessment of the following parameters:

- Assessment of diagnostic quality. The first step in interpreting radiographs is to decide whether or not the radiographs are of diagnostic quality. The following parameters should be evaluated:
  - Labelling: case number or name, date of examination, left–right, front–hind (necessary for projections distal to the carpus/tarsus), projections (including direction and degree of obliquity).
  - Positioning/centring: is the area of interest on the radiograph?
  - Sufficient number of views, additional projections required?
  - Artefacts: movement, processing artefacts etc.

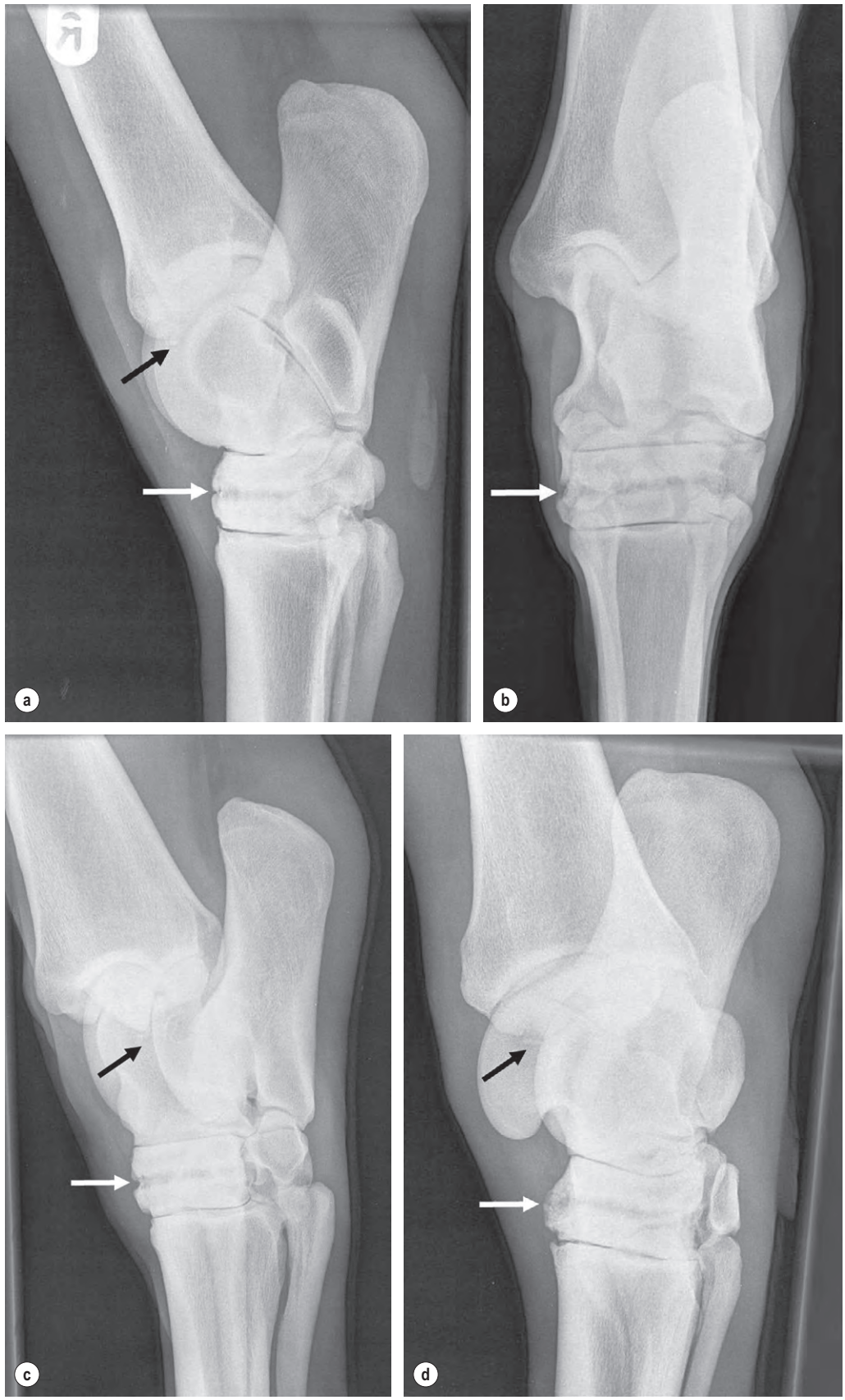


Figure 25.7 Example of bone spavin and OCD. Lateromedial (a), dorsopalmar (b), DLPMO (c) and DMPLO (d) radiographs of the tarsus of a horse with radiographic evidence of severe osteoarthritis of the distal intertarsal joint (white arrow) and moderate osteoarthritis of the tarsometatarsal joint, often referred to as bone spavin: irregular obliteration of the joint space with subchondral bone resorption, loss of definition between cortex and medulla of central and third tarsal bone indicating bone sclerosis, mottled new bone formation on the dorsal aspect of the joint and a spike-shaped osteophyte on the dorsal end of the third metatarsus. There is also an oval shaped, loose bony opacity distal to the intermediate ridge of the tibia and evidence of joint effusion of the tarsocrural joint, indicating OCD (black arrow).

Figure 25.8 Olecranon fracture. This mediolateral radiograph of an elbow shows a comminuted, articular fracture of the olecranon.

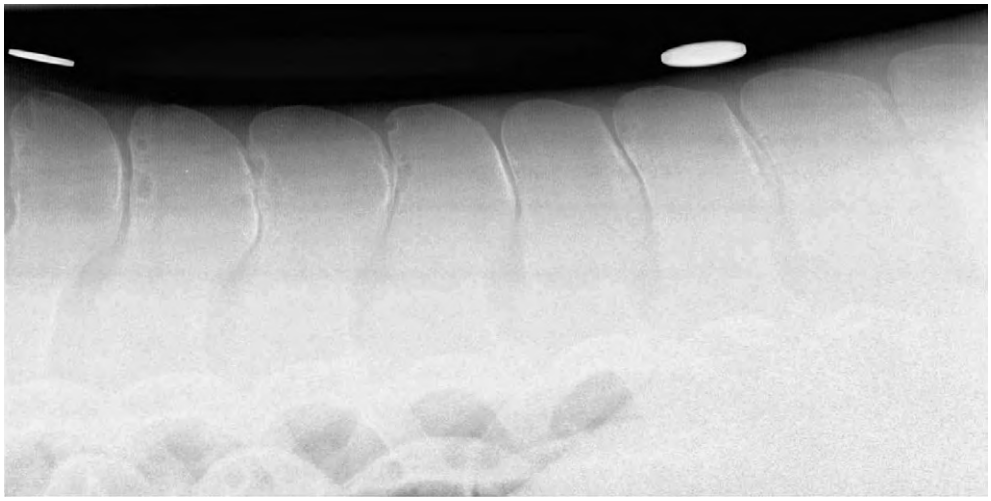


Figure 25.9 'Kissing spines': the lateral radiographs of the spinous process show narrowing of the interspinous space with sclerosis and lucencies in the underlying bone. Note the metal markers placed to help matching up radiographs with the live horse.

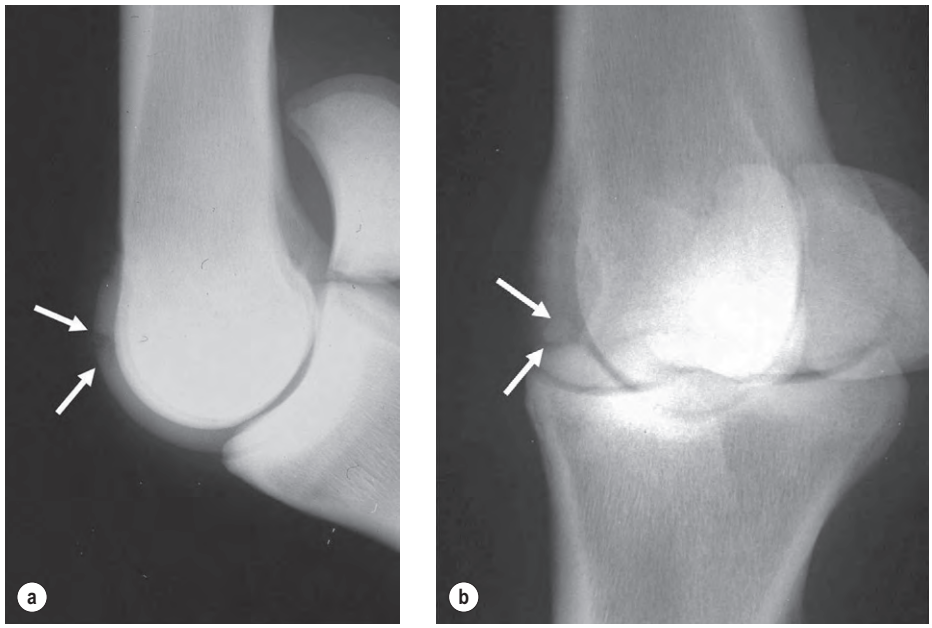


Figure 25.10 Two examples of fetlock joint osteochondrosis. (a) Flexed lateromedial radiograph of fetlock with a defect in the dorsoproximal aspect of the sagittal ridge indicating OC, (b) oblique radiograph of a fetlock with an oval shaped bony opacity on the dorsal aspect of the fetlock joint indicating OCD.

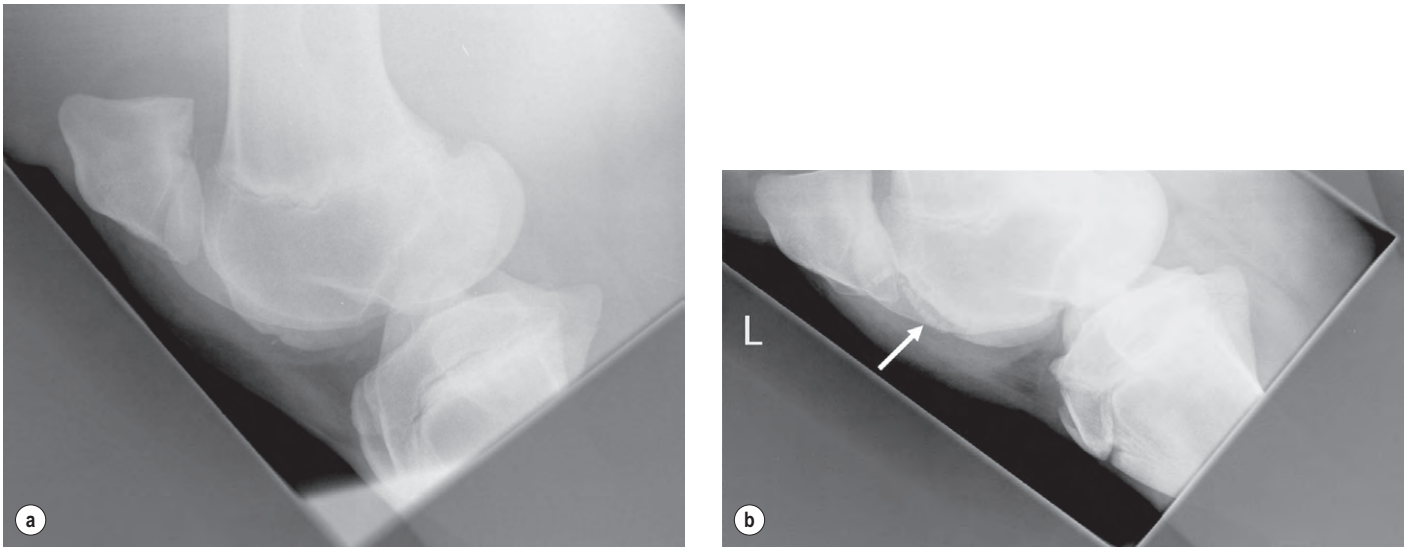


Figure 25.11 Two examples of stifle osteochrondrosis. (a) Lateromedial radiographs of an equine stifle: the lateral trochlear ridge has lost its regular, rounded appearance and shows an indentation with underlying subchondral bone sclerosis, indicating OC. (b) Multiple mineral opacities are evident at the lateral trochlear ridge, indicating OCD (white arrow). Note the open growth plates identify these animals as skeletally immature.

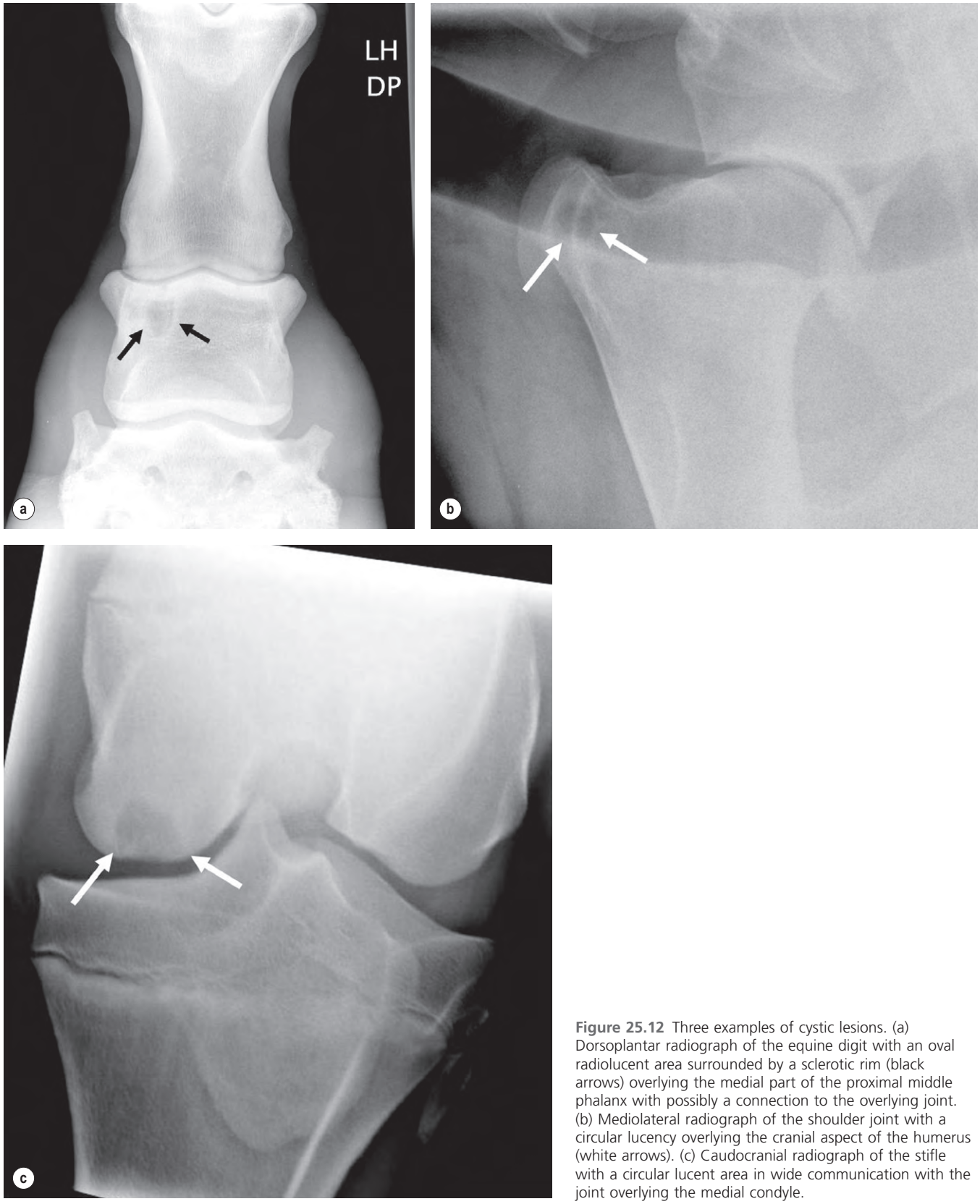


Figure 25.12 Three examples of cystic lesions. (a) Dorsoplantar radiograph of the equine digit with an oval radiolucent area surrounded by a sclerotic rim (black arrows) overlying the medial part of the proximal middle phalanx with possibly a connection to the overlying joint. (b) Mediolateral radiograph of the shoulder joint with a circular lucency overlying the cranial aspect of the humerus (white arrows). (c) Caudocranial radiograph of the stifle with a circular lucent area in wide communication with the joint overlying the medial condyle.

Table 25.1 Physeal fusion in the horse

		Months
Humerus	Proximal epiphysis	18–30
	Distal epiphysis	15–21
	Medial epicondyle to condyles	6–12
	Lateral tuberosity	24–36
Radius	Proximal epiphysis	15–21
	Distal epiphysis	24–30
Ulna	Proximal epiphysis	24–36
	Distal epiphysis	6–9
Metacarpus (distal)		8–12
Proximal phalanx (proximal)		6–9
Distal phalanx (proximal)		6–9
Femur distal epiphysis		24–30
Tibia	Proximal epiphysis	25–30
	Tibial tuberosity to epiphysis	9–12
	Tibial tuberosity to shaft	30–36
	Distal epiphysis	18–24
Lateral malleolus of distal tibia		6
Os calcis		24–30

Table 25.2 An example of a radiographic report

Radiology report form

1. DESCRIPTION – SPECIES, AGE, TYPE.
   Skeletally immature horse.
2. ANATOMICAL AREA AND PROJECTIONS.
   Right stifle, lateromedial projection.
3. QUALITY – INCLUDING FILM FAULTS.
4. RADIOLOGICAL DESCRIPTION.
   There are two bony opacities, oblong, 5 × 3 mm, clearly demarcated, smoothly outlined on the cranial aspect of the lateral trochlear ridge of the femur just cranial to a step formation in the outline of the bone. The bone underlying the disruption appears sclerotic.
5. CONCLUSION AND RADIOLOGICAL DIAGNOSIS(ES).
   Osteochondrosis dissecans.
6. FURTHER INVESTIGATIONS REQUIRED.
   Radiograph other contralateral stifle.

  - Exposure: this is less crucial when using CR or DR systems compared to conventional film-screen combinations because these systems allow adjustment of contrast and brightness, however only within a certain range and a bad radiograph cannot be turned into a good radiograph after it has been taken!
  - Collimation: this is of concern regarding radiation safety but also influences the amount of scatter produced and hence influences image quality.
- Identification of subject: species, age (skeletally mature/immature). Table 25.1 shows the radiographic closure times for growth plates in the horse.
- Identification of lesions.
  - Develop a systematic approach!
  - There are two basic approaches to evaluating radiographs: systems-based and 'geographical'. A systems-based approach is most appropriate in complex areas such as the thorax and abdomen. Typically, the radiologist works through a checklist for each organ system, such as respiratory, cardiovascular, gastrointestinal, lymphoreticular and musculoskeletal, for the thorax, and scrutinizes the anatomical locations of each part of each system to avoid missing an abnormality. For simpler situations, such as a joint, a 'geographical' approach is more commonly used.
  - Most people have the tendency to focus on the centre of an image thereby running the risk of missing lesions in the periphery. This can be avoided by systematically evaluating a radiograph e.g. from the periphery to the centre and from proximal to distal.
  - It does not really matter what approach or what order is adopted; however, it is **helpful to stick with the same approach every time!**
- Description of a lesion by its Röntgen signs.
  - Radiodensity: lucency versus opacity (opacities can be further categorized into soft tissue, mineral (often bone), metal density).
  - Size: in SI units.
  - Shape (spherical, elliptical, cylindrical, etc.).
  - Number (single, multiple).
  - Margination (discrete, well/ill-defined or demarcated etc.).
  - Location: use correct anatomical nomenclature!
- Conclusion. Lesions should be interpreted first on their radiographic appearance only and only then secondly in the light of the clinical history. It is usually helpful to think about what process can lead to the observed radiographic changes. There are several possibilities which should be listed as differential diagnoses; however, one should always try to come up with the most likely diagnosis in the end taking all the information into account.
- Diagnosis. Combine the radiographic interpretation with existing clinical data and come up with (in your opinion) the most likely diagnosis.
- Recommendations for further investigations. Other imaging techniques (ultrasound, MRI, CT, scintigraphy), tests (e.g. blood work) or surgery.

Table 25.2 shows an example of a radiographic report form.

Radiographic changes associated with musculoskeletal problems

See Chapters 15 to 18.

Osteoarthritis (Figures 25.4, 25.6, 25.7)

- Radiographic changes associated with osteoarthritis are:
  - Joint effusion: soft-tissue swelling around the affected joints.
  - Periarticular osteophytes: new bone formation at the articular margins.
  - Subchondral bone sclerosis: increase in bone opacity just below or above the joint space. In some bones this results in a loss of the differentiation between cortex and medulla, e.g. the third or central tarsal bone.

  - Narrowing of the joint space. This can progress to partial or complete obliteration of the joint space. Care has to be taken that this is not a projection artefact, comparing medial and lateral sides on DP or CrCd views, comparing left and right or repeating the projection helps to decide.
  - Subchondral bone resorption: diffuse or focal lucencies. The exact location, extent and combination of these radiographic signs vary between joints and severity of disease.
- High-motion joints differ considerably from low-motion joints in the radiographic appearance of osteoarthritis, e.g. subchondral bone lucencies are much more commonly seen in low-motion joints, such as the tarsometatarsal joint, distal intertarsal joint or proximal interphalangeal joint (Figures 25.6, 25.7).
- Osteoarthritis is often secondary to another disease (e.g. osteochondrosis) and care should be taken to evaluate the radiographs for any signs of primary disease (Figures 25.4, 25.7).
- There is a poor correlation between radiographic signs and lameness. Remember that: the absence of radiographic changes does not rule out osteoarthritis and the presence of radiographic signs does not indicate pain!

Osteochondrosis (Figures 25.10, 25.11)

The fetlock, tarsocrural, femoropatellar and shoulder joints are the most commonly affected joints.

- Radiographic changes associated with osteochondrosis:
  - Changes in contour of bone surface (Figures 25.10a, 25.11a), most commonly flattening or depression, e.g. trochlear ridges of the talus or femur, humeral head.
  - Subchondral bone sclerosis: on its own or in association with any of the other signs (Figure 25.11a,b).
  - Subchondral bone lucency: focal or along the joint surface.
  - Fragments: solitary or multiple (Figures 25.7, 25.10b, 25.11b).
- There is a poor correlation between radiographic signs and lameness. It has been suggested that subchondral bone changes are more likely to be of clinical significance than contour changes alone.
- Osteochondrosis is a developmental problem and may affect more than one joint.
- Osteochondrosis may cause osteoarthritis, especially when osteochondral fragments become dislodged (e.g. through trauma) and irritate the joint, hence radiographs should be thoroughly evaluated for signs of secondary osteoarthritis.
- Osseous cyst-like lesions and subchondral bone cysts (Figure 25.12).
- Radiographic changes associated with cystic lesions:
  - Well-defined (semi) circular or oval shaped lucency.
  - Often surrounded by a sclerotic ring.
  - Most commonly seen in the subchondral bone; however, may also be seen away from the joint.
  - A neck-like connection between the cyst and the joint surface may be present.
  - Can have more than one compartment.
- Predilection sites are the medial condyle of the femur and the glenoid cavity of the scapula, but can be seen in any bone.
- Clinically often insignificant. Lesions with a connection to the joint surface are thought to be more likely associated with lameness.

Infectious osteitis and osteoarthritis (Figure 25.1)

- Infection can either result in new bone formation or in bone resorption, and often a combination of the two, and is commonly associated with soft-tissue swelling.
- Radiographic changes associated with infection are usually much more pronounced compared to degenerative problems:
  - Severe soft-tissue swelling around the affected area.
  - Aggressive periarticular new bone production.
  - Bone lysis characterized by irregular, poorly defined lytic areas.
- A sequestrum (Figure 25.2) is a form of localized osteitis, where a bit of bone dies and is visible as a bony opacity (sequestrum) surrounded by a lucent halo with a sclerotic rim of bone (involucrum). The involucrum may be itself surrounded by sclerotic bone, where the body tries to wall off the infected area. In some cases the presence of a discharging sinus tract from the infected area to the outside may be visible as disruption of the overlying soft tissues.

Enthesiopathies (Figures 25.5, 25.6)

- Enthesiopathy is a term used for new bone formation at the attachment sites of tendons, ligaments and joint capsules.
- Enthesiophytes represent the reaction of bone to the strain it is put under by the attaching soft-tissue structure.
- Enthesiophytes are slow to develop and they are often of no clinical significance.
- In areas where soft-tissue attachments are close to joints it is often difficult to differentiate between osteophytes or enthesiophytes, e.g. on the dorsal aspect of the proximal metatarsus, where the attachment sites of the peroneus tertius and the tibialis cranialis are very close to the margin of the tarsometataral joint.
- Common sites are the distal attachment of the oblique distal sesamoidean ligament on the middle phalanx, the attachment of the intertarsal ligaments on the dorsal aspect of the tarsal bones, the origin of the nuchal ligament from the occipital bone.

(Sub)luxations (Figure 25.13)

- (Sub)luxation presents as a partial (subluxation) or complete (luxation) separation of joint surfaces.
- They are the result of fractures or ligament ruptures and the radiographs need to be evaluated for any evidence of the underlying problem.
- Stressed views, where either the medial or the lateral aspect of a joint is pulled apart, may be necessary to make some cases of subluxation radiographically apparent.
- Ultrasonography is indicated to assess ligament damage.

Fractures

- Fractures are radiographically seen as radiolucent lines.
- Radiographic description of fractures should answer the following questions:
  - Is the fracture complete? The fracture line will extend from one end of the bone to the other. A single plane, complete fracture will appear as two lucent lines where the fracture plane traverses the two cortices!
  - Is it a simple or comminuted fracture and if so how many separate fragments are there?
  - Is the fracture displaced? This will lead to a disruption of the continuity of the bone surface.
  - Does the fracture involve a joint? The fracture line will extend into one or two joints.
  - Is the fracture open? Communication with the outside and/or soft tissue disruption will be visible.
  - Are there any signs of secondary problems present?

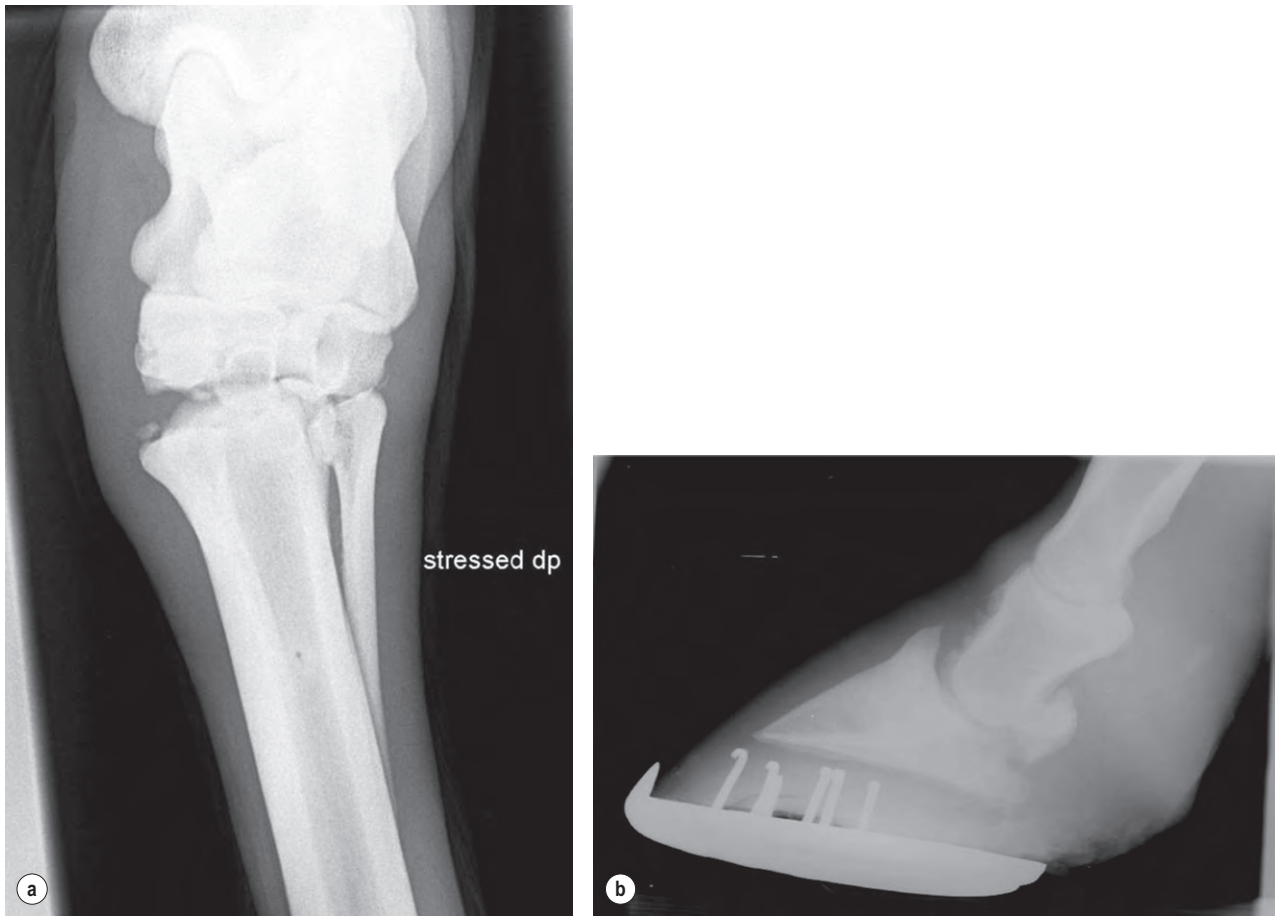


Figure 25.13 Examples of subluxations. (a) Stressed dorsoplantar view of the tarsus. The joint space is wide open medially indicating subluxation of the tarsometatarsal joint, several bony fragments are evident, likely due to avulsion of the medial collateral ligament, which should be assessed ultrasonographically. (b) Lateromedial radiograph of the foot. The joint space is wide open dorsally and there is extensive new bone formation around the joint margins, indicating chronic subluxation of the DIP joint.

- Terminology often used in association with fractures in the horse are:
  - Long bone fractures (Figure 25.14).
  - Condylar fractures (Figure 25.15).
  - Slab fractures: extend from one articular surface to the other, in carpal and tarsal bones (Figure 25.16).
  - Chip fractures: small fragments on articular margins (Figure 25.17).
  - Avulsion fractures: at the attachment site of a ligament, tendon or muscle.
- Fracture lines usually widen in the first 5–10 days after injury due to osteoclastic activity along the fracture line (Figure 25.18). This is also the reason why fractured bones are at their weakest at this time and care should be taken when moving horses at that stage.
- Pitfall in fracture identification:
  - Mach lines: these are radiolucent lines resulting from enhancement at the edge of two superimposed structures, e.g. the splint bones over the cannon bone (Figure 25.2).
  - Nutrient foramina: these are usually wider than fracture lines and in known locations (Figure 25.19).
  - Vascular channels in the distal phalanx (Figure 25.20).
  - Frog clefts, especially when they are inadequately packed: these usually extend beyond a single bone (Figure 25.21).

Figure 25.14 Example of a catastrophic long-bone fracture. Lateromedial radiograph of the stifle of skeletally immature horse. A displaced Salter-Harris type II fracture of the distal femur is evident. Note the wide open growth plates identify this animal as a foal.

Figure 25.15 Example of a condylar fracture. Dorsoplantar radiograph of the metatarsophalangeal joint. A non-displaced lateral condylar fracture with surrounding soft-tissue swelling is evident.

Figure 25.17 Example of a chip fracture. Dorsomedial-plantarolateral view of the metatarsophalangeal joint. A smoothly marginated oval, bony opacity is evident proximal to the medial plantar process representing a resorbing chip fracture (white arrow). There is also soft-tissue swelling on the dorsal aspect of the MTP joint probably representing joint effusion.

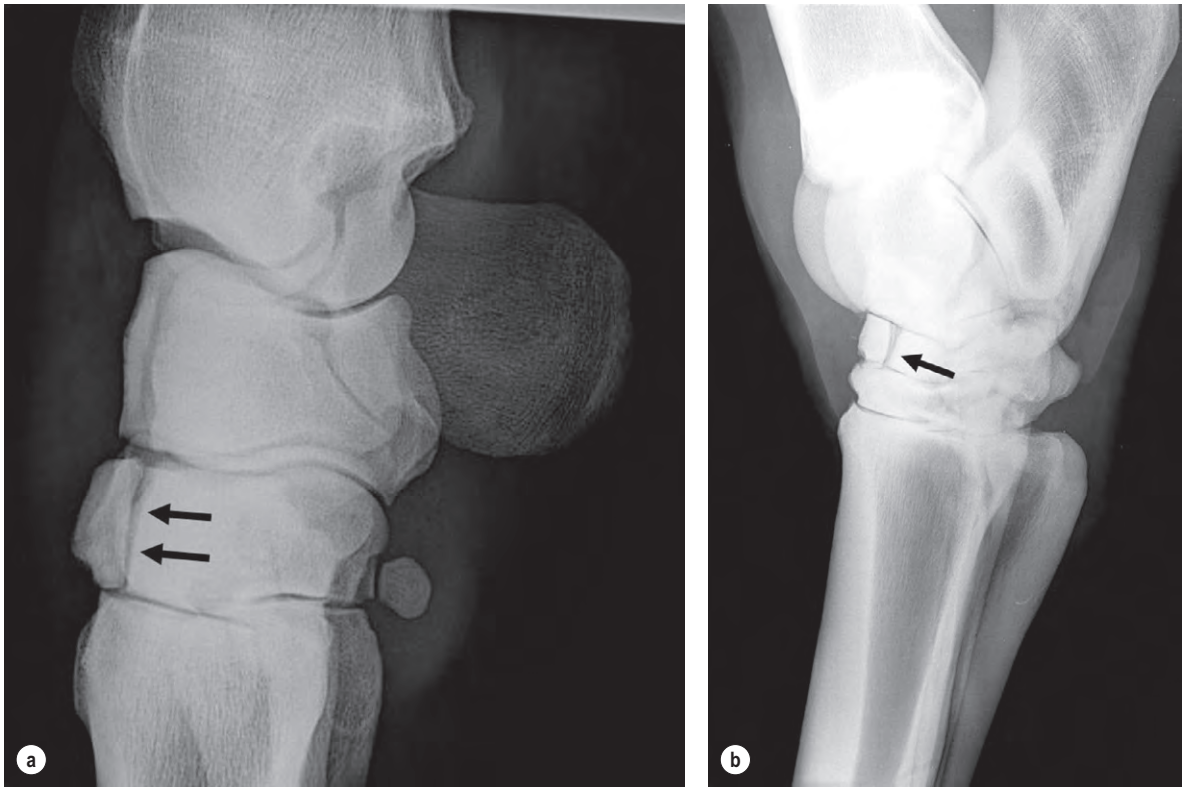
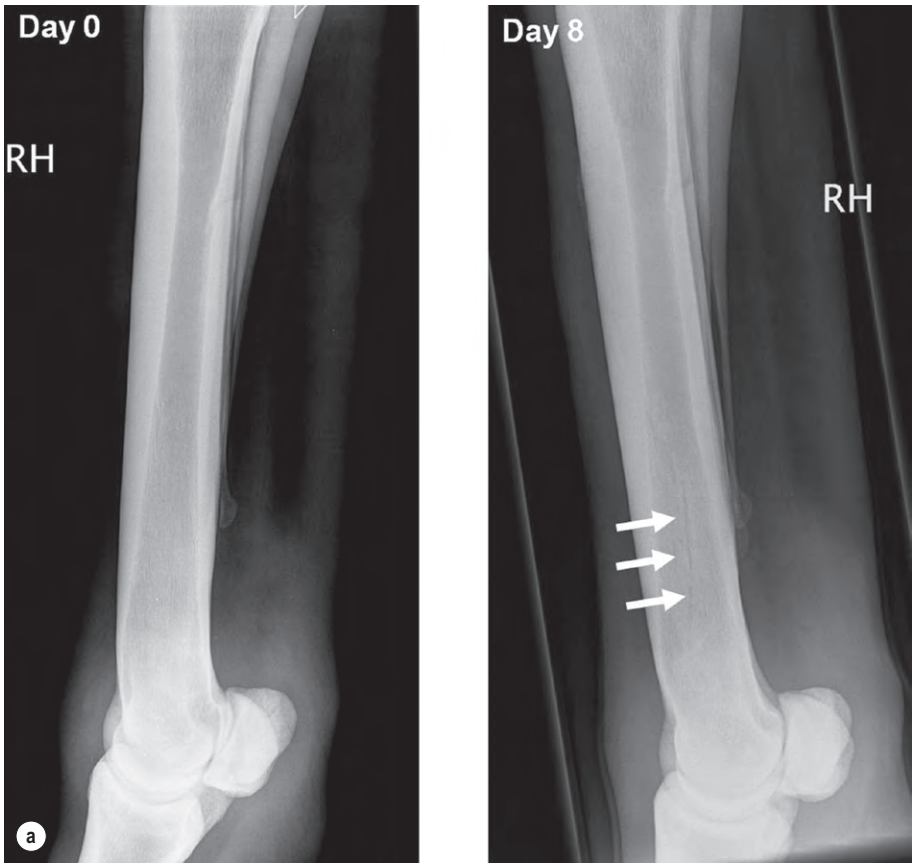


Figure 25.16 Example of slab fractures. Lateromedial radiograph of the (a) carpus and (b) tarsus. There is a slab fracture of the third carpal bone (black arrows) and a minimally displaced slab fracture of the central tarsal bone (black arrow) and soft-tissue swelling on the dorsal aspect of the tarsus (probably representing tarsocrural joint effusion).



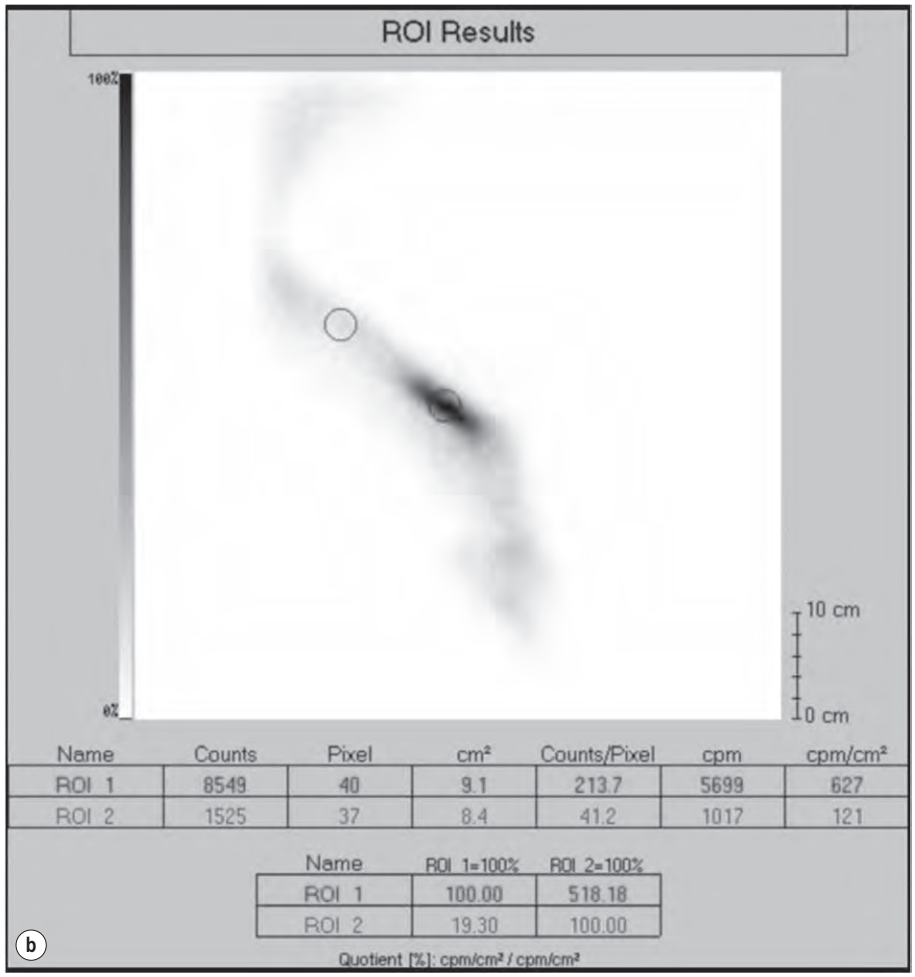


Figure 25.18 Hairline fracture of the third metatarsus. (a) On day 0 the fracture line is not visible radiographically; on day 9 a fracture line is visible (white arrows). (b) The scintigram acquired on day 0 shows a markedly increased uptake of radiopharmaceutical over the metatarsus, indicating a fracture. Two regions of interest were drawn, one over the increased uptake, one as a reference to compare the ratios of both with the other leg.

Figure 25.19 Nutrient foramen versus fracture. Dorso 10° lateral-palmaromedial oblique radiograph of the metacarpus. A thin, well demarcated lucent line crosses the body of the metacarpus representing a fracture of the third metacarpus (black arrow). Compare this to the much wider radiolucent line which represents the nutrient foramen (white arrow).

Figure 25.20 Fracture of the distal phalanx versus normal vascular channels. Dorsolateralproximal-plantaromedial distal 20° oblique radiograph highlighting the lateral plantar process ('wing') of the distal phalanx. There is an indistinct lucent line extending over the whole width of the bone into the DIP joint representing a complete, intraarticular fracture (black arrows). Compare this to the numerous radiating vascular channels normal for the distal phalanx.

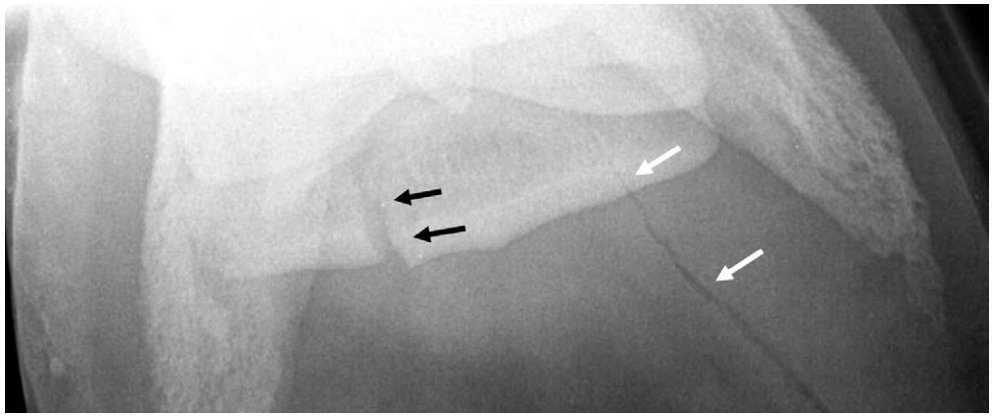


Figure 25.21 Navicular bone fracture versus packing artefact. Palmaroproximal-palmarodistal oblique of the navicular bone. There is a complete parasagittal fracture of the navicular bone with slight displacement (black arrows). The radiolucent line coursing the medial aspect of the navicular bone is an air artefact due to inadequate packing of the collateral sulci (white arrows). In most cases packing artefacts can be distinguished from pathological changes by the fact that they extend beyond boundaries of bones.

- Radiolucent lines between separate centres of ossification, especially fibula and hoof cartilages (Figure 25.22).
- Non-displaced fissure fractures may not be radiographically visible at first and scintigraphy is indicated in horses where a fracture is suspected, but cannot be visualized radiographically. Alternatively, the radiography can be repeated after 7–10 days (Figure 25.18).

Figure 25.22 Example for separate ossification centre. Caudocranial projection of the stifle. There is a radiolucent line coursing the fibula transversely which is due to the fibula having separate centres of ossification (white arrow).

- Assessment of fracture healing:
  - Callus: develops within 14–20 days of injury, often requires lower exposures for optimal visualization, fracture lines are less distinct and the fracture gap becomes narrowed and more radio-opaque.
  - Periosteal callus can be seen from 14 days on, and the degree of periosteal reaction reflects the stability of the fracture.
  - Fractures are often radiographically still visible while the fracture is already stable.
  - In some bones a radiolucent line may persist, e.g. the accessory carpal bone or the proximal and distal sesamoid bones.
  - A delayed union is present if a fracture line persists longer than 6 months.
- Soft-tissue calcification.
  - Soft-tissue calcification in the horse is usually secondary to soft-tissue injury causing haemorrhage and/or inflammation, e.g. suspensory ligament injuries.
  - Because there is a lag between the original soft-tissue injury and the appearance of radiographically visible changes, these are often not associated with clinical signs.
- Wounds.
  - While plain radiographs are not very good in differentiating between soft-tissue densities, contrast radiographs are very useful in the evaluation of soft-tissue injuries because they can show the extent of the wound and the involvement of synovial structures.
  - This involves the use of positive-contrast media, which appear white on radiographs and are hence easily differentiated from the surrounding soft tissues.
  - A metal probe can first be inserted to assess the direction and depth of the wound. Two radiographs at 90° to each other are necessary to allow the visualization of the metal probe in 3D (Figure 25.23).
  - If the wound comes close to a synovial cavity (joint, tendon sheath, bursa) a positive-contrast solution is injected into the

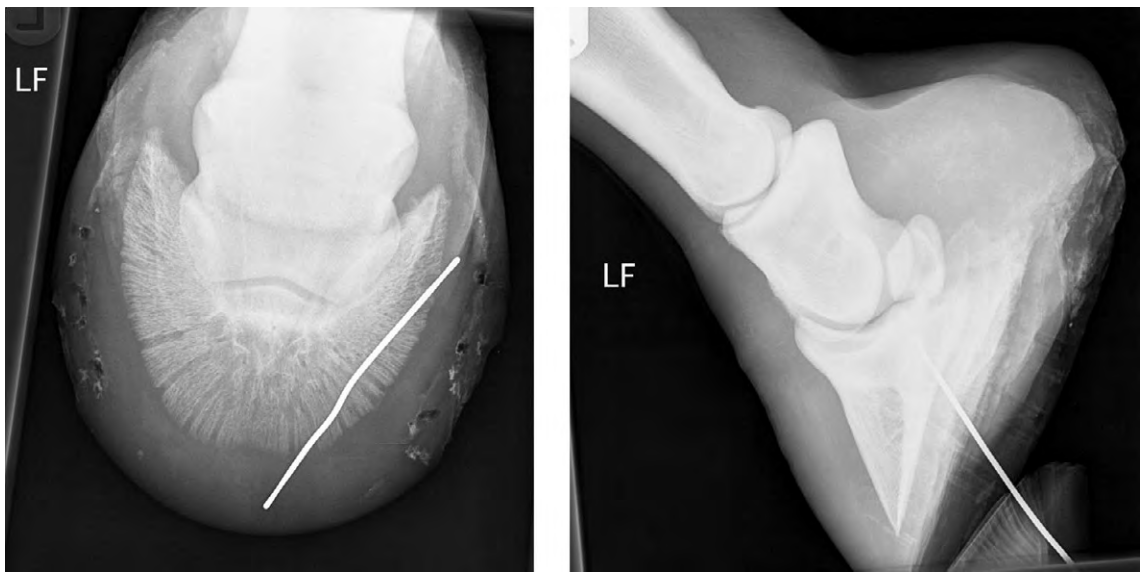


Figure 25.23 Radiographic evaluation of a penetrating wound. Dorsoproximal-palmarodistal 60° oblique (left), and a lateromedial (right) radiograph of the foot after inserting a metal probe into the tract. The two radiographs demonstrate the value of obtaining orthogonal views to aid localization of the wound in 3D.

Figure 25.24 Use of contrast study for the radiographic evaluation of wounds. Lateromedial radiograph of the carpus after injection of positive contrast medium in the antebrachiocarpal joint. Contrast is filling the antebrachiocarpal joint and extending to the skin surface, demonstrating communication of the joint with a wound on the dorsal aspect of the carpus. Note the remnant of the distal ulna present in this horse caudal to the radius.

synovial cavity (NOT in the wound!) and the radiographs are repeated.
- Care has to be taken that the synovial injection is performed under aseptic conditions to prevent iatrogenic contamination!
- Contrast medium exiting from the wound indicates a connection between wound and synovial cavity (Figure 25.24).
- Contrast media for musculoskeletal assessment are iodine-based solutions. They can be categorized into ionic and non-ionic solutions. Ionic solutions are much cheaper, but have been associated with allergic reactions and local irritation in humans whereas non-ionic solutions, which are more expensive, are less irritating. Non-ionic contrast media have to be used for myelography, where contrast media are used to outline spinal cord compression in horses with neurological deficits.

Advantages/disadvantages of radiography

Advantages

- Cheap compared to other imaging modalities.
- Quick.
- Artefacts and normal variations are well known compared to newer imaging techniques.

Disadvantages

- Radiographs represent 2D image of 3D structure.
  - Superimposition of structures and summation of densities.
  - Loss of depth perception.
  - Two images necessary to locate lesion in space.
  - Distortion.
- Involve ionizing radiation.
- Not very sensitive: a 30–50% change in mineralization is required before it becomes radiographically apparent.
- Radiographic changes correlate badly with pain and clinical signs!

Radiography and radiographic anatomy

Patient preparation

Good patient preparation aims to:

- make the process quick and efficient.
- minimize risk to the personnel from the horse.
- minimize radiation exposure by avoiding repeats.
- prevent artefacts: movement, dirt etc.

This is achieved through:

- Patient restraint.
  - Depending on the temperament of the horse, the number and type of projections an appropriate method of restraint should be chosen.
  - Very cooperative horses may need no additional constraint.
  - For a very quick examination, a nose twitch may be sufficient; however, proper sedation is preferable.
  - The authors sedate the majority of horses for radiographic examination with a combination of detomidine or romifidine combined with butorphenol.
  - Some clinicians use stocks for radiography and have found this to help with patient restraint.
  - The use of a head stand is also very beneficial for head and neck radiographs, so are the use of blinkers and cotton wool ear plugs (Figure 25.25).
- Avoiding dirt artefacts: always assess the area which you wish to radiograph to ensure that the area is free from dirt or matted hair which will show up as artefacts on radiographs and can often cause confusion resulting in repeat exposures. Iodine-based scrubs should be avoided as they will act as a contrast medium.
- Positioning of horse: take time to position your horse correctly with the horse standing square and equally weight-bearing. A horse which is stands toed-in or out or is leaning forward or backwards will make correct alignment of the X-ray beam more difficult. This will result in non-diagnostic images due to superimposition of joint spaces.
- For head radiographs and cranial neck radiographs, a halter without metal buckles should be used to avoid superimposition of metal on bone (Figure 25.25).
- For feet radiographs, a little more work is required (see below).

Radiographic projections

Animal size, and the fact that the majority of radiographs are produced from the standing animal, have important implications for what is considered an adequate study in a large animal. As with small animals, the minimum number of projections required to evaluate any region is two. However, the size and density of equine bones means that it can be very difficult to recognize subtle changes. Such changes, whether productive or involving bone loss, are best viewed as 'skylined' objects, necessitating the use of oblique projections.

Figure 25.25 Methods to aid horse restraint: head stand, blinkers, cotton-wool ear plugs. A rope head collar is advisable for imaging of the head to avoid metal artefacts. This horse is positioned for scintigraphy of the head; however, the illustrated methods are helpful for any imaging procedure.

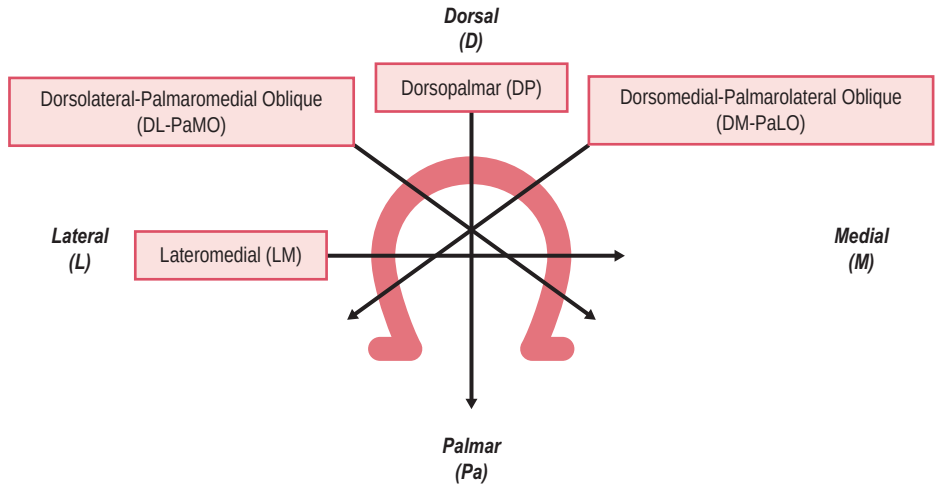


Figure 25.26 Radiographic projections are named by their point of entrance of the X-ray beam into a structure followed by their point of exit (Smallwood et al 1985). Standard anatomical terminology following the NAV should be used; however, many projections have additional names, which are used in everyday equine work.

- A 'standard set' of radiographs of an equine bone would be made up of four projections:
  - Lateromedial: highlights the dorsal and palmar aspects of the limb.
  - Dorsopalmar: highlights the lateral and medial aspects of the limb.
  - Dorsolateral-palmaromedial oblique (DLPMO) – highlights dorsomedial and palmarolateral structures.
  - Dorsomedial-palmarolateral oblique (DMPLO) – highlights dorsolateral and palmaromedial structures.
- Projections are named by their point of entrance of the X-ray beam into a structure followed by their point of exit (Figure 25.26). Standard anatomical terminology following the NAV should be used, however many projections have additional names, which are used in everyday equine work.

Table 25.3 Exposure guide based on a 550 kg Thoroughbred adult horse for a high-output generator X-ray system used with a CR system

Region/ projection	Focus–film distance	Grid	kV	mAs
Saculohumeral joint – shoulder				
ML	120 cm	Parallel	90	50
CrM-CaLO	100 cm	/	90	28
Elbow				
ML	120 cm	Parallel	70	20
CrCa	100 cm	/	77	16
Carpus				
LM, DLPMO and DMPLO	100 cm	/	66	10
DP	100 cm	/	66	12
DPr-DDiO – skylines	100 cm	/	63	10
Metacarpus/tarsus				
LM, DLPMO and DMPLO	100 cm	/	63	10
DP	10 cm	/	66	10
Fetlock joint				
LM, DLPMO and DMPLO	100 cm	/	63	10
DP	10 cm	/	66	10
Feet				
LM, DP	100 cm	/	68	16
DPr-PaDiO – pedal bone	100 cm	/	63	12
DPr-PaDiO – navicular bone	100 cm	/	70	20
PaPr-PaDiO	Depends on horse	/	68	16
Stifle joint				
LM	100 cm	/	68	10
CdCr	100 cm	/	75	20
CaL-CrMO	100 cm	/	66	10
CrPr-CrDi – skyline patella	100 cm	/	66	12
Tarsus				
LM, DLPlMO and DMPlLO	100 cm	/	63	16
DPl	100 cm	/	70	16
Head				
Lateral sinuses	100 cm	/	55	10
Oblique tooth roots	100 cm	/	68	16
DV rostral	120 cm	Parallel	70	30
Pharynx	120 cm	Parallel	68	30
Cervical spine				
Cranial	120 cm	Parallel	73	32
Mid	120 cm	Parallel	80	32
Caudal	120 cm	Parallel	84	36
Caudal T1	120 cm	Parallel	150	71
Thoracolumbar spine				
Wither – spinous processes	120 cm	Parallel	70	40
Mid thoracic	120 cm	Parallel	70	40
Caudal thoracic	120 cm	Parallel	80	40
Lumbar	120 cm	Parallel	90	50
Mid thoracic vertical bodies	120 cm	Parallel	100	120
Pelvis				
Sacroiliac joint	130 cm	Focus	150	280
Coxofemoral joint	130 cm	Focus	130	220
Ilial wings	130 cm	Focus	130	220
Thorax				
Craniodorsal	120 cm	Parallel	90	36
Cranioventral	120 cm	Parallel	110	36
Caudodorsal	120 cm	Parallel	80	28
Caudoventral	120 cm	Parallel	80	28

- For safety, projections are usually acquired with the machine staying on the lateral side of the horse, thus a DMPLO is most commonly taken as a PLDMO, but is (incorrectly) often labelled DMPLO.

Exposure settings

Exposure settings not only vary between the different anatomical regions of the horse and projections, they also vary between X-ray generators (even if it is the same make/model) and detection systems (e.g. CR systems usually require higher exposures than conventional film-screen systems). Table 25.3 gives an example of exposure settings for a high-output generator paired with a CR system.

Exposure adjustment

Some anatomical structures have different thicknesses in different parts and would require repeat exposures to optimize image quality for each part, e.g. the cranial part of the shoulder or stifle require less exposure than the caudal part or the spinous processes and the

vertebral bodies in the equine back. This can be avoided by the use of aluminium dodgers or wedges that have the same radiographic density as soft tissue and are placed between the X-ray tube and the structure to compensate for the differences in exposures required.

Focus–film distance

This is the distance from the focal point of your X-ray machine to the cassette. The focal point should be marked by the manufacturer and is somewhere in the middle of your tube (not at the level of the collimation window). As a general rule, the longer the distance the better the quality of the image, but the higher the exposures needed. It is common practice to use a focal-film distance of 100 cm. This may change due to anatomical constraints, e.g. the distance for the flexor view of the navicular bone is often shorter since the belly of the horse is in the way. In these cases the exposure settings have to be adjusted according to the inverse square law (see above).

Labelling of cassettes

Each cassette has to be labelled with the horse's name and/or number and the date of the examination; this is usually done before or after the examination. A metal clip indicating left/right and if necessary front/hind and the type of projection is placed in the beam during examination. Conventionally these markers are placed on the lateral side and in the case of LM projections on the cranial/dorsal side of the cassette. This can then be used to identify sides on the resulting radiograph.

Scatter reduction

- Scatter radiation deviates from the straight line path, which the primary X-ray beam follows between the object and the cassette.
- The main source of scatter is the Compton interaction, one of three ways X-rays interact with matter.
- The problem with scatter radiation is that it reduces image contrast thus having a negative effect on image quality.
- Scatter is especially a problem where the X-rays have to travel through thick tissue mass, as is the case in the proximal region of the horse.
- There are different ways of controlling scatter:
  - Collimation of the primary beam.
  - Use of a grid:
    A grid is essentially a flat plate with a series of very narrow lead foil strips arranged in parallel lines, in squares or at angles.
    This is positioned between the patient and the cassette.
    These lead foil strips only allow straight X-rays through and hence reach the cassette and absorb the ones that are at an angle.
    Exposure factors will have to be increased and grid lines will be visible in your images.
    Exact alignment of the X-ray beam with the grid is necessary.

Radiography of the different anatomical regions

The foot

See also Chapter 16.

Indication

- Positive palmar digital nerve block.
- Positive distal interphalangeal joint (DIP) block.
- DIP joint effusion.
- Penetrating injury to sole.
- Wounds.
- Laminitis.
- Assessment of foot conformation.
- Prepurchase/presale radiographs.

Equipment

- Portable X-ray machine is sufficient.
- Farriery kit for shoe removal and hoof preparation.
- Play dough or other material to pack frog grooves.
- Flat blocks to raise feet for DP and LM projections.
- Block (tunnel or Hickman block) for oblique projections.
- Lead gowns, thyroid protectors, gloves.

Preparation

- It is preferable to remove the shoes.
- The foot should then be trimmed with hoof knives to remove any loose horn and dirt.
- Careful packing of the foot with e.g. play dough improves image quality by eliminating gas shadows created by the frog.
- Some people prefer not to pack the sole since they prefer to see the outline of the frog grooves over their whole length and not to run the risk of being confused by packing artefacts (Figure 25.21).
- Avoid any pearlized/glitter play dough when packing feet as the metallic component can cause artifacts – especially if the horse is going on to MRI or CT.

Radiographic projections

- Standard foot series:
  - Lateromedial (LM).
  - Dorsopalmar (DP).
  - Dorsoproximal-palmarodistal oblique (DPrPaDiobl) for distal phalanx and navicular bone.
  - Palmaroproximal-palmarodistal (PaPrPadiobl) oblique – Sky-line view, flexor view.
- Additional projections:
  - Dorso 60° proximo 45° lateral-palmarodistomedial oblique projection.
  - Oblique projections such as this may be used for evaluating the wings of both the navicular bone and the pedal bone, and they are particularly useful for confirming the presence of wing fractures of the pedal bone suspected in the 60° DP projection.

Radiographic technique

X-ray machines usually do not go low enough to the ground to allow for centering of the X-ray beam on the DIP joint, hence it is necessary to elevate the foot. It is preferable to take this projection with the foot weight-bearing and have both feet elevated onto two separate blocks which allows for the horse to stand level.

- Lateromedial (LM) projection (Figure 25.27).
  - This projection is used to assess:
    Dorsopalmar foot conformation, hoof-pastern axis.
    Pedal bone rotation/sinking.
    DIP joint: periarticular margins, subchondral bone, extensor process.
    Navicular bone: compacta-spongiosa definition, enthesiophytes, osteophytes.
  - Position both feet on blocks.
  - Position the foot of interest at the edge of the block.
  - Position the X-ray machine lateral to the foot.
  - Centre the X-ray beam about 1 cm below the coronet, halfway between the dorsal hoof wall and the heel.
  - Align the beam parallel to the bulbs of the heel.

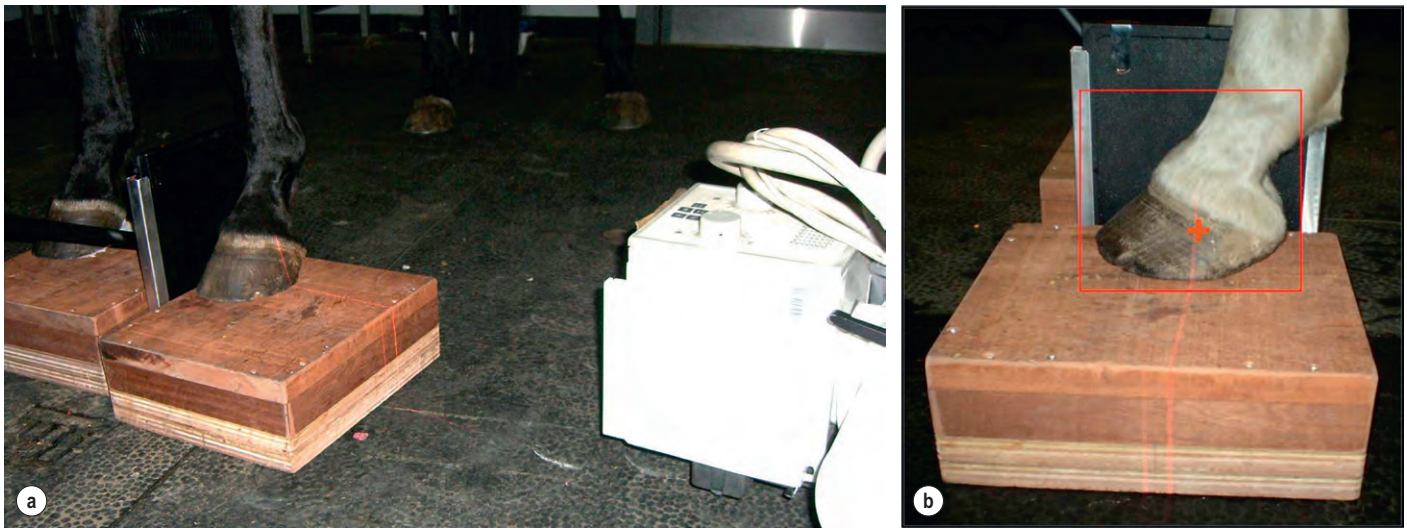


Figure 25.27 Positioning (a) and centring (b) for a lateromedial radiograph of the left foot. Both feet are positioned on blocks to allow for centring of the X-ray beam on the DIP joint with the foot of interest as close to the edge of the block as possible. The cassette is positioned medial to the foot in close contact with the foot using a long-handled cassette holder. The red frame indicates the collimated area.

Figure 25.28 Positioning (a) and centring (b) for a dorsopalmar radiograph of the left foot. Both feet are positioned on blocks to allow for centring of the X-ray beam on the DIP joint with the foot of interest as close to the edge of the block as possible. The cassette is positioned palmar to the foot in close contact with the foot using a long-handled cassette holder. The red frame indicates the collimated area.

- The X-ray beam should be horizontal; however, depending on conformation the X-ray beam may have to be angled to account for obliquity of joint space.
- Place the cassette on the medial side as close as possible to the foot, lower than the solar surface.
- Align the cassette perpendicular to the X-ray beam. Since you cannot see the X-ray beam it is easier to align the cassette parallel to the front of your X-ray machine, alternatively you can use a measuring stick to indicate the direction of the beam, which also allows you to measure the focus-film distance accurately.
- Collimate around the foot up to mid pastern region, making sure you can see all four margins of the collimated area on the cassette.

Radiographic technique (especially angles) described in textbooks works for the 'textbook' case. Look at the individual horse and do not be afraid to adjust your radiographic technique accordingly!

- Dorsopalmar (DP) projection (Figure 25.28).
  - This projection is used to assess:
    Mediolateral foot balance.
    Ossifying collateral cartilages.
    Integrity of the pedal bone.
  - Position both feet onto separate blocks with the foot of interest at the palmar aspect of the block.
  - Position the X-ray machine dorsal to the foot.
  - Centre the horizontal X-ray beam about 1 cm distal to the coronet.

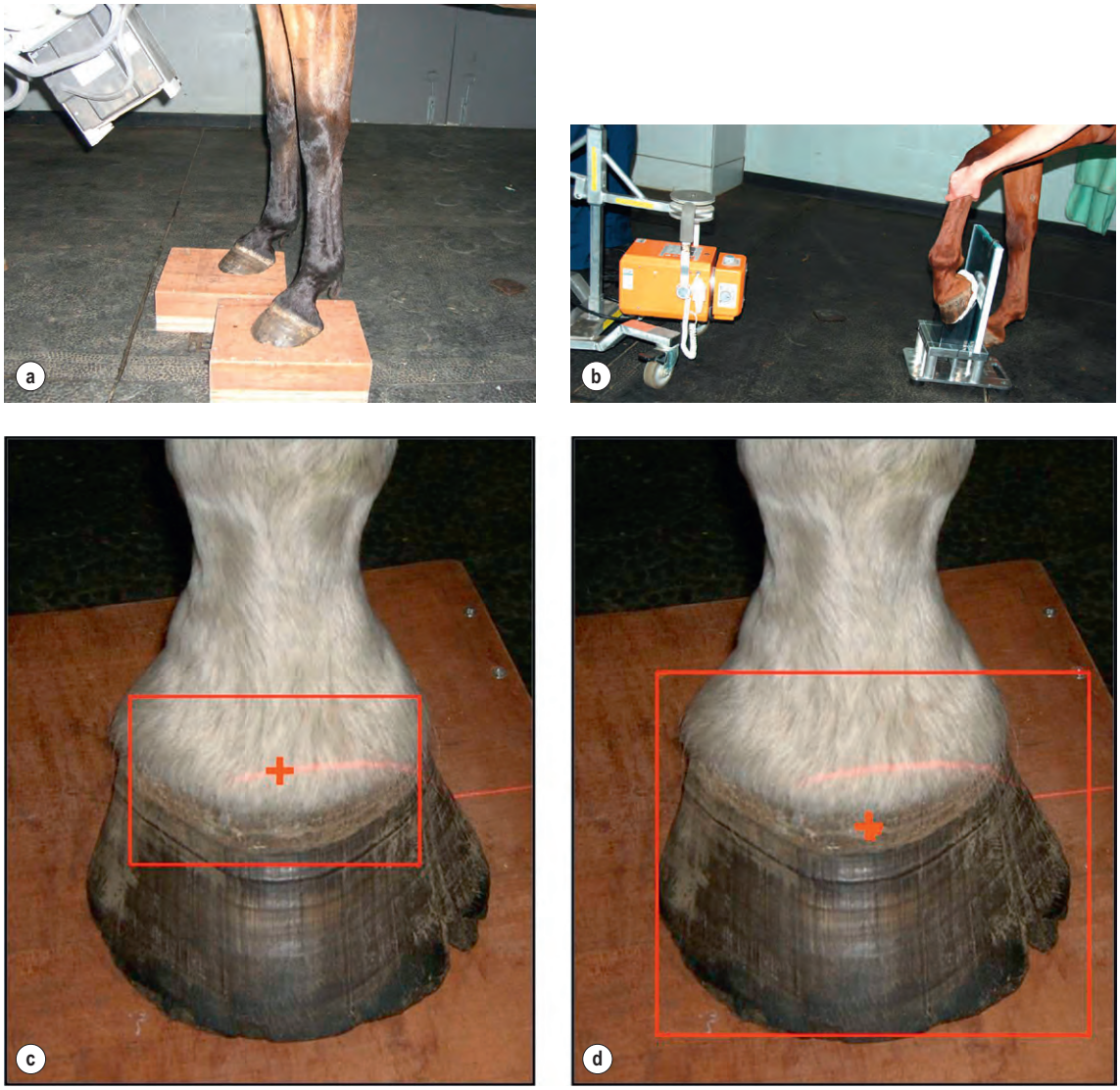


Figure 25.29 Positioning (a), centring and collimation (b, c) for a dorsoproximal-palmarodistal oblique radiograph of the left navicular bone (b) and the pedal bone (c) with the foot resting on a tunnel block that holds the cassette. The red frame indicates the collimated area.

- Place the cassette on the palmar aspect as close as possible to the foot, lower than the solar surface.
- Collimate around the foot up to mid pastern region, making sure you can see all four margins of the collimated area on the cassette.

- Dorsoproximal-palmarodistal oblique projections (Figure 25.29).
  - As standard this projection is performed twice to image the navicular bone (Figure 25.29a) and the distal phalanx (Figure 25.29b).
  - Depending on the obliquity of the beam either the proximal or the distal border of the navicular bone gets highlighted.
  - Dorso 60° proximal palmarodistal oblique (60° DP) projection.
    Centred on and collimated around the navicular bone for evaluating the distal border.
    Centred on the pedal bone, it is used for evaluating its integrity.
  - Dorso 45° proximal palmarodistal oblique (45° DP) projection.
    Centred on the navicular bone, this view has been used to evaluate its proximal horizontal border.
  - Bear in mind that foot conformation and hence angle of the navicular bone varies greatly between horses, in some cases it is advisable to assess the angle of the navicular bone on the lateromedial projection and adjust the angle for the DPr-PaDiobl projection accordingly.
  - There are multiple different methods of taking DPr-PaDiO projections:
    With the horse weight-bearing standing on a tunnel block and angling the X-ray beam in a proximodistal direction.
    With the horse foot positioned in a Hickman block, thus angling the foot to the horizontal X-ray beam. This is favoured by some as it results in less distortion, but it may require more patient restraint than the tunnel block

technique which is to be preferred from a radiographic safety point of view.

- Palmaro-45°-proximal palmarodistal oblique projection (flexor view, skyline view) (Figure 25.30).
  - This projection is used to assess:
    The flexor cortex of the navicular bone.
    The compacta-spongiosa junction.
    Palmar processes of the third phalanx.
  - Place the foot on a tunnel block.
  - Position the foot as far back underneath the horse as possible whilst still keeping the foot weight-bearing. This will help to avoid superimposing the proximal sesamoid bones and the proximal and middle phalanx over the navicular bone.
  - The X-ray machine is positioned on the palmar aspect of the leg under the abdomen of the horse. It facilitates positioning of the X-ray tube when the foot is slightly placed pointing away from the horse.
  - Angle the X-ray beam in a proximo-distal direction at 45°.
  - Centre the X-ray beam just proximal to the heel bulbs.
  - Collimate tightly around the navicular bone to reduce scatter.

Radiographic anatomy

- Radiographic anatomy on the LM projection (Figure 25.31).
  - The pedal bone develops from a single centre of ossification, which is present at birth. It ossifies until at least 18 months

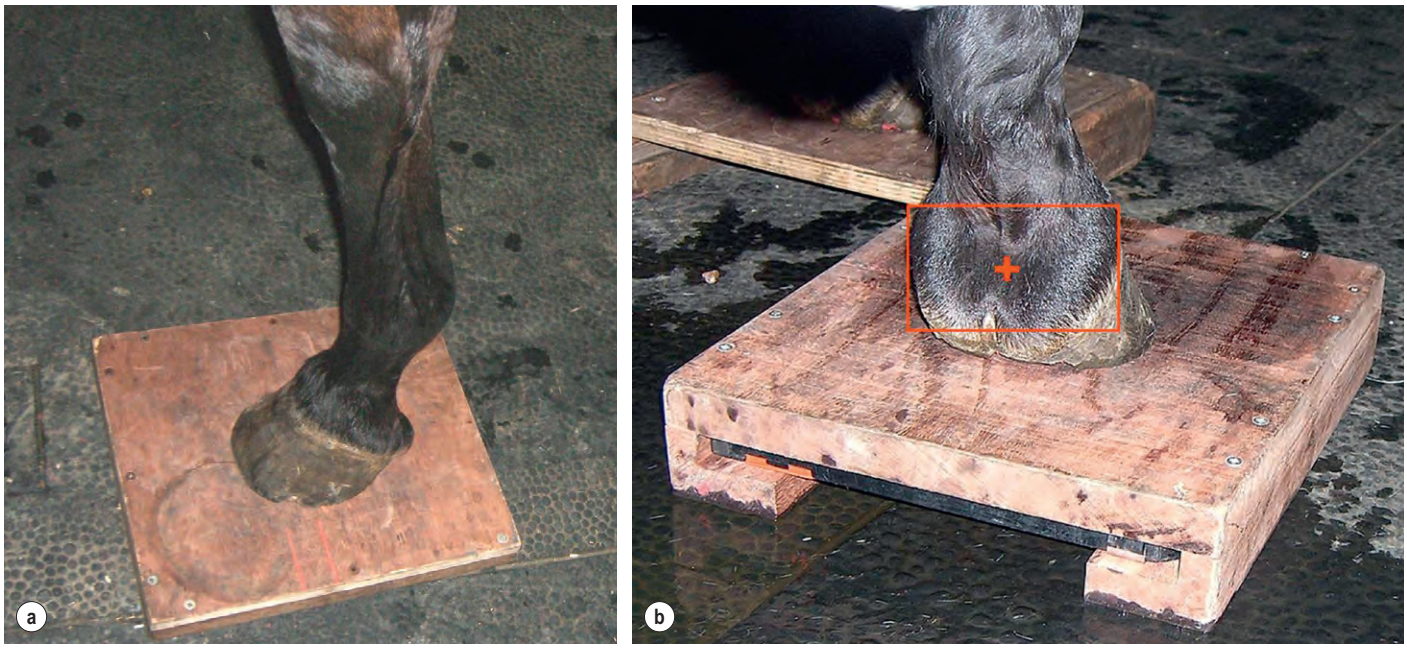
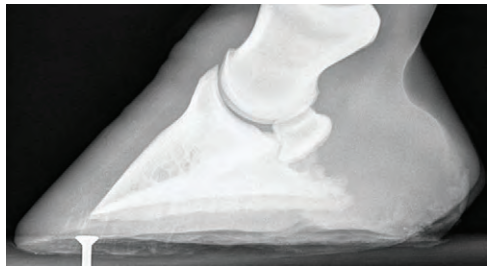


Figure 25.30 Positioning (a) and centring (b) for a palmaroproximal-palmarodistal oblique ('flexor view', 'skyline view') of the navicular bone. The X-ray machine is positioned under the belly of the horse and the foot rests on a tunnel block. The red frame indicates the collimated area.



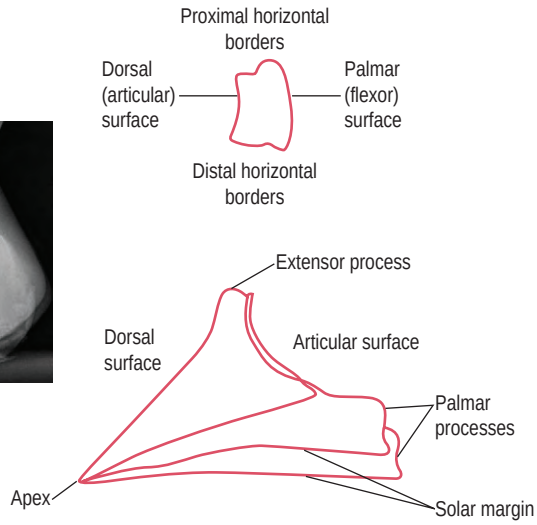


Figure 25.31 Radiographic anatomy of the foot in a lateromedial projection.

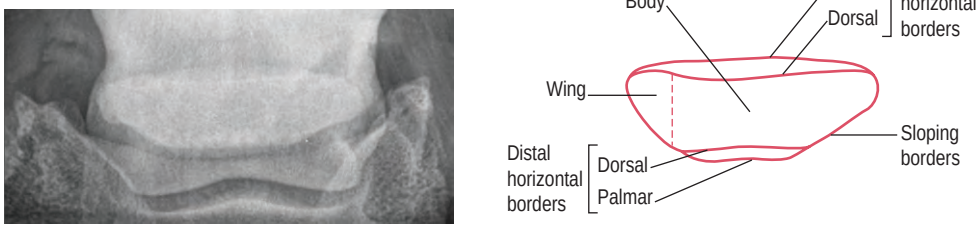


Figure 25.32 Radiographic anatomy of the navicular bone in a dorsoproximal-palmarodistal projection.

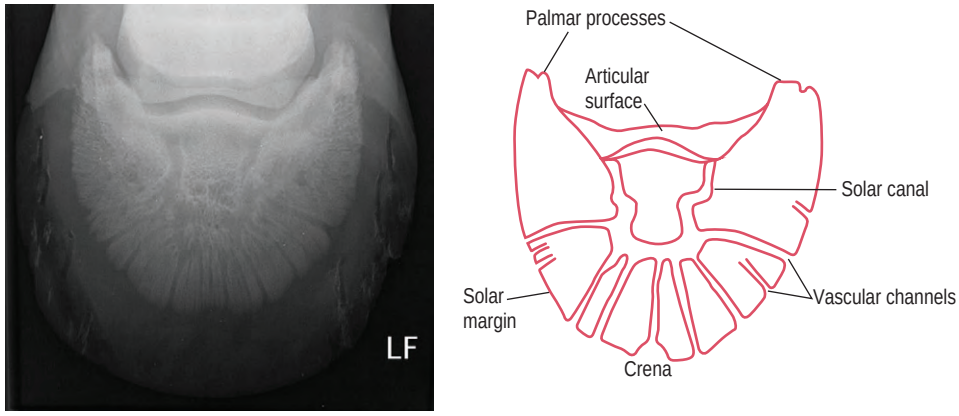


Figure 25.33 Radiographic anatomy of the foot in a dorsoproximal-palmarodistal projection.

of age. The palmar processes are not evident at birth, but gradually ossify over 12 months.
- The solar canal is seen between the solar surface of the bone and the distal interphalangeal joint.
- Palmar to the solar canal, there is a sharply defined, smoothly outlined radio-opaque area, which represents the facies flexoria (attachment of the deep digital flexor tendon).
- There are morphological variations in the appearance of the extensor process, and it is not always easy to distinguish normal from abnormal.
- Ossifying collateral cartilages may be superimposed on the navicular bone.
- The navicular bone is usually trapezoid-shaped, but can have a proximal and distal elongation of the flexor cortex, which can be normal or represent new bone formation.
- The spongiosa and spongiosa of the navicular bone are distinctly outlined in a normal horse on a perfect LM view.
- There is usually a notch of variable depth within the distal surface, where the synovial fossae are located.

- Radiographic anatomy on the dorsoproximal-palmarodistal oblique projection.
  - Navicular bone (Figure 25.32).
    - It ossifies from a single centre and has an oval shape at birth. It is completely ossified by approximately 18 months of age.
    - The outline of the navicular bone varies between animals, but is often a mirror image to the contralateral limb.
    - Depending on the projection the proximal border may be visible as two lines. The more distal of these lines represents the proximal articular border, which can vary in shape.
    - The distal border is visible as two lines; the more proximal represents the distal border of the articular surface. The more distal line represents the distal border of the flexor surface from which the distal impar sesamoidean ligament originates.
    - Small, round or triangular shaped lucent zones can be seen in the distal border and represent synovial fossae. It is generally accepted that more than 7 foramina of more variable size and shape are more likely to be clinically significant.
  - Pedal bone (distal phalanx) (Figure 25.33).
    - The solar margin is well defined, but some irregularities may be present. The vascular channels are seen as radiolucent lines, radiating from the solar canal to the periphery.
    - The solar canal is visible as a roughly U-shaped radiolucent canal in the centre of the bone.
    - A semicircular notch (crena) may be present on dorsal midline.
    - The pedal bones of the hind limbs are narrower and have a slightly more pointed outline at the toe than those of the front feet.
- Radiographic anatomy of the palmaro45°proximal-palmarodistal oblique (Pa45°PrPaDIO) projection (Figure 25.34).
  - The navicular bone is characterised by a more radiolucent spongiosa centrally surrounded by more radiodense compact bone.
  - Superimposed on the spongiosa the synovial fossae are seen as round to oval shaped lucencies.
  - The flexor compacta is relatively even in thickness and may have a small crescent shaped lucency in the sagittal ridge.

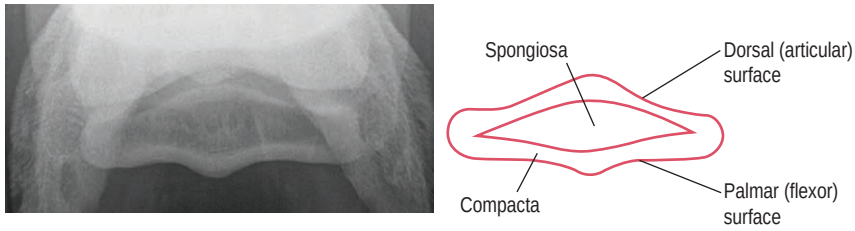


Figure 25.34 Radiographic anatomy of the navicular bone in a palmaroproximal-palmarodistal oblique projection.

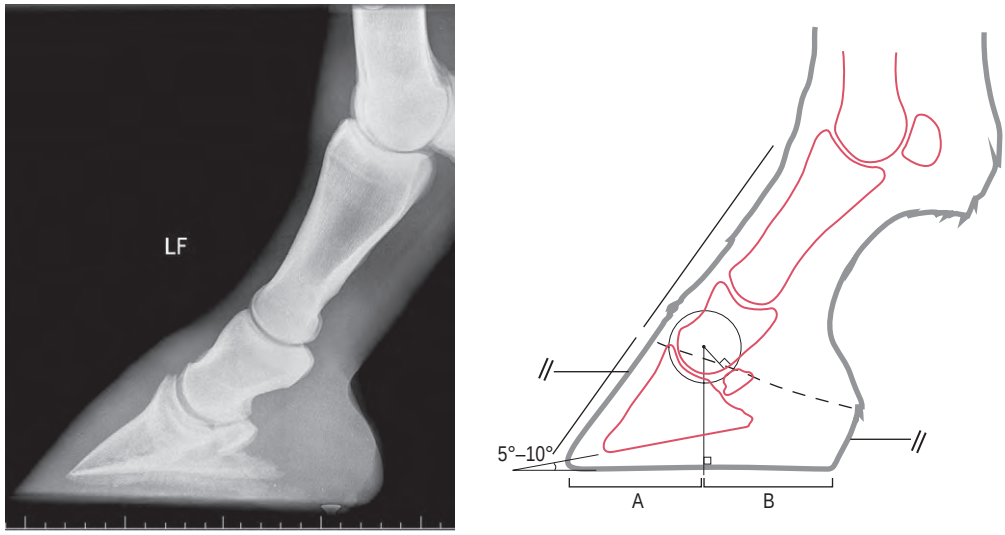


Figure 25.35 Assessment of foot conformation on a lateromedial radiograph. The schematic drawing shows the 'ideal' conformation. The example radiograph shows a long-toe, flat sole conformation (often seen in Thoroughbreds) and a broken-back hoof-pastern axis.

  - Care should be taken, when evaluating the thickness of the flexor compacta, the opacity of the spongiosa and the spongiosa-compacta interface. Even on relatively well positioned views, the thickness of the flexor compacta might appear falsely increased, the spongiosa more opaque and the interface poorly demarcated.
  - Increased opacity overlying the mid-navicular bone can result from thick overlying frog.
- Radiographic anatomy of the dorsopalmar (DP) projection.
  - The solar canal is seen as two distinct, circular foramina distal to the articular surface of the pedal bone.

Radiopathology

- Assessment of foot conformation (Figure 25.35).

Foot conformation influences the forces acting on the structures in the foot, especially the deep digital flexor tendon, the navicular bone and the distal interphalangeal joint. This may predispose horses to developing pathology and radiographs are hence often used to assess foot conformation in lameness work-ups but also as guidance for farriery.

One should bear in mind that many of the parameters used are influenced by the current trimming and/or shoeing status and by the stance of the horse when the radiographs are taken.

Figure 25.35 demonstrates conformation assessment on radiographs.

- Dorsopalmar imbalance:
  - Assessed on lateromedial projections.
  - The slope of the heels should equal the slope of the dorsal hoof wall.
  - A plumb line drawn from the centre of the distal end of the middle phalanx to the ground should transect the solar surface in its middle third.
  - The solar surface should show a 2 to 10 degree upward slope from dorsal to palmar/plantar.
  - The dorsal surface of the pedal bone and hoof wall should be parallel.
  - There are variations in the thickness of the dorsal hoof wall and the sole.
- Hoof pastern axis:
  - Assessed on a lateromedial projection.
  - The dorsal hoof wall and the dorsal surface of the pastern should have the same slope; this is assessed by drawing lines along the long axis of the bones and assessing the angles between those lines.
- Lateromedial imbalance:
  - Assessed on a dorsopalmar projection.
  - The height of the lateral heel should equal the height of the medial heel.
  - A line bisecting the proximal, middle and distal phalanx should be straight and perpendicular to the floor.
  - These may be influenced by the way the horse is standing!
- Laminitis (Figure 25.6). Although clinical examination is usually sufficient to diagnose laminitis, radiographs are essential to determine the severity of disease and hence treatment and prognosis. The distal phalanx is suspended within the hoof wall by laminar attachment. In horses suffering from laminitis this laminar attachment is disrupted. Depending on the degree and

pattern of laminar detachment this leads to rotation and/or sinking of the distal phalanx within the horn capsule. Lateromedial radiographs with markers outlining the dorsal hoof wall and the coronary band are standard procedure. The following parameters are evaluated:

- The presence of a radiolucent line within the dorsal hoof wall indicates air between the separating laminae.
- The dorsal hoof wall and the dorsal surface of the distal phalanx should be parallel. Diverging angles indicate rotation of the distal phalanx away from the hoof capsule.
- An increase in distance between the coronet and the extensor process of the distal phalanx indicates sinking of the distal phalanx within the hoof capsule.
- A radiolucent line along the coronet is also an indicator of sinking.
- Both rotation and sinking causes the tip of the toe to move closer to the solar surface which it breaks through in extreme cases.
- Chronic radiographic signs of laminitis includes deformation of the tip of the distal phalanx to resemble a 'slipper', thickening and ring formation of the hoof wall, signs of osteitis and disuse osteopenia.

- Pus-in-the-foot (Figure 25.36).

This may be primary, or secondary to laminitis. Its presence may be recognized by gas lucencies deep to the horny tissues.

- Keratoma (Figure 25.37).

This is an unusual horny neoplasm of the foot. Its presence can be inferred from a well-defined lucency in the distal phalanx associated with pressure-induced bone resorption. However, the normal crena should not be mistakenly identified as an abnormality.

- Pedal osteitis.
  - This condition is secondary to inflammation of the sensitive laminae following laminitis or chronic foot imbalance. Localized pedal osteitis may be seen following chronic foot infections.
  - Classic changes are a reduction in opacity of the distal phalanx seen in the DPrPaDiobl projection, together with increased number and size of vascular channels extending to its margin.
  - In the LM projection, new bone formation may be visible on the dorsal margin of distal phalanx.
  - Unless these changes are gross, or radiographs exist showing the pedal bone prior to onset of the condition, it is extremely difficult to diagnose.
- Fractures.
  - These are best divided into four categories, descriptively, according to the course of the fracture.
    Wing fractures may be articular or non-articular (Figure 25.20).
    Sagittal fractures.
    Extensor process fractures (Figure 25.38).
    Solar margin fractures.

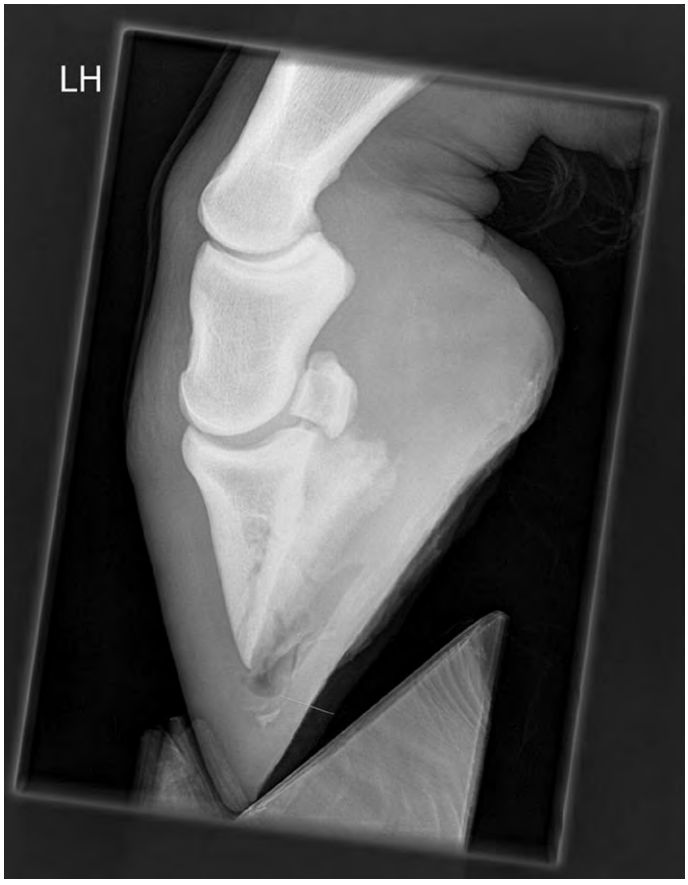


Figure 25.36 Pus in the foot. Lateromedial radiograph of the foot shows a gas shadow within the horn at the tip of the toe.

Figure 25.37 Keratoma. This dorsoproximal-palmarodistal radiograph of the foot shows a circular disruption of the outline of the distal phalanx consistent with bone absorption due to pressure from a keratoma. The keratoma itself cannot be distinguished from the surrounding horn since it has the same radiodensity.

Figure 25.38 This lateromedial radiograph of the foot shows an isolated, rounded, smoothly outlined bony opacity at the extensor process of the distal phalanx. This represents most likely an extensor process fragment; however, it canot be ruled out that it is due to non-union of the separate ossification centre from which the extensor process ossifies.

  - Lesion orientated projections are often necessary to show the fracture radiographically.
- Osteoarthritis.
  - New bone production is recognized particularly on the extensor process of the distal phalanx in the LM projection and at the dorsoproximal edge of the navicular bone.
  - DIP joint effusion can be seen radiographically as a soft-tissue opacity bulging out on the dorsal aspect just proximal to the coronet.
  - Subchondral bone sclerosis and narrowing of the joint space can be seen in advanced cases.
- Bone cysts.

These can occur in the distal phalanx, as elsewhere. Their clinical significance will depend on size and proximity to the distal interphalangeal joint.

- Ossification of the collateral cartilages (Figure 25.39).
  - Degree varies between individual horses, heavy breeds seem predisposed.
  - They can ossify from separate centres with radiolucent lines present throughout life.
  - Often more pronounced on the lateral side.
  - Referred to as 'sidebone' in equestrian terms when extensive.
  - Usually of no clinical significance, but can impede movement when extensive and can be associated with lameness if fractured, infected or if associated ligaments are injured.
- Navicular bone.

Navicular syndrome is a term used for horses suffering from chronic forelimb lameness associated with the navicular apparatus (see Chapter 16). The navicular apparatus comprises the navicular bone and the soft-tissue structures associated with it, including the navicular bursa, the DDFT, the collateral ligaments of the navicular bone and the distal sesamoidean impar ligament. Soft-tissue changes cannot be appreciated on radiographs and require MRI. Radiographs do however allow the assessment of the navicular bone itself and secondary bony changes such as enthesiophyte formation at the attachments of ligaments. A grading system for radiographic evaluation has been developed; however, there is often a poor correlation between radiographic signs and lameness. With the exception of flexor surface erosion, that represent radiographically as large round lucencies in the spongiosa on the DPrPaDio and as a disruption of the flexor surface on the

Figure 25.39 Ossified collateral cartilage. This dorsopalmar radiograph shows a unilaterally ossified collateral cartilage.

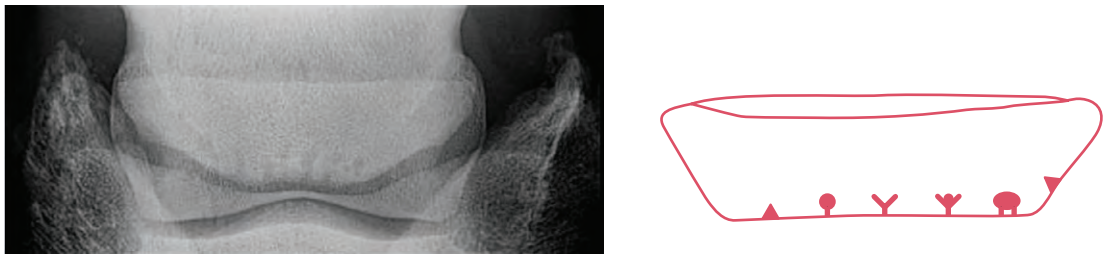


Figure 25.40 Synovial fossae in the distal border of the navicular bone. Fewer than six, narrow, conical shaped fossae are considered normal, more than six of variable shape or less than six large ones are considered abnormal.

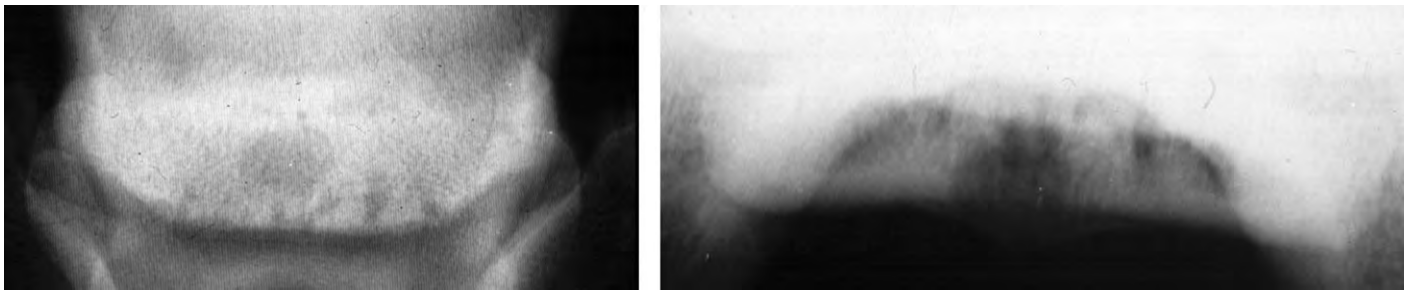


Figure 25.41 The two radiographs show a large defect in the flexor compacta of the navicular bone, which presents as a large, circular lucency overlying the spongiosa in the dorsoproximo-palmarodistal oblique projection and as a defect in the outline of the flexor compacta in the palmaroproximal-palmarodistal oblique projection. There are also several synovial fossae of variable shape and size.

PaPrPaDiobl, none of the evaluated parameters are pathognomonic for navicular disease, however the likelihood for a horse to suffer from navicular disease seems to go up with the number of radiographic abnormalities present (Figures 25.40, 25.41).

The following parameters should be evaluated systematically:

- Distal border: (Figures 25.40, 25.41).
  - Synovial fossae on the distal border: shape, number, size, location.
  - Fewer than six, narrow, conical shaped fossae are considered normal.
  - Mineralized opacities off the distal border.
  - New bone formation on the flexor cortex.
- Opacity of the bone:
  - Lucent areas within the spongiosa or compacta (Figure 25.41).
  - Poor demarcation between compacta and spongiosa (this however is influenced by projection).
  - Loss of trabecular pattern of the bone.
  - Thickness of the flexor compacta.
- Shape of the navicular bone.
  - Navicular bones should be symmetrical and similar in both legs.
- Proximal border:
  - The proximal border shows two margins (Figure 25.32): the articular border and the flexor border, which is variable in outline. Concave or undulating borders have been suggested to be associated with a higher risk of navicular disease in Warmbloods.
  - New bone formation representing enthesiophytes at the attachment sites of the collateral ligaments.
- The distal interphalangeal joint should be carefully evaluated for radiographic signs of osteoarthritis.

The fetlock

See Chapter 16.

Indication

- Positive low four-point nerve block.
- Positive fetlock joint block.
- Fetlock swelling.
- Wounds.
- Trauma.
- Prepurchase/presale radiographs.

Equipment

- Portable X-ray machine is sufficient.
- Cassette holder, either free-standing or hand-holder with long handle.
- Lead gowns, thyroid protectors, gloves.

Preparation

- Area to be examined should be brushed to remove any dirt.
- Patient should be adequately sedated and be standing on an even surface while weight bearing evenly on all four limbs with the cannon bone vertical.

Radiographic projections

- Standard fetlock series:
  - Lateromedial (LM) (Figures 25.42, 25.43).
  - Dorsopalmar (DP) (Figures 25.44, 25.45).
  - Dorso 45° lateral-palmaromedial oblique (DLPMO) projection.
  - Dorso 45° medial-palmarolateral oblique (DMPLO) projection (Figure 25.46).

- Flexed lateromedial: shows more of the sagittal ridge compared to the standing LM (Figure 25.47, Figure 25.10, right image).

- Additional projections:
  - There are a variety of oblique projections to highlight different parts of the proximal phalanx, the distal metacarpus and the proximal sesamoid bones.

Technique

- Lateromedial projection (Figures 25.42, 25.43).
  - Position X-ray machine lateral to the horse.
  - Centre on fetlock joint space.
  - Use horizontal X-ray beam.
  - Align X-ray beam perpendicular to the leg; because you cannot see the X-ray beam it is usually easier to align the glass front of the X-ray machine parallel to the leg and cassette and/or use measuring stick as a guide mimicking the direction of the X-ray beam.
  - Position cassette on the medial side of the joint, parallel to the leg, perpendicular to the X-ray beam.
  - Collimate around the fetlock joint, taking care that all edges of the X-ray window are on the plate.
- Dorsopalmar (DP) (Figures 25.44, 25.45).
  - This projection is in fact usually acquired as a Dorso 15° proximal – palmarodistal oblique (D15°Pr-PaDi oblique) projection to prevent superimposition of the proximal sesamoid bones on the joint space.
  - Position X-ray machine dorsal to the horse.
  - Centre on fetlock joint space.
  - Angle X-ray beam 15° in a dorsoproximal-palmarodistal direction.
  - Position cassette on the palmar aspect of the joint perpendicular to the X-ray beam.
- Dorsolateral-palmaromedial and dorsomedial-palmarolateral obliques (DLPMO/DMPLO) fetlock (Figure 25.46 and Figure 25.10, left image).
  - The DLPMO highlights the lateral proximal sesamoid bone and the dorsomedial and palmarolateral aspect of the joint.
  - The DMPLO highlights the medial proximal sesamoid bone and the dorsolateral and palmaromedial aspect of the joint. This projection is often acquired with the X-ray machine on the lateral side of the horse as a PLDMO.
  - Position X-ray machine dorsolaterally (DLPMO) or palmarolaterally (DMPLO/PLDMO) to the leg.
  - Align X-ray beam at 45° angle to sagittal plane.
  - Use a horizontal beam.
  - Centre on fetlock joint space.
  - Position cassette perpendicular to the X-ray beam.
- Flexed lateromedial fetlock (Figure 25.47 and Figure 25.10, right image).
  - Allows the assessment of the sagittal ridge of the third metacarpal bone and the articular surfaces of the proximal sesamoid bones.
  - There are two different techniques:
    Either rest the horse's toe on a block with the fetlock joint flexed *or* it is possible to hand-hold the limb with the

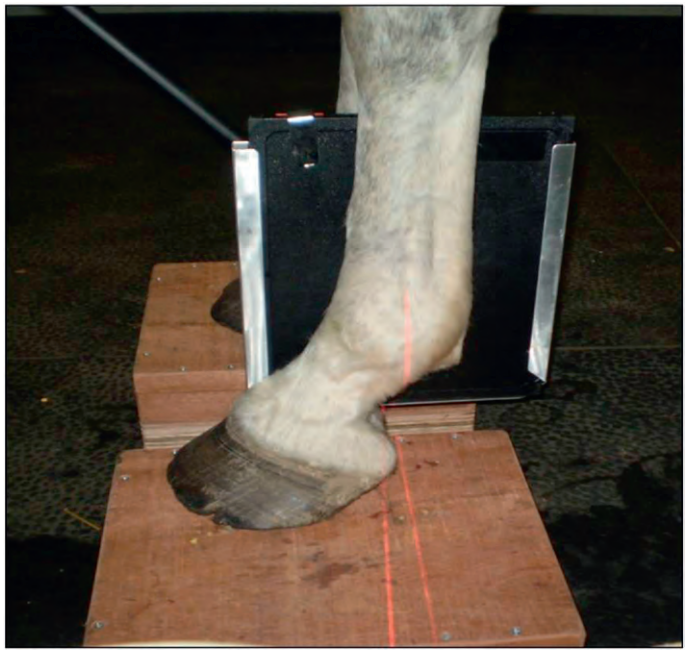


Figure 25.42 Positioning and centring for a lateromedial radiograph of the left fetlock: depending on the size of the X-ray tube, the feet may need positioned on blocks to allow for centring of the X-ray beam on the fetlock joint with the leg of interest as close to the edge of the block as possible. The cassette is positioned medial to the leg in close contact with the foot using a long-handled cassette holder. The red frame indicates the collimated area.

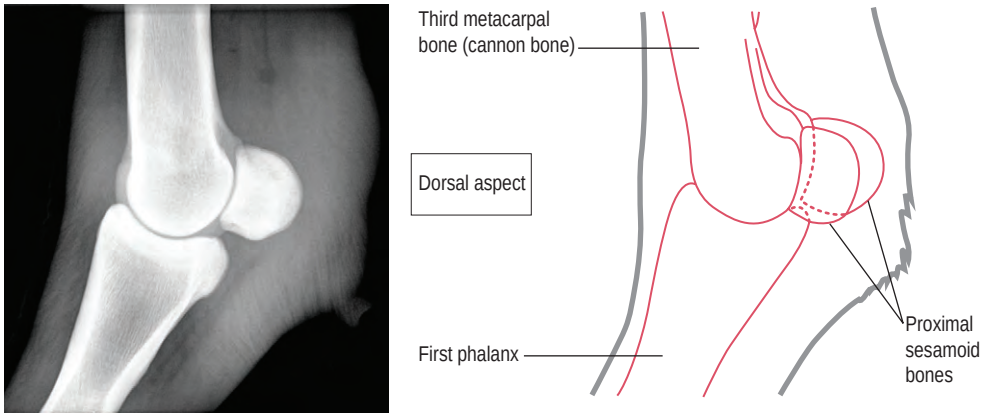


Figure 25.43 Radiographic anatomy of the fetlock in a lateromedial projection.

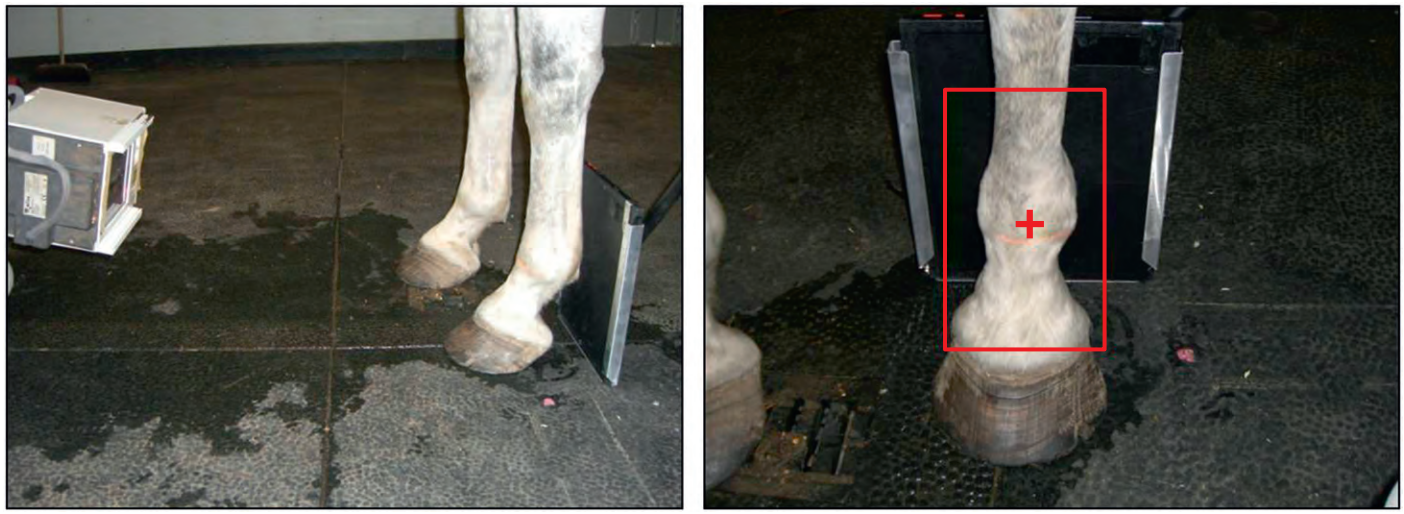


Figure 25.44 Positioning (left image) and centring (right image) for a dorsopalmar radiograph of the left fetlock. The cassette is positioned palmar to the foot in close contact with the foot using a long-handled cassette holder. The beam is angled down by about 15° to prevent superimposition of the proximal sesamoid bones on the joint space. The red frame indicates the collimated area.

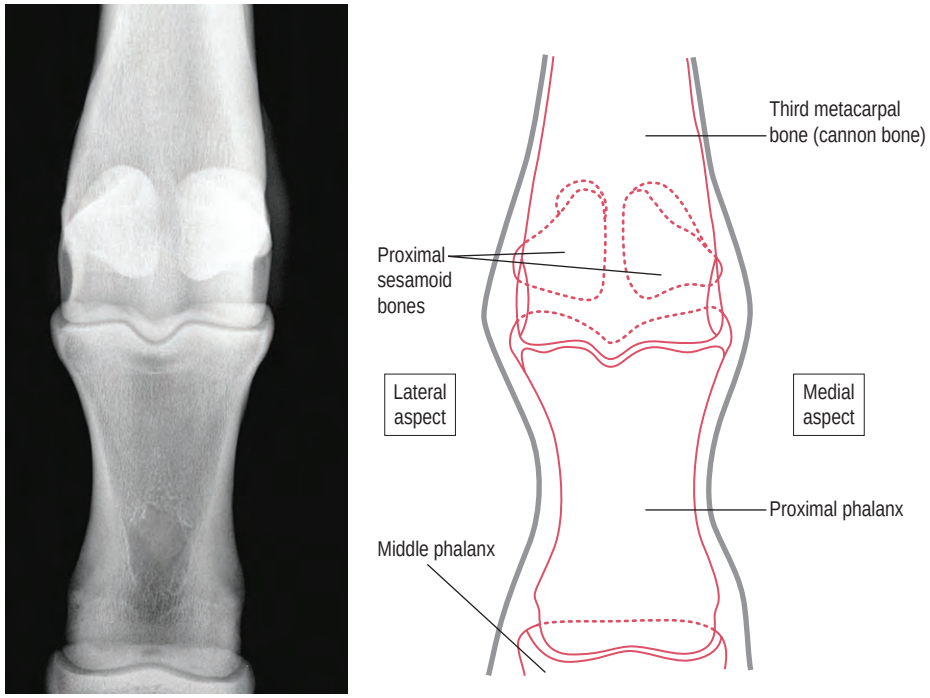


Figure 25.45 Radiographic anatomy of the fetlock in a dorsopalmar projection. The oval radiolucency, devoid of trabecular pattern shown in the distal first phalanx represents the marrow cavity of the proximal phalanx and is normal.

person holding the leg wearing lead-lined gloves, holding the tip of the toe with one hand and supporting the radius with the opposite hand. Carefully collimate ensuring that the gloved hand is not in the primary beam. Proceed like a normal LM.

Radiographic anatomy

- The fetlock joint is formed by the third metacarpal bone, the proximal phalanx and the proximal sesamoid bones. It has a single joint compartment.
- The fetlock joint is a high-motion joint, hence even subtle radiographic changes may be clinically significant.
- The distal articular surface of the third metacarpus carries the sagittal ridge, a predilection site of OCD (Figure 25.10).
- The medial proximal sesamoid bone is larger than the lateral, the lateral sesamoid bone is more L-shaped (Figure 25.45).
- The medial condyle of the distal metacarpus is wider than the lateral (Figure 25.45).
- The hind limb proximal sesamoid bones are more triangular in shape than in the forelimb and often more irregular in outline.

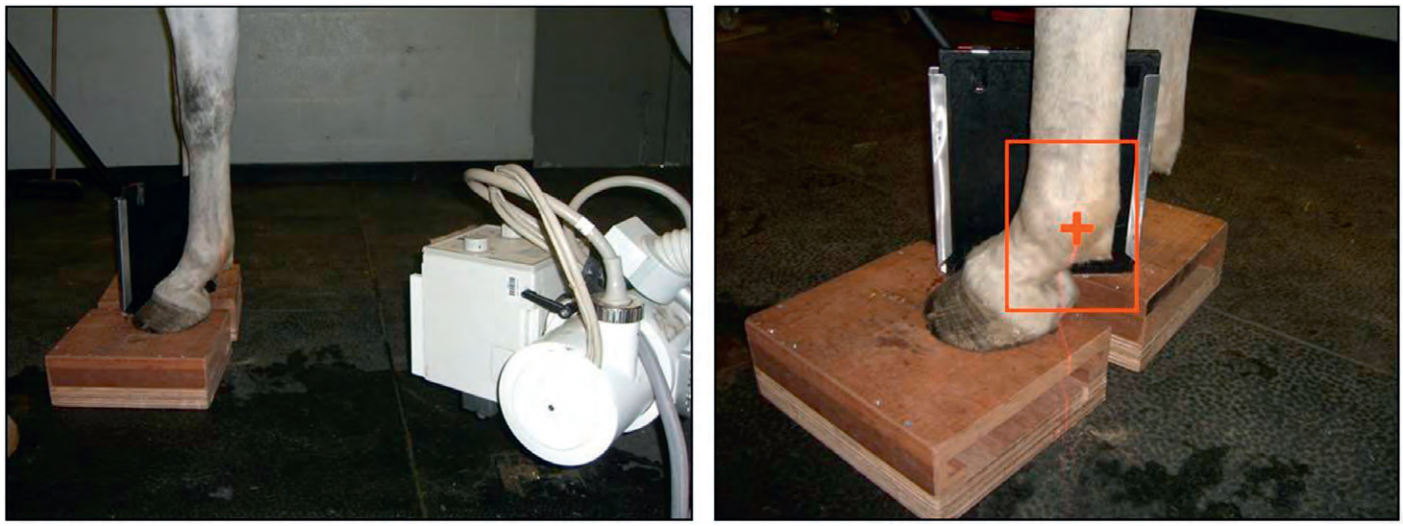


Figure 25.46 Positioning (left image) and centring (right image) for a palmarolateral-dorsomedial oblique radiograph of the left fetlock. This projection can also be taken as a dorsomedial-palmarolateral view with the X-ray machine positioned in front of the horse and the cassette on the palmarolateral aspect of the fetlock. For the opposite oblique, the dorsolateral-palmaromedial the X-ray beam would be at 90° angle to the projection shown in the picture.

Figure 25.47 Positioning for a flexed lateromedial of the right fetlock.

- The dorsoproximal end of the sesamoid bones vary in shape. This is also a predilection site for osteophyte formation and it may be difficult to differentiate between normal variation and pathology.
- The ergot may be seen as a radio-opaque structure superimposed on the proximal phalanx.
- A circular radiolucency, devoid of trabecular pattern, is usually found in the distal first phalanx in the dorsoproximal-palmarodistal oblique projection. This represents the marrow cavity of the first phalanx and is normal (Figure 25.45).
- Nutrient foramina are variably seen in the cortices of the proximal phalanx in the lateromedial projections. They are

Figure 25.48 Sesamoiditis. Two oblique radiographs of fetlock joints: the image on the left shows radiating, linear lucencies in the highlighted proximal sesamoid bone, compared to the proximal sesamoid bone in the left image, which is normal.

usually identified proximally on the dorsal cortex and distally on the palmar cortex.

- Occasionally, a small, smooth, trabeculated bony fragment can be identified on the dorsoproximal aspect of the second phalanx. These are usually insignificant but need to be differentiated from true chip fractures.

Radiopathology

- Osteoarthritis.
  - The fetlock joint can show all the classic radiographic signs of OA (Figure 25.4), including:
    Joint effusion: can be appreciated as outbulging of the normal joint outline on the dorsal aspect.
    Narrowing of the joint space: often best appreciated on the DP projection.
    Subchondral sclerosis: often best seen on the DP and/or LM projection. Subchondral sclerosis is a characteristic of advanced degenerative joint disease and is thought to arise from increased point forces on the subchondral bone as a result of cartilage loss.
    Osteophytes: these result from ossification of the remodelling periarticular margin of cartilage. They are seen at the margins of the joint and are particularly evident in the fetlock joint at four sites on the lateromedial view – dorsoproximal sagittal ridge of MCIII, dorsoproximal P1, and the apex and base of the proximal sesamoid bones.
    If signs of OA are present, the joint should be evaluated (radiography and ultrasonography) for any underlying causes (OCD, osseous cyst-like lesions, fractures, soft tissue lesions, etc.).
    Spurlike new bone formation representing periarticular osteophytes are often seen on the dorsoproximal aspect of the middle phalanx. The pastern joint is a low motion joint and this is usually of no clinical significance.
- Entheseophytosis.
  - New bone formation representing entheseophytes is commonly seen on the palmar aspect of the first phalanx and the dorsal aspect of the second phalanx – the former is the site of attachment of the oblique distal sesamoidean ligaments and the latter the attachment of collateral ligaments of the distal interphalangeal joint. They are particularly evident in the oblique projections and are usually of no clinical significance (Figures 25.4c, 25.5).
- Sesamoiditis (Figure 25.48)
  - An increase in the number and prominence of the vascular channels within the proximal sesamoid bones has been described as sesamoiditis. However, these changes can also be seen in sound horses and there is some debate as to their significance.
  - There does not appear to be any net loss of bone, and the increased number and width of lucencies are thought to arise from entheseophyte formation within the suspensory ligament surrounding the vascular channels. Such changes are usually secondary to other pathology.
  - Suspensory branch injury can result in entheseophytosis or avulsion fractures of the abaxial surface of the proximal sesamoid bone. It is often unclear whether these osseous fragments are actual avulsion fractures or entheseophytes. They can be co-incidental findings not associated with lameness.
- Proximal sesamoid fractures.
  - Occur through overextension of the fetlock joint and are frequently an avulsion type fracture.
  - They can be divided into apical (involving less than a third of the bone), mid-body (involving the middle third of the bone proximally), basilar (involving less than one third of the bone distally), abaxial, and sagittal/axial (often associated with metacarpal condylar fractures).
  - Greater clarification of these fractures can be obtained using a LPr-MDi-O projection.

- Osteochondral fragments.
  - Osteochondrosis (Figure 25.10).
    Predilection sites: dorsal sagittal ridge (proximally and distally), palmaroproximal aspect and, possibly, dorsomedial and dorsolateral proximal end of the proximal phalanx.
    Fragments off the palmaroproximal part of the proximal phalanx are usually close to the sagittal groove and are often difficult to positively identify in the routine views and require specialized views.
  - Traumatic (Figure 25.10).
    Overextension of the fetlock joint can result in osteochondral fractures of dorsoproximal aspect of the proximal phalanx because of its impingement on the dorsodistal aspect of the third metacarpus.
    They are most commonly found either medial or lateral to the sagittal ridge.
    Small rounded fragments can be found in sound horses with no history of fracture and may represent another manifestation of osteochondrosis.
    Osteochondral fractures of the palmaromedial and palmarolateral eminences of the proximal phalanx need to be differentiated from the other types of palmar fragments, especially osteochondrotic lesions which are smaller intra-articular fragments involving the same area. With fractures, there is usually a history of sudden severe lameness. Small basilar fractures of the proximal sesamoid bones may represent avulsion fractures, while other palmar fragments are thought to represent enthesiophytosis of the deep or cruciate distal sesamoidean ligaments.
- Fractures with diaphyseal components.
  - Distal metacarpus: medial condylar fractures tend to spiral up the metatarsus, while lateral condylar fractures tend to be restricted to the lateral condyle (Figure 25.15).
  - Proximal and middle phalanges: midline sagittal fractures are the most common (Figure 25.3).
  - Incomplete/non-displaced sagittal fractures or medial or lateral metacarpal condylar fractures may require multiple views at slightly different angles or, alternatively, more specialized views. If still no fracture is identified and the clinical picture is highly suspicious of a fracture, gamma scintigraphy can be undertaken or the animal box-rested for ~10 days when the fracture line should be apparent on further radiographs. Periosteal new bone on the dorsoproximal aspect of the proximal phalanx in the lateromedial projection is very suggestive of a previous incomplete sagittal fracture of the proximal phalanx.
- Periosteal new bone.

Periosteal new bone can form in response to local trauma, localized sepsis (osteitis), fracture, following advanced joint sepsis, and in hypertrophic osteopathy.

- Degenerative bone lysis.

There are three predilection sites for degenerative erosions:

  - Supracondylar lysis: chronic synovitis can produce remodelling of the palmar aspect of the third metacarpus immediately proximal to the condyles (Figure 25.49).
  - Dorsodistal metacarpus: chronic irritation within the joint results in hypertrophy and fibrosis of the villonodular pad. As it enlarges, it encroaches on the articular surface and results in degenerative changes in the articular cartilage and underlying bone. Radiographically this is apparent as remodelling of the third metacarpal bone just proximal to the dorsal articular surface.

Figure 25.49 Supracondylar lysis. This lateromedial radiograph shows remodelling of the palmar aspect of the third metacarpus immediately proximal to the condyles usually produced by chronic synovitis.

  - Palmarodistal MCIII. Erosions in the articular surface of the palmarodistal MCIII opposite the base of the proximal sesamoid bones can occur in racehorses and most commonly in the forelimb. Specialized projection (D 125° Di-PaPr-O) is useful for identifying these lesions.
- Septic arthritis.
  - In cases of joint sepsis, few changes are apparent until late in the course of the condition when they resemble advanced OA: joint effusion, gas lucencies, osteolysis (often seen in the proximal sesamoid bones), aggressive new bone formation, subchdronal bone lucencies and sclerosis (Figure 25.1).
  - Osteomyelitis and sequestrum formation can occur in the subchondral bone, most commonly in foals, where the changes are a combination of lysis and sclerosis (Figure 25.2).
- Soft-tissue pathology.
  - Joint effusion can be identified as outbulging of the dorsal pouch of the joint on the LM projection or as lateral/medial outbulging on the DP projection.
  - Digital sheath effusions associated with annular ligament syndrome are indented with a 'notch'.
  - Tendinitis and suspensory ligament 'desmitis' also produce soft-tissue swelling, radiographically seen as changes in shape and thickening on the palmar aspect of the metacarpus.
  - Suspensory ligament problems are thought to be associated with radiolucencies in the proximal sesamoid bones (sesamoiditis) (Figure 25.48, left image). Severe injury to the suspensory ligament can result in distal displacement of the proximal sesamoid bones.

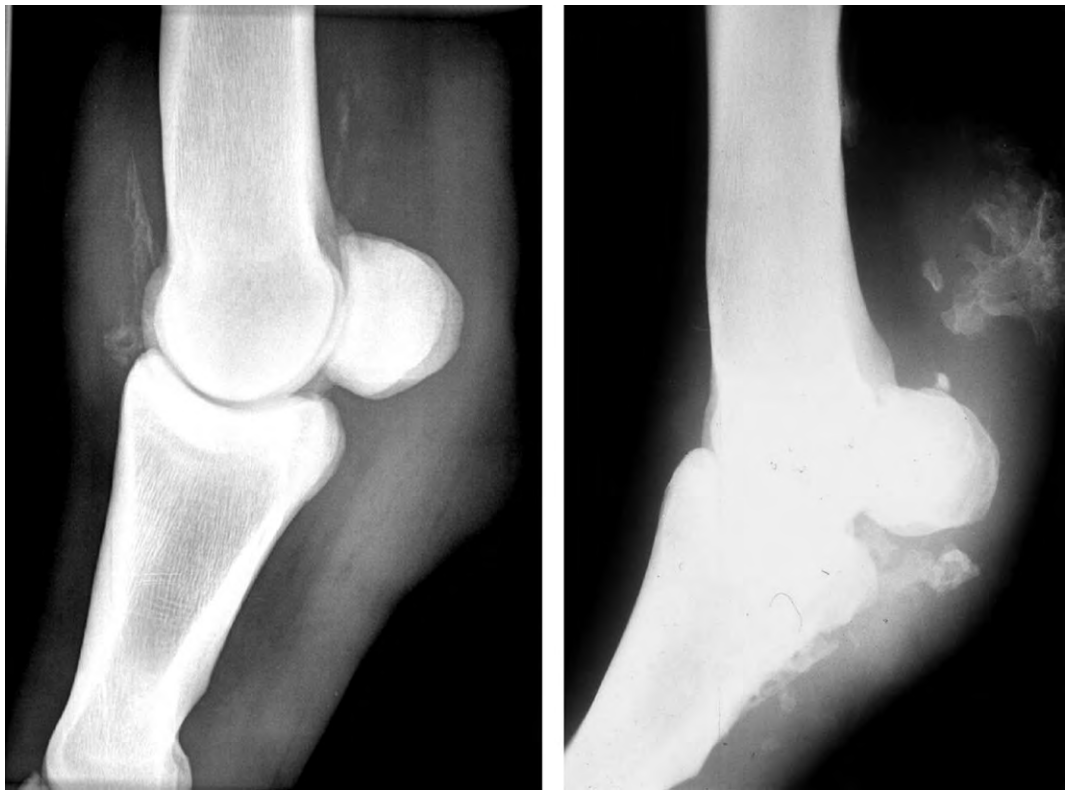


Figure 25.50 Soft-tissue mineralization around the fetlock joint.

- Metaplastic calcification can be seen in chronic soft-tissue injuries (Figure 25.50).
- Collateral ligament damage can be appreciated as unilateral narrowing of the joint space.
  Medial/lateral luxations/subluxations of the fetlock joint occur associated with damage to medial or lateral collateral ligaments, and are often associated with displaced avulsion fractures of proximal aspect of the proximal phalanx.
  Confirmation of collateral ligament injury can be sought by obtaining stressed dorsopalmar radiographs with the limb abducted and adducted – which will result in an abnormal widening of the joint space on the affected side.
  Avulsion fractures and/or entheseophytosis at the insertion of the collateral ligaments may be seen on the dorsopalmar projection.
- Dorsal subluxations often involve the proximal interphalangeal joint, and are associated with damage to the supporting structures of the palmar aspect of the limb.

- Cystic lesions.
  - Subchondral cystic lesions can be identified in the distal metacarpus and proximal phalanx.
  - A communicating tract may be identified connecting the cyst with the joint and osteoarthritis is sometimes present concurrently.
  - Radiographic changes are variable:
    An indentation or saucer-shaped lesion in the subchondral bone.
    Oval or circular radiolucency, with or without a sclerotic rim.
- Angular and flexural deformities of the fetlock and pastern region.
  - Angular limb deformities of the fetlock are best assessed in the dorsopalmar view where measurements can be made of the deviation angle.
  - Physitis, alone or associated with angular deformity, is represented by irregular thickness to the physis and surrounding sclerosis.
  - Flexural deformities can be identified in the lateromedial projections.

Carpus

See Chapter 17.

Indications

- Conditions of this region are mainly seen in young athletic horses performing at high speed, often as the result of exercise-induced trauma.
- Soft-tissue swellings: joint, tendon sheath or bursa effusion.
- Bony swellings: carpal or metacarpal bones.
- Carpal deformities: angular or flexural.
- Carpal wounds.
- Positive regional or intra-articular analgesia.
- Premature/dysmature foals to assess ossification status.
- Prepurchase/presale radiographs.

Equipment

- Portable X-ray machine is sufficient.
- Cassette holder, either free-standing or hand-holder with long handle.
- Lead gowns, thyroid protectors, gloves.

Preparation

- Area to be examined should be brushed to remove any dirt.
- Patient should be adequately sedated and be standing on an even surface while weight bearing evenly on all four limbs with the cannon bone vertical.

Radiographic projections

- Standard carpus series.
  - Lateromedial (LM).
  - Dorsopalmar (DP).
  - Dorso 30° lateral-palmaromedial oblique (DLPMO) projection.
  - Dorso 30° medial-palmarolateral oblique (DMPLO) projection.
  - Flexed lateromedial.
- Additional projections:
  - Dorsoproximal-dorsodistal oblique, 'skyline' projections at varying angles to highlight the distal radius, the proximal and distal row of carpal bones, mainly indicated in horses performing at high speed.

Technique

Projections are similar to fetlock just centred on the carpus, see Figures 25.42, 25.44, 25.46 and 25.47).

- Dorsopalmar projection.
  - Use a horizontal beam and centre on the midcarpal joint. An easy rule of thumb is to aim the central beam 2 cm distal to the tip of the accessory carpal bone.
  - Position the X-ray machine dorsal to the carpus.
  - Position the cassette on the palmar side of the carpus, as close as possible to the horse and aligned perpendicular to the X-ray beam.
  - A good DP radiograph is characterized by a prominent radiolucent space between the intermediate and radial carpal bones.
- Lateromedial projection.
  - Use a horizontal beam and centre on the midcarpal joint.
  - Position the X-ray machine lateral to the carpus.
  - Position the cassette on the medial side of the carpus, as close as possible to the horse and aligned perpendicular to the X-ray beam.
  - A good LM radiograph is characterized by the absence of any superimposition of bony structures at the level of the ulnar carpal bone-accessory carpal bone articulation.
- Flexed lateromedial projection:
  - Gloved assistant holds the foot up to achieve maximal flexion of the carpus.
  - Do not pull the distal limb laterally or medially, and do not rotate the limb, to avoid undesirable obliquities.
  - Proceed like a weight-bearing LM.
- Dorsolateral-palmaromedial oblique projection:
  - This highlights the most commonly affected dorsomedial parts of the radial and third carpal bones.
  - Position the X-ray machine dorsolateral to the carpus at an angle of 30° to the lateromedial view.
  - Centre on midcarpal joint, use a horizontal beam.
  - Position cassette perpendicular to X-ray beam on the palmaromedial aspect of the carpus.
  - Further oblique projections at different angles may be necessary to highlight lesions, especially fractures.
- Dorsomedial-palmarolateral oblique projection:
  - Highlights the intermediate and ulnar carpal bones and the lateral part (intermediate facet) of the third carpal bone.
  - Position the X-ray machine dorsomedial (or palmarolateral) to the carpus at an angle of 30° to the mediolateral axis.
  - Centre on midcarpal joint, use a horizontal beam.
  - Position cassette perpendicular to X-ray beam on the palmarolateral (dorsomedial) aspect of the carpus.
- Dorsoproximal-dorsodistal oblique 'skyline' projections.
  - These projections provide extra information about the localization of chip fractures, the alterations in bone density of the articulating facets of the different bones, peripheral new bone deposits and the presence of sagittal bone defects that cannot be visualized on other projections.
  - The carpus should be flexed to the greatest possible degree and brought cranially as much as possible.
  - A cassette is held in the horizontal plane along the long axis of the cannon bone.
  - The central beam should be directed in the sagittal plane of the limb. The radius can serve as a guide to prevent obliquity.
  - Skyline distal radius (80° to horizontal): highlights the distal dorsal radius.
  - Skyline proximal row of carpal bones (55° to horizontal): highlights the dorsal margins of the radial, intermediate and ulnar carpal bones.
  - Skyline distal row of carpal bones (30° to the horizontal): highlights the dorsal margins of the second, third and fourth carpal bones; in this view the radial facet of the third carpal bone is particularly important.

Radiographic anatomy (Figures 25.51 to 25.54)

- The carpus is a complex structure which is formed by the radius, two rows of carpal bones (7–9) and the metacarpal bones.
- It has three horizontal joints (antebrachiocarpal, midcarpal and carpometacarpal joints) and various intercarpal joints between the cuboidal carpal bones.
- The two distal joints share the same joint compartment, the proximal joint is separate.
- Most movement occurs in the antebrachiocarpal joint.
- Vestigial remnants of the distal ulna may sometimes be seen as a thin radiodense line on the caudolateral aspect of the distal radius (Figure 25.54).
- A first carpal bone is seen in 30–50% of horses (Figures 25.16, 25.53). It may be unilateral. It is usually the size of a pea and found behind the second carpal bone. If it is in close association with the 2nd carpal and/or metacarpal bones, these adjacent bones may show lucencies within them.
- A fifth carpal bone is rare (<10% of horses) and should not be mistaken for a fragment.
- Small zones of fat are present in the joint capsule of the antebrachiocarpal joint and the capsule of the tendon sheath of the extensor carpi radialis muscle (Figure 25.51). They can be identified radiographically as double irregular radiolucencies dorsal to the carpus. Obliteration or loss of the double shadow of these radiolucencies indicates the presence of excess fluid in the radiocarpal joint.
- The transverse radiopaque line in distal radius seen on the DP radiograph is the remnant of the distal radial physis. Bony ridge across the distal caudal aspect of the radius (remnant of physis) can have roughened, prominent appearance.
- Distal to the physis there is a large semicircular depression (synovial fossa) in the caudal radial cortex, which has the radiographical appearance of a well circumscribed lucency (Figure 25.52).
- The antebrachiocarpal joint and the midcarpal joints are uneven in width.

Figure 25.51 Radiographic anatomy of the carpus in a lateromedial projection. Note that the distal growth plate of the radius is still open.

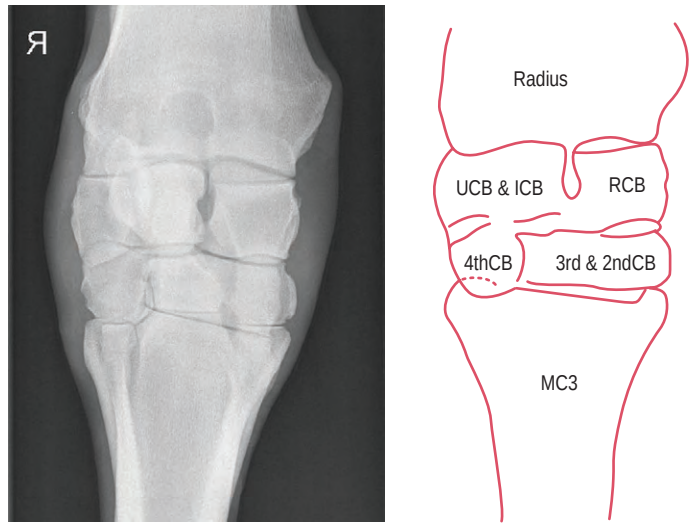


Figure 25.52 Radiographic anatomy of the carpus in a dorsopalmar projection.

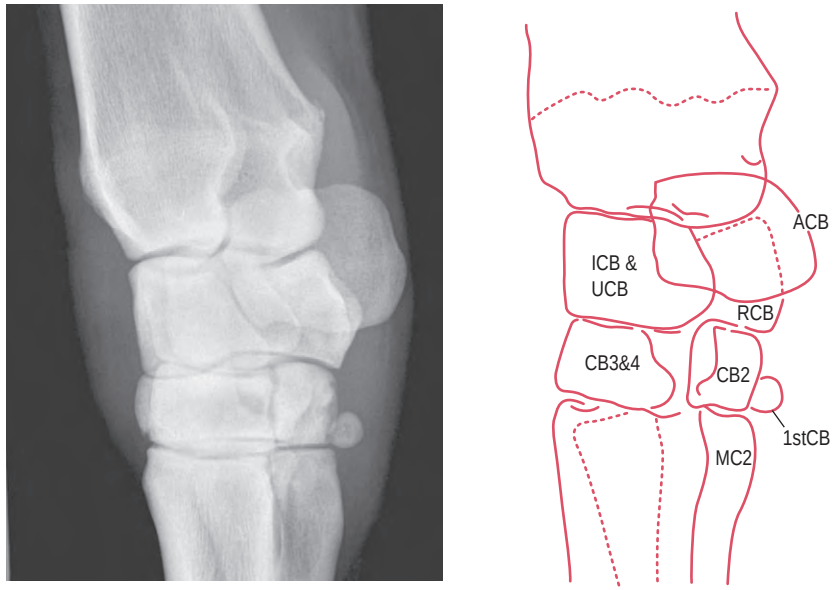


Figure 25.53 Radiographic anatomy of the carpus in a dorsolateral-palmaromedial projection.

- The interosseous spaces between the second, third and fourth metacarpal bones appear as irregular and multiple vertical radiolucent lines, sometimes further complicated by Mach lines.
- Occasionally the caudal radial cortex is irregular in outline, just proximal to the physis, at the level of origin of the superior check ligament.
- A faint radiolucent line may be visible on the lateral aspect of the distal radius representing a separate ossification centre.
- On the skyline projections each bone should have a smooth outline, with an even trabecular pattern and sharply defined corticomedullary junction.

Radiopathology

- Fractures.
  - Mainly in racing Thoroughbreds, less common in Standardbreds.
  - Intra-articular chip fractures: Fractures that involve one articular surface only. Occur at the distal radial carpal bone, third carpal bone, intermediate carpal bone. Radiography of the opposite carpus is advisable since 20% of cases present with bilateral chip fractures.
  - Slab fractures (Figure 25.16): Fractures that extend through the full thickness of a bone to involve both proximal and distal joint surfaces. These occur most commonly in the third carpal bone, but also in the intermediate and radial carpal bone.
  - Comminuted fractures: radial > intermediate > fourth carpal bone.
  - Accessory carpal bone fractures: These fractures occur most commonly in horses that race over fences. Bony union exceptional! Usually fibrous union with a persistent radiolucent line, but this is normally adequate for return to function.

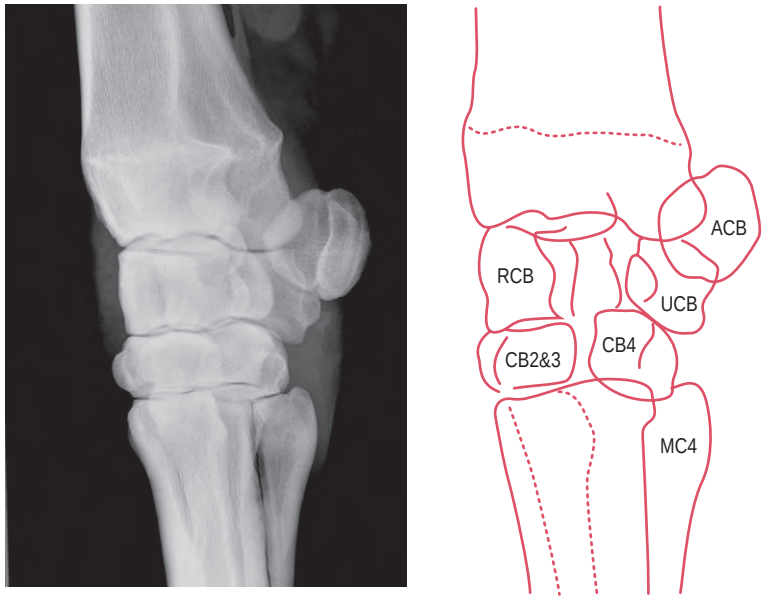


Figure 25.54 Radiographic anatomy of the carpus in a palmarolateral-dorsomedial projection.

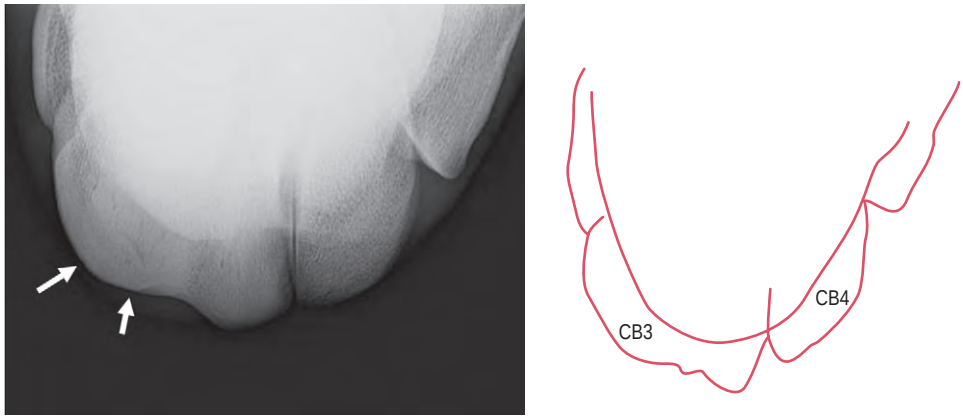


Figure 25.55 Sclerosis of the third carpal bone show on the 30° skyline as a loss of both trabecular structure and corticomedullary definition (white arrows).

- The fracture may only be visible on one particular oblique view.
- There may be more than one fracture line affecting more than one bone.
- The radiological abnormalities found must explain the degree of pain.

- Third carpal bone sclerosis: (Figure 25.55).
  - In young racehorses repeated loading on bones can lead to exercise induced trauma, the third carpal bone is most commonly affected.
  - This sclerosis of the third carpal bone can be recognized on the 30° skyline: it is appreciated as a loss of both trabecular structure and corticomedullary definition.
  - It is often bilateral.
  - It has been suggested that this condition precedes third carpal bone fractures, although similar patterns of increased radiodensity and loss of trabecular detail may be found as an end-stage of normal, exercise-induced remodelling processes with no actual clinical significance at the time.
  - The presence of any radiolucencies in the radial facet of the third carpal bone, however, is abnormal.

- Osteoarthritis.
  - Occurs predominantly in racing Thoroughbreds.
  - Often secondary to intra-articular fractures.
  - In the acute stage often no radiological abnormalities present.
  - Radiological abnormalities most commonly observed in the midcarpal joint:
    Joint space between distal radial carpal bone and proximal third carpal bone narrowed.
    Indistinct, slightly irregular outline to subchondral bone.
    Periarticular osteophytes.
    Saddle-shaped remodelling of the distal dorsal aspect of the radial carpal bone.
    Supracondylar lysis associated with chronic distension of the antebrachiocarpal joint.
    The skyline projections may show alteration to the contour of the margins of the carpal bones due to periosteal new bone proliferation.
    Degenerative changes in the carpometacarpal joint may be limited to the articulation between the second carpal and second metacarpal bone or the fourth carpal and fourth

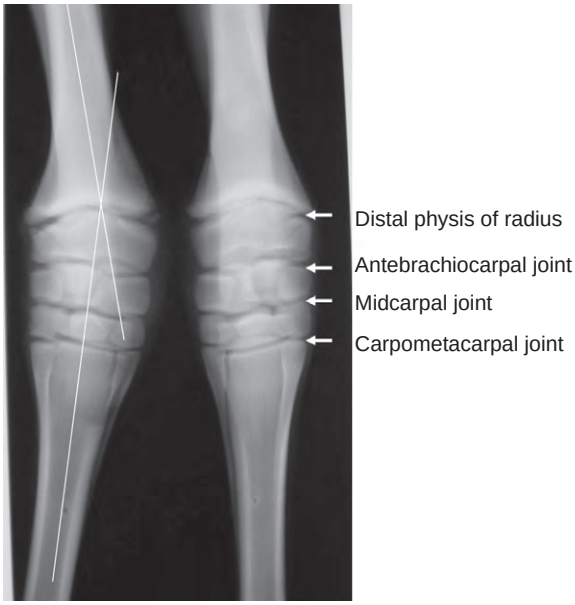


Figure 25.56 Carpus valgus deformation in a foal. The lines through the middle of the radius and metacarpus identify the distal radial physis as the pivot point.

metacarpal bone. These changes are often seen in association with periostitis of the splint bone on that side.

- Carpitis/capsulitis.
  - This term is used to refer to new bone formation on the dorsal aspect of one or more carpal bones, not involving the joint margins.
  - This can be either:
    entheseophytes, that form in association with tearing of the intercarpal ligaments. This is often seen in association with other changes, indicating the concurrent presence of OA, or periostitis due to direct trauma to the periosteum.
  - Often there is no long-term clinical importance if the exostosis remodels to show a smooth, well defined outline.
- Physitis and angular limb deformities (Figure 25.56).
  - Distal radial growth slows down at 12 months of age and stops at 18 months. Radiographic closure of the physis does not occur until 2 to 3 years of age.
  - Physitis is common in rapidly growing yearlings, often with pain and swelling at the level of the physis.
  - Two routine views are taken (dorsopalmar and lateromedial).
  - Identification of morphological changes to the physeal area:
    widening of the physis.
    ill defined margins to the physis.
    flaring of the metaphysis.
    flaring of the epiphysis.
    sclerosis of the metaphysis adjacent to the distal radial physis.
    cortical tunnelling in the metaphysis due to increased bone remodelling.
  - Identification of morphological changes associated with angulation:
    wedging of the epiphysis.
    changes in the shape of the carpal bones: incomplete ossification (round, small), wedging, collapse, fracture, subluxation.
    shortening of the proximal part of the splint bones, with widening of the corresponding joint space.
    delayed development of the lateral styloid process of the radius.
  - Identification of the centre of deviation (pivot point) (Figure 25.56).
    Draw two vertical lines through the centres of the radius and metacarpus on a DP radiograph.
    The point of intersection between these two lines forms the point of deviation; this allows identification of the site of origin and measurement of the degree of angulation.
    Pivot point growth plate = asynchronous metaphyseal growth.
    Pivot point epiphysis = asynchronous epiphyseal growth.
    Pivot point in carpal joint: cuboidal hypoplasia of carpal bones.
    Pivot point distal to intercarpal joint: splint bone hypoplasia.
  - When the pivot point is difficult to identify, more than one site may be contributing to the angular deformity!
  - Dysmature/premature foals should be radiographed shortly after birth to check whether ossification of the cuboidal bones (carpus and tarsus) is complete. These bones should be approximately cuboidal. Rounded margins indicate incomplete ossification and increased vulnerability to trauma and permanent deformation.
- Subchondral cyst-like lesions/carpal bone cysts:
  - Frequently incidental findings unassociated with lameness.
  - The larger cysts, adjacent to an articular surface are more likely to be of clinical significance.
  - Distal radial epiphyseal cysts: usually communication with antebrachiocarpal joint.
  - Cystic lucencies in second carpal bone are often incidental in association with the presence of first carpal bone.
- Proximal metacarpal syndrome:
  - Proximal metacarpal disease, proximal metacarpal syndrome and high suspensory disease are terms used to describe lameness referable to the proximal, palmar third of the metacarpus. Lameness in this area can be due to several different disease processes that are often impossible to be differentiated from one another:
    Avulsion fractures of the proximal palmar cortex.
    Fatigue damage and stress fractures of the proximal palmar cortex.
    Tearing of Sharpey's fibres (overextension injury?).
    Desmitis of the proximal part of the suspensory ligament.
  - Radiographic changes:
    Subtle changes to trabecular pattern of proximal palmar cortex (remodelling).
    Faint radiolucent line can reflect incomplete stress fracture.
  - Scintigraphy: to recognize increased bone remodelling.
  - Ultrasonography: to identify changes to proximal suspensory ligament.
  - MRI.
- Splints (Figure 25.57)
  - A splint is any bony enlargement (exostosis) involving the 2nd or 3rd metacarpal or metatarsal bones.

The elbow

Indication

- Wounds.
- Swellings: capped elbow, joint effusion, trauma.
- Suspected fractures: dropped elbow, severe lameness, crepitus.
- Lameness localized to the elbow by intra-articular analgesia.

Figure 25.57 This dorsomedial-palmarolateral radiograph of the metacarpal region shows an enlargement (exostosis) involving the 2nd metacarpal bone, commonly referred to as splint.

Equipment

- Depending on size of horse and indication, portable machine may be sufficient. For example if the presence of a fracture needs to be confirmed in the field.
- Due to the expertise and equipment required, it is advisable to perform radiography of the elbow in a hospital setting.
- Mounted cassette holder or holder with long handle.
- Lead gowns, thyroid protectors and gloves.

Projections (mediolateral and cranio-caudal) – technique

- Mediolateral projection (Figure 25.8)
  - The leg of interest is pulled forward with the carpus flexed, the radius horizontal and the shoulder extended, to move the elbow away from the trunk.
  - The cassette is positioned against the lateral aspect of the affected elbow by means of a ceiling/wall mounted cassette holder or a different holding device (e.g. shopping bag suspended from drip stand).
  - The X-ray machine is positioned on the unaffected side of the horse.
  - The X-ray beam is directed at the medial aspect of the joint, perpendicular to the long axis of the leg and centred on the joint.
  - Use of a grid is advisable to reduce scattered radiation; exposures need to be increased accordingly.
  - In some painful cases this technique is not possible and an attempt can be made to acquire a lateromedial radiograph. This often provides a satisfactory view of the olecranon which may be sufficient in cases of olecranon fractures. The cassette is positioned medial to the olecranon and pushed as far cranial as possible. The X-ray machine is situated lateral to the affected leg.
- Craniocaudal projection.
  - The leg is ideally weightbearing and may be pulled slightly in abduction (rope around foot). A rotated large cassette is pushed into the chest wall behind the olecranon. The X-ray beam is aimed at the joint and perpendicular to the cassette.

Radiographic anatomy

- The elbow joint is formed by the humerus proximally and the radius and ulna distally.
- The ulna is incomplete and fuses distally with the radius. Occasionally a complete ulna is observed.
- In addition to the main growth plates it has various ossification centres from which the anconeal, coronoid processes and the tuber olecrani ossify.
- The anconeal process of ulna can have variably rounded or sharply pointed appearance.
- The trochlear (semilunar) notch of the ulna is divided into a proximal articulating part and a distal non-articulating part by a transverse ridge. This is important to know to decide if a fracture is articular or not.
- The interosseus space between the ulna and the radius may be clearly or poorly defined, depending on the angle of projection.
- The proximal cranial margin of the radius can have several osteophyte-like projections. These 'lips' are normal and do not reflect the presence of OA.
- Proximal radial tuberosity (insertion biceps brachii) may appear irregular when viewed obliquely.
- On a CrCd projection the joint space is often uneven in width with the lateral joint space being narrower, shorter and more undulating than the medial one.

Radiopathology

- Fractures (Figure 25.8).
  - Olecranon fractures are most common. They occur mainly following external trauma (kick) or a fall. Horses typically show a dropped elbow appearance due to failure of the triceps brachi muscle.
- Subchondral cyst-like lesions.
  - The proximomedial aspect of the radius is most commonly involved, often in association with periosteal reaction at the radial insertion of the medial collateral ligament.
  - Occasionally cysts are found in the distal humeral condyles.
  - Often secondary OA.
- Osteoarthritis.
  - Periarticular osteophytes, entheseophytes (capsular and ligamentous attachments), and narrowing of the joint space.
  - Usually best shown on a craniocaudal projection.
  - Usually secondary to infection, subchondral cyst-like lesions, fractures.
- Penetrating joint injuries and infectious arthritis:
  - With any wound on the lateral aspect of the elbow region, there is a considerable risk of penetration of the joint capsule which has little soft tissue cover laterally.
  - Radiographic signs:
    The presence of gas lucencies over the joint may be an indication of joint sepsis.
    Injection of radiopaque contrast into the joint, in an area remote from the wound, may be necessary to establish whether communication exists between the joint and the entry wound.

The shoulder

See Chapter 17.

Indication

- Local trauma with regional swelling.
- Wounds.
- Lameness localized to the shoulder with intra-articular analgesia.

Equipment

- High-output generator needed to get adequate exposures.
- Large cassettes.
- Mounted cassette holder (suspended from the ceiling or drip stand).
- Grid for scatter reduction.
- Ideally aluminium dodger/wedge to compensate for different radiodensity on the cranial and caudal part of the joint.
- Lead gown, thyroid protector, gloves.

Projections

- Mediolateral.
- Craniomedial-caudolateral or caudolateral-craniomedial oblique.
- Cranioproximal-craniodistal projections allow to skyline the different tubercles of the cranial humerus.

Technique

- Mediolateral projection (Figure 25.12).
  - The limb is pulled as far forward as possible and the neck is kept in a straight position to try to superimpose the radiolucent trachea over the joint, which will increase visibility of some parts of the joint.
  - The cassette is positioned lateral to the leg of interest.
  - The X-ray machine is positioned on the other side of the horse.
  - The X-ray beam is centred approximately 10 cm in front of the distal aspect of the scapular spine of the weight-bearing limb. Use a skin marker for reference.
  - Tight collimation is essential to reduce scatter.
  - Adequate exposures for the humeral head and glenoid cavity often lead to overexposure of the greater, lesser and intermediate tubercles of the humerus in the cranial part of the radiograph (this can be avoided by using an aluminium dodger). If a dodger is not available it is often necessary to take two views of different exposures when conventional film-screen systems are used. When using CR or DR systems it is often possible to get round this (within limits) by post-acquisition adjustment of collimation and brightness/contrast settings.
- Craniomedial-caudolateral oblique (CrM-CaLO).
  - This projection highlights the cranial part of the greater and lesser tubercle, the intermediate tubercle, the intertubercular groove and the deltoid tuberosity.
  - The leg is pulled forward and the humerus is held in a vertical position.
  - The cassette is held caudal to the shoulder muscle mass.
  - The X-ray beam is centred at the level of the greater tubercle of the humerus. Palpate and mark with tape.
- Caudolateral-craniomedial oblique.
  - This view highlights the same anatomical structures as the previous one, but with a different degree of distortion since the cassette and the central beam are nearer perpendicular to each other.
  - The choice of view depends on the individual examiner.

Radiographic anatomy

- The shoulder joint is formed by the glenoid cavity of the scapula and the humeral head.
- The supraglenoid tubercle and the coracoid process are located on the distal end of the scapula.
- The proximal end of the humerus carries the greater (laterally) and lesser (medially) tubercle both of which have a cranial and caudal part in the horse. Situated between the two is the intermediate tubercle.
- The tendon of origin of the biceps muscle runs in the intertubercular groove. While the groove itself can be visualized radiographically with a skyline projection, the tendon can be imaged with ultrasound.
- The deltoid tuberosity is seen as radio-opaque structure distal to the tubercles. The caudolateral curve of the deltoid tuberosity should not be confused with a chip fracture.
- Most of these bony protuberances have their own ossification centres.
- An irregular shaped radiolucent area (<0.5 cm) in the middle of the glenoid cavity is seen in approximately 5% of horses.
- The joint space appears wider in the middle in some horses.
- A faint vertical radiolucent line between cranial and middle third of glenoid cavity representing the glenoid notch, the remnant of the fusion between different ossification centres.

Radiopathology

- Osteochondritis dissecans.
  - The caudal head of the humerus or the glenoid cavity are usually affected.
  - Radiographic signs are:
    Abnormal contour of joint surfaces (flattening, irregular), poor joint congruity.
    Diverging caudal articular surfaces.
    Subchondral lysis, often with a cyst-like appearance.
    Presence of dissecting osteochondral lesions uncommon in equine shoulder.
    Arthrography may identify dissecting cartilage flaps.
    Often signs of secondary OA.
- Osteoarthritis.
  - Periarticular osteophytes (craniodistal angle of the humerus, caudal margin of the humeral head).
  - Irregular joint space.
  - Subchondral radiolucencies.
  - Subchondral sclerosis.
  - Remodelling (rounding off) of the (normally sharpish) caudodistal angle of the scapula.
  - Secondary to primary conditions affecting the joint: OCD, intra-articular fractures, infectious arthritis.
- Fractures.
  - Scapula.
    - Supraglenoid tuberosity, scapular spine, scapular neck.
    - Often associated with sequestration with discharging sinus tract.
    - Tangential radiographs may be necessary to show fracture line/fragment.
    - More commonly seen in foals (being trodden on by mare) and polo ponies (regular shoulder contact).
  - Humerus.
    Deltoid tuberosity: following kicks often with open wound, in chronic stage sometimes bone sequestration with discharging sinus.
    Greater and lesser tubercle humerus: following local trauma, often in communication with bicipital bursa.

- Soft-tissue calcification:
  - Metaplastic calcification in chronic bicipital tendinitis, chronic bicipital bursitis.
  - Periostitis proximal cranial humerus and supraglenoid tuberosity, possibly in association with infectious bursitis.
  - Calcification biceps tendon, sometimes in association with fracture of the supraglenoid tubercle, or bilateral shoulder OA.
  - Dysplasia of the scapulohumeral joint (Figure 25.58).

Figure 25.58 Dysplasia of the scapulohumeral joint. This mediolateral radiograph of the shoulder joint shows a marked discrepancy between the glenoid cavity and the humeral head.

  - This condition has been reported in Shetland Ponies and Miniature horses.
  - May be accompanied by subluxation of the joint and/or degenerative joint disease.
  - Radiographic signs: joint incongruency, modelling of the articular margins of the glenoid cavity, mild subluxation, fragmentation of the ventral angle of the scapula.

The tarsus

See Chapter 17.

Indications

- Soft-tissue swellings.
- Bony swellings.
- Wounds.
- Tarsal deformities.
- Positive regional or intra-articular analgesia.
- Prepurchase/presales radiographs.
- Premature/dysmature foals to assess ossification status.

Equipment

- Portable X-ray machine is sufficient.
- Cassette holder, either free-standing or hand-holder with long handle.
- Lead gowns, thyroid protectors, gloves.

Preparation

- Area to be examined should be brushed to remove any dirt.
- Patient should be adequately sedated and be standing on an even surface while weight bearing evenly on all four limbs with the cannon bone vertical.

Radiographic projections (Figures 25.59 to 25.62)

- Standard tarsus series:
  - Lateromedial (LM).
  - Dorsoplantar (DP).
  - Dorso 45° lateral-plantaromedial oblique (DLPMO) projection.

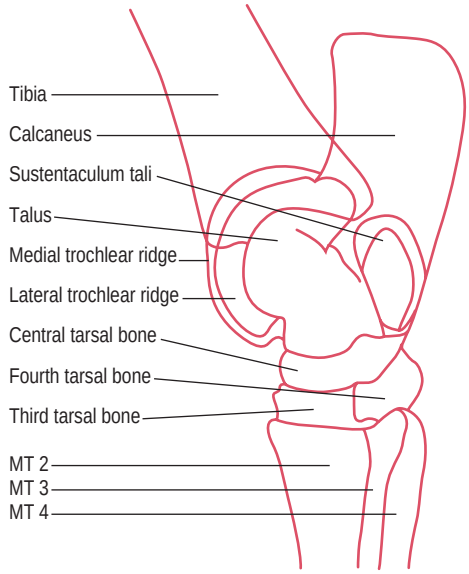


Figure 25.59 Radiographic anatomy of the tarsus on a lateromedial projection. Note that the chestnut is visible on the plantar aspect.

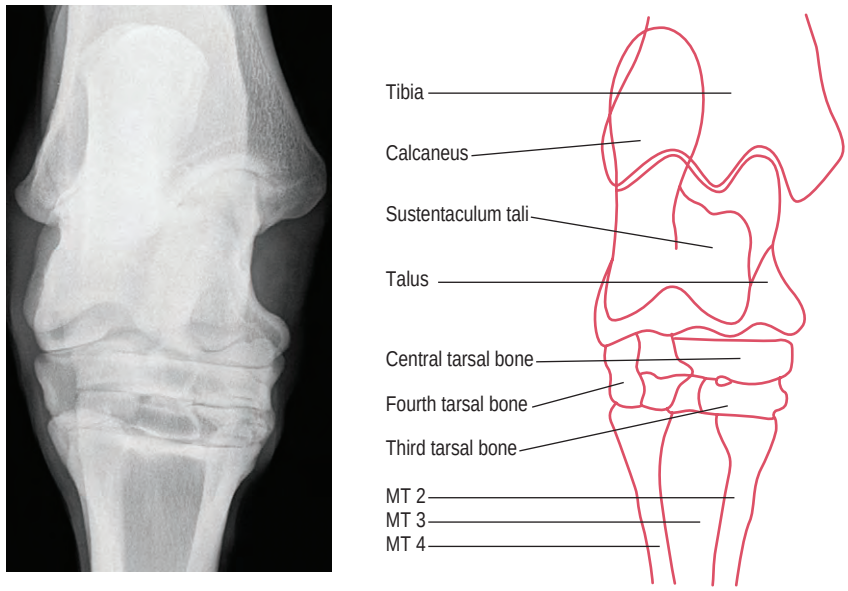


Figure 25.60 Radiographic anatomy of the tarsus on a dorsopalmar projection.

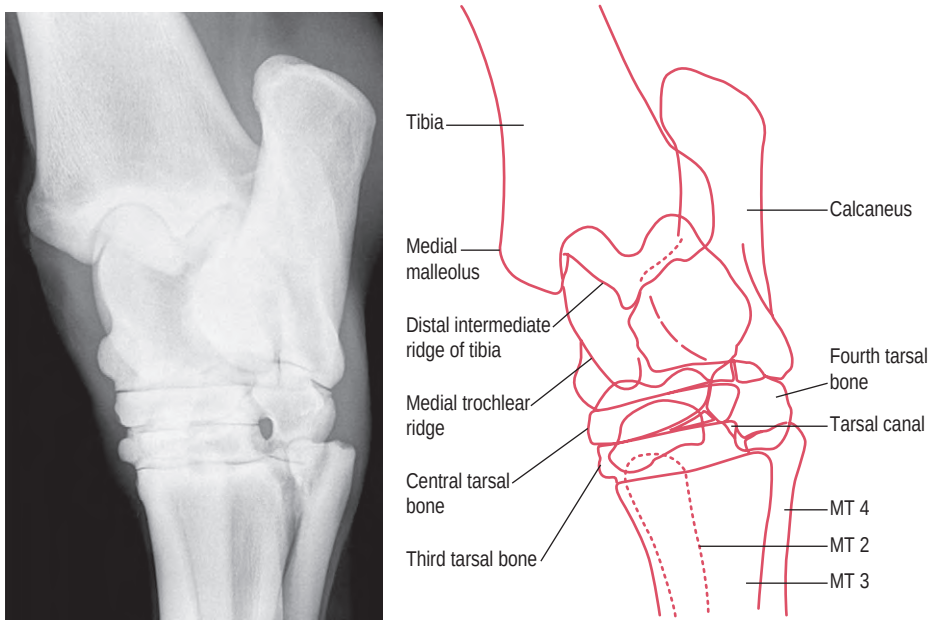


Figure 25.61 Radiographic anatomy of the tarsus on a dorsolateral-palmaromedial projection.

  - Plantaro 45°-lateral dorsomedial oblique (PLDMO) projection.
- Additional projections:
  - Flexed LM: this is useful for evaluating the caudal aspect of the distal tibia.
  - Flexed DP projection of the calcaneus and sustentaculum tali.
  - Various lesion-oriented obliques can be designed to look at pathology in individual animals based on findings in the 'standard' projections.

Technique

- Lateromedial projection (Figure 25.59).
  - This is particularly useful for evaluating the medial trochlear ridge of the talus, the calcaneus and the dorsal aspects of the proximal intertarsal, distal intertarsal and tarsometatarsal joints.
  - Position the X-ray machine lateral to the tarsus.
  - As many horses have a tendency to stand 'toe out', it is important that the lateromedial view is lateral to the axis of the limb and not the horse.
  - As the intertarsal joints are angled approximately 5–10° proximolateral to distomedially, angle the X-ray beam distally by about 8° and centre about10 cm distal to the point of the hock.
  - As an alternative the X-ray beam can be centred on the lateral malleolus of the tibia (which is easily palpable) which will result in an angled beam at the level of the intertarsal joints due to the diverging nature of the X-ray beam.

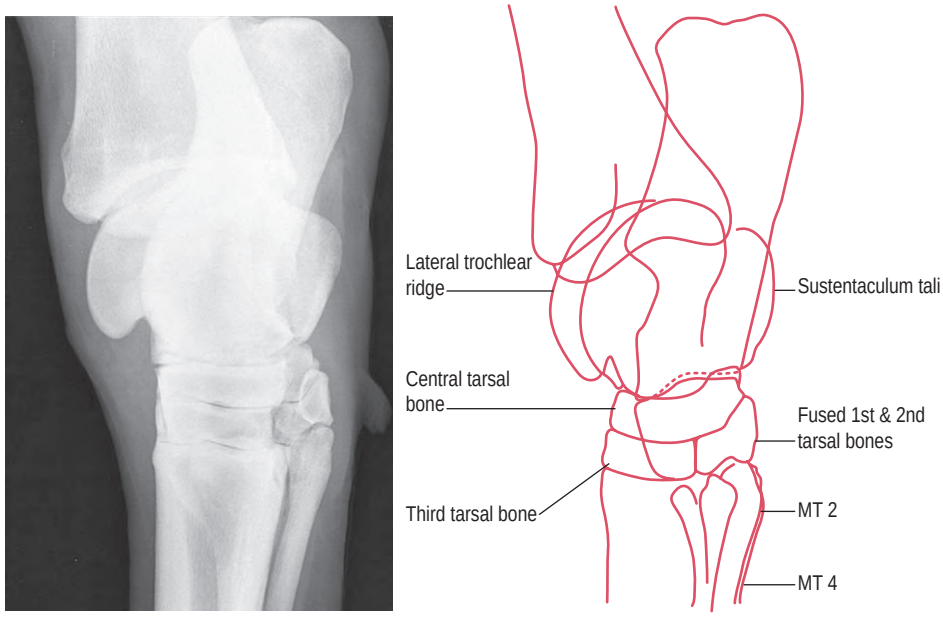


Figure 25.62 Radiographic anatomy of the tarsus on a palmarolateral-dorsomedial projection.

- Position the cassette on the medial side of the tarsus, as close as possible to the horse and aligned perpendicular to the X-ray beam.
- On a truly lateromedial radiograph the trochlear ridges and the second and fourth tarsal bones are not completely superimposed, since the medial trochlear ridge is larger and projects more dorsally than the lateral trochlear ridge and the head of the fourth metatarsal bone is larger than the head of the second and is positioned in a more plantar location.

- Dorsoplantar projection (Figure 25.60).
  - This is particularly useful for evaluating the medial aspects of the proximal intertarsal, distal intertarsal and tarsometatarsal joints. However, it is relatively difficult to produce well-aligned images of the distal tarsal joints in this view, compared to the DLPMO.
  - Position the X-ray machine dorsal to the tarsus. A true dorsoplantar view will need to take into account the 'toe out' nature of the hind limb of the majority of horses.
  - Centre on the central tarsal bone, depending on the angulation of the tarsus use a horizontal beam or angle the beam distally accordingly.
  - Position the cassette on the plantar side of the tarsus, as close as possible to the horse and aligned perpendicular to the X-ray beam.
- Dorsolateral-palmaromedial oblique projection: (Figure 25.61).
  - This is useful for evaluating the dorsomedial aspects of the proximal intertarsal, distal intertarsal and tarsometatarsal joints, the calcaneus, the fourth tarsal bone and the fourth metatarsal (splint) bone.
  - Position the X-ray machine dorsolateral to the tarsus at an angle of 45° to the lateromedial view.
  - Centre on the central tarsal bone, use a horizontal beam.
  - Position cassette perpendicular to X-ray beam on the plantaromedial aspect of the tarsus.
  - In a well positioned DLPMO, the dorsal opening of the tarsal canal should be clearly visible.
- Plantaro 45° lateral dorsomedial oblique (PLDMO) projection (Figure 25.62).
  - This is the view of choice for evaluation of the lateral trochlear ridge of the talus and the sustentaculum tali of the calcaneus. It also skylines the second metatarsal bone and the dorsolateral aspects of the proximal intertarsal, distal intertarsal and tarsometatarsal joints.
  - Position the X-ray machine plantarolateral (or dorsomedial) to the tarsus at an angle of 45° to the mediolateral axis.
  - Centre on central tarsal bone, use a horizontal beam.
  - Position cassette perpendicular to X-ray beam on the dorsomedial (palmarolateral) aspect of the tarsus.
- Flexed LM projection.
  - This is useful for evaluating the caudal aspect of the distal tibia.
  - The foot is elevated flexing the fetlock as well as the tarsocrural joint. The metatarsal bones are not parallel to the ground but instead are angled down approximately 30°.
  - The beam is centred dorsal to the calcaneus.
  - Care should be taken to ensure that the leg is not abducted.
- Flexed DP projection of the calcaneus and sustentaculum tali (plantaroproximal plantarodistal oblique).
  - This allows evaluation of the calcaneus and sustentaculum tali from a proximal to distal direction.
  - The foot is elevated and the tarsal joint is flexed with the metatarsal bones parallel to the ground.
  - The X-ray tube is positioned above the tarsus and the X-ray cassette placed nearly parallel with the ground. The X-ray beam is directed as close to vertical as possible.
  - Film-focal distance may need to be reduced, which causes a degree of magnification and exposure factors may need to be reduced accordingly.

Radiographic anatomy (Figures 25.59 to 25.62)

- The tarsus is a complex structure which is formed by the tibia, the talus and calcaneus, the central, second, third and fourth tarsal bones and the second, third and fourth metatarsal bones.
- It has four horizontal joints: the tarsocrural joint, the proximal and distal intertarsal joint and the tarsometatarsal joint.

- The two proximal joints share the same joint compartment; the distal joints are separate.
- Most movement occurs in the tarsocrural joint, which is a high-motion joint, whereas the distal tarsal joints are low-motion joints, mainly acting as shock absorbers.
- There are multiple vertically orientated, intertarsal joints between the different bones.
- The medial and lateral trochlear ridges should be smoothly curved but may be slightly flattened in the middle portion.
- The lateral trochlear ridge has a distinct notch in its distal extent.
- The central tarsal bone and third tarsal bone should be similar in height over their whole length.
- There should be a good definition between the cortex and medulla of the central and third tarsal bone.
- The mottled opacity of the torus tarseus (chestnut) may be seen on the plantar aspect of the fourth tarsal bone.
- The tarsus is the most complex anatomical structure (with maybe the exception of the head and the carpus) that is routinely radiographed in equine practice and the use of a skeletal specimen is often advisable to be able to distinguish between the different superimposed structures.

Radiopathology

- Osteoarthritis.
  - This affects both the high-motion (tarsocrural) joint and the low-motion (proximal intertarsal, distal intertarsal and tarsometatarsal) joints of the hock.
  - Tarsocrural joint: new bone formation is more limited compared to the low motion joints and will be recognized on the distal tibia in the lateromedial projection and on the medial malleolus in the dorsoplantar projection.
  - Distal intertarsal and tarsometatarsal joint (Figure 25.7):
    OA of these joints are called bone spavin.
    The low-motion joints will demonstrate variable degrees of new bone formation and subchondral bone destruction, usually best visualized in the DLPMO view.
    New bone formation is commonly observed on the dorsal aspect of the joints and/or obliterating the joint space.
    In cases of 'lytic' spavin the normal, smooth joint outline takes on an irregular, widened appearance.
    Subchondral bone sclerosis and loss of cortico-medullary definition of the central and third tarsal bone can be observed.
    The central and third tarsal bone can be wedge-shaped and irregular in outline.
- Infective arthritis.
  - In its early stages, this will be recognized as soft-tissue swelling alone; later, there will be variable degrees of destruction of subchondral bone, together with new bone formation around the joints. This will generally be more extensive and irregular than that related to degenerative joint disease.
- Osteochondrosis dissecans (Figure 25.7).
  - In the tarsus this affects the lateral trochlear ridge and the distal intermediate ridge of the tibia. Both locations are best evaluated in the PLDMO projection. Radiographic changes range from mild flattening of the ridge to large dissecting fragments.
- Cystic lesions.
  - These may be found in the small tarsal bones, as elsewhere.
- Fractures (Figure 25.16).
  - These take various configurations, and their clinical significance will depend on their effect on stability of the hock and the individual joints affected.
- Tarsal collapse.
  - In young foals, tarsal collapse involving the central and third tarsal bones may cause a 'curby-hocked' appearance, associated with dorsal movement of the proximal part of the calcaneus. This may also lead to slab fractures of the dorsal aspects of these bones.
- Dislocation.
  - This may affect any of the joints, but clearly luxation of the tibiotarsal joint is more of a disaster than luxations affecting the other joints.
  - Stressed DP views show an increase in joint space width on the affected side; care has to be taken during acquisition with collimation since these require an assistant to handhold the leg.
  - (Sub)luxations (Figure 25.13) are associated with disruption of the ligaments on the affected side and ultrasonography is recommended in these cases.
  - The superficial digital flexor tendon runs over the tuber calcanei and may slip off to the side. Radiographs are recommended to assess bony involvement.
- Angular deformities.
  - These are similar but less common than in the carpus. Lateromedial deviation are usually valgus and associated with the distal tibia.
- Tenosynovitis of the tarsal sheath.
  - The tarsal sheath envelops the deep digital flexor tendon as it runs over the sustentaculum tali.
  - In some animals, tenosynovitis of the tarsal sheath (thoroughpin) is associated with lameness. In these cases, the sustentaculum tali should be evaluated to look for roughening and new bone formation which is damaging the deep digital flexor tendon. Alternatively, a few cases of thoroughpin may be associated with ossification of the tarsal sheath, rather than new bone formation on the sustentaculum tali.
- Other synovial distensions.
  - Enlargement of various bursae, other tendon sheaths or the tibiotarsal joint can be assessed using contrast radiography if required. This is also useful for assessing communication between structures and leakage from them if there is an overlying wound.
- Calcinosis:
  - Although classically associated with the shoulder and stifle joint in the horse, calcinosis can be seen as circular or oval granular radio-opaque deposits adjacent to the tarsus.

The stifle

See Chapter 1.

Indications

- Joint effusion.
- Wounds.
- Positive intra-articular analgesia.
- Prepurchase/presales radiographs.

Equipment

- Due to the equipment and personnel required stifle radiographs are easier performed in a hospital setting than in the field.
- Portable X-ray machines are sufficient in smaller horses; however, especially the caudocranial projection requires higher exposures and a high-output generator is recommended.
- Large cassette.
- Lead gowns, thyroid protectors and gloves.

- Short handled cassette holder if available.
- Aluminium wedge/dodger beneficial to adjust exposures, which are lower for the femoropatellar joint than for the femorotibial joint.

Preparation

- Area to be examined should be brushed to remove any dirt.
- The majority of horses will need to be well sedated for stifle radiographs since most horses resent being touched between their legs.
- Patient should be standing on an even surface while weight bearing evenly on all four limbs with the cannon bone vertical.

Radiographic projections

- Standard stifle series:
  - Lateromedial (LM).
  - Caudocranial (CdCr).
  - Caudo30°lateral-craniomedial oblique projection (CdL-CrMO).
- Additional projections:
  - Flexed LM: This allows for better assessment of the femoral condyles and the proximal part of the trochlear ridges.
  - Cranioproximal-craniodistal oblique (Skyline view of the patella). This view highlights the patella and its position in the intertrochlear groove.

Technique

- Lateromedial (Figure 25.11).
  - Exposures differ for the femoropatellar and femorotibial joints unless an aluminium wedge filter is used to filter the incoming radiation over the cranial part of the joint to avoid overexposure. This is less of an issue with CR and DR system where post-acquisition adjustment is possible. Alternatively two radiographs have to be taken.
  - A large cassette is advanced into the inguinal region medial to the stifle. The cassette should remain as parallel to the sagittal plane of the stifle as possible (which is usually rotated slightly outward).
  - The use of large cassettes, lead gloves and tight collimation of the X-ray beam help to avoid the presence of gloved fingers in the primary beam. Short-handled cassette holders can be used.
  - The X-ray beam is centred on the femorotibial joint, just proximal to the tibial crest, 10 cm caudal to the cranial aspect of the limb, and is kept as perpendicular as possible to the stifle.
- Caudo 30°lateral-craniomedial oblique projection.
  - A true lateromedial projection is often difficult to perform due to the inability to align the cassette truly with the sagittal plane of the limb. The caudo 30°lateral-craniomedial oblique view is a valuable alternative projection and allows for:
    Greater ease of cassette placement.
    Separation of the trochlear ridges and femoral condyles.
    Centred over the trochlear ridges, this projection provides a clear view of the femoropatellar joint, with lower exposures than needed for the full LM projection.
    Angling the beam 10° proximodistally increases the ease of cassette alignment further and separates the lateral and medial joint compartments slightly in a proximodistal direction.
- Caudocranial projection: (Figure 25.22).
  - The machine head is positioned behind the horse.
  - The X-ray beam is centred on the tibial tuberosity and angled down 10°. It is helpful to palpate the slope of the tibial plateau and outline its position with tape; the angle of the X-ray beam should follow this marker.
  - The cassette is held in front of the stifle, perpendicular to the incoming central beam.

Radiographic anatomy

- The stifle includes two synovial joints: the femoropatellar and the femorotibial joint.
- The femoropatellar joint is formed by the patella and the trochlear ridges of the femur.
- The femorotibial joint is formed by the tibial plateau and the femoral condyles.
- The femorotibial joint has a medial and lateral compartment, which communicate in most horses. The medial compartment also frequently communicates with the femoropatellar joint.
- The medial trochlear ridge is bigger than the lateral trochlear ridge and is more beak shaped (remember the patella 'hooks' over the medial ridge when the horse engages its patellar locking mechanism).
- The lateral trochlear ridge is much smaller and rounded.
- Both trochlear ridges have an irregular outline with roughened edges in juvenile animals.
- In some horses a small area of flattening can be seen at the most distal extremity of the lateral trochlear ridge, at the level of its transition into the lateral condyle. A small area of radiolucency is often seen just proximal to this flattening. This is normal and should not be confused with osteochondritis dissecans of the lateral trochlear ridge.
- Fabellae (sesamoid bones associated with the origin of the gastrocnemius muscle on the caudal aspect of the distal femur) are extremely rare but may occur occasionally in the horse.
- On the caudo-cranial projection the patella is situated more laterally (bears on lateral trochlear ridge).
- The lateral condyle femur is smaller and is more pointed distally.
- The medial condyle usually has a smoothly rounded contour, but some flattening of the weightbearing surface may be acceptable if the trabecular pattern of the subchondral bone is normal.
- There are two intercondylar tubercles (medial and lateral) projecting proximally from the tibial plateau into the intercondylar fossa, the medial being the bigger one. The cruciate ligaments do not originate from these, but right next to them.
- The lateral joint space is narrower than the medial joint space, although joint space width is affected by positioning.
- The fibula is situated lateral to the tibia and often shows radiolucent line(s) due to ossification from different centres, which often remains incomplete throughout the horse's adult life.

Radiopathology

- Osteochondrosis dissecans: (Figure 25.11).
  - Location of lesions: generally on the lateral trochlear ridge of the femur, more specifically centred at its middle third. Lesions of the medial trochlear ridge or the articular surface of the patella are much less common.
  - Lesions are often most easily detected on the Cr30°L-CdM Oblique projection.
  - Radiographic abnormalities:
    Flattening of the lateral trochlear ridge.
    Irregular outline of the lateral trochlear ridge.
    Undersized and flattened lateral trochlear ridge due to chronic remodelling in longstanding cases.
    Focal radiolucencies within the lateral trochlear ridge.

Fragments attached within crater-like defects in the lateral trochlear ridge.
Free fragments with varying location in the joint on consecutive radiographs.
Irregular outline to the articular surface of the patella.
Osseous irregularity to the patellar apex, with/without fragmentation.
- Radiographic signs of secondary OA in chronic cases.
- Frequently bilateral: X-ray both sides.

- Subchondral cyst-like lesions: (Figure 25.12).
  - Predilection site is the medial femoral condyle.
  - Most easily detected on CdCr projections.
  - Radiographic abnormalities:
    Saucer-shaped to dome-shaped lucent area which is confluent with a flattened joint surface of the medial femoral condyle.
    Circular lucent area within the medial condyle with a thinner radiolucent 'neck' connecting it to the articular surface.
  - Other osseous cyst-like lesions in the stifle are relatively rare and occur in different locations on the proximal tibia and femoral condyles.
  - Explanations for these lesions have included osteochondrosis and trauma.
  - Cyst-like radiolucencies in the intercondylar area have been associated with chronic damage to the origin/insertion of the cruciate ligaments.
  - There is little correlation between clinical progress following either conservative or surgical treatment and the radiographic appearance of a cyst.
  - Cysts rarely fill in or change radiographically, but radiographic resolution is not necessary for a satisfactory clinical outcome.
  - Intense osteosclerotic margination around the cyst cavity indicates an attempt at demarcation of the affected area by the surrounding normal reactive bone, and indicates some degree of chronicity.
  - Vague, poorly defined loss of radiodensity with poor definition to the cyst margins indicates a more recent, active process with little 'healing' response from the surrounding bone.
  - Long standing lesions may be associated with signs of OA.
- Osteoarthritis.
  - The radiological signs of OA are usually less dramatic than the clinical picture and the prognosis.
  - Most commonly affected are:
    Femoropatellar joint secondary to chronic OCD.
    Medial femorotibial joint secondary to chronic cyst-like lesions or ligamentous trauma.
  - Femorotibial OA is best observed on the CdCr projection, femoropatellar OA is best observed on the LM projection.
  - Radiographic abnormalities:
    Periarticular osteophytes: patellar apex, medial border tibial plateau, intercondylar area.
    Subchondral lucencies.
    Enthesiophytes along the medial surface of the medial femoral condyle.
- Infectious arthritis (joint ill) in foals commonly localizes in the medial femorotibial joint. It is characterized by extensive osteolysis of the medial femoral condyle, and widening of the joint space.
- Soft-tissue injuries.
  - The complex of soft tissue injuries in the equine stifle is still poorly understood.
  - It is generally postulated that injuries of the medial meniscus, medial collateral ligament and anterior cruciate ligament go hand in hand.
  - There are often little or no radiographic abnormalities detectable initially. In horses suffering from meniscal damage a narrowing of the joint space may be observed. Generally with ligamentous damage there may be new bone formation or irregularities at the attachment site of the affected ligament.
  - Ultrasonography and arthroscopy are much better suited to evaluate the soft-tissue structures of the stifle! This is often performed after scintigraphy has identified the location (but not the nature) of the problem. Recently a method was described that allowed imaging of the stifle with CT in the anaesthetized horse.
- Fractures.
  - Patellar fractures: Skyline projection indispensable.
  - Chip fractures off the intercondylar tubercles: Sometimes in association with cranial cruciate ligament rupture.
  - Tibial tuberosity fractures: Caused by external trauma. Severity depends on the remaining attachment of the middle patellar ligament to the parent bone of the tibia.
  - Tibial stress fractures: Most common location proximolateral cortex of the tibia. May not be visible radiographically in acute stage (scintigraphy).
- Calcinosis circumscripta.
  - On the lateral aspect of the stifle, often in close association with the lateral femorotibial joint.
  - It consists of a soft tissue mass containing multiple amorphous areas of mineralization.

The pelvis

See Chapter 1.

Indications

- External asymmetry pelvis and quarters.
- Positive rectal examination: crepitus and asymmetry.
- Lameness with elimination of the distal limb, hock and stifle.
- Positive pelvic scintigram.

Equipment

- High output generator: exposure factors range from 70–150 kVp and 300–600 mAs.
- Large cassette.
- Grid.
- For recumbent horse: GA equipment, tunnel construction to allow positioning of cassettes under the horse, ropes to secure horse in frog-leg position.
- For standing horse: Ceiling mounted cassette holder that allows tilting.
- Handholding of cassette should be avoided due to the high exposures and scatter radiation involved!

Projections

- Ventrodorsal:
  - The commonly used projection is the ventrodorsal view.
  - This is generally performed under general anaesthesia, but with the appropriate equipment available, can also be done in the standing horse. Radiography under GA is more expensive, time consuming and carries the risk of the GA.
- Lateral views of limited diagnostic value due to superimposition and often poor quality.
- Oblique views (Ventral 45° left-dorsal right oblique).

Technique

- Recumbent ventrodorsal projection:
  - Horse under GA in dorsal recumbency with sagittal plane perpendicular to cassette-tunnel. Horse needs to be secured in the frog-leg position, so that no personnel need to be present during exposures. The best guide to ensure that positioning is correct is to assess the linea alba, which should be straight and lie vertically above the spine.
  - A tunnel construction must be available to allow the radiographer to advance the cassette dorsal to the animal's quarters without moving it (perspex). The cassette needs to be perpendicular to the sagittal plane of the horse and to the incoming central beam. The central beam is directed to the area of interest and the cassette positioned accordingly. Seven overlapping views are recommended for a full radiographic examination of the pelvis: 3 midline views, 2 tuber coxae views, and 2 tilted acetabulum views.
  - A grid or double grid is essential to eliminate scattered radiation. Other methods of minimizing this are, the use of an air gap between the object and the cassette, a lead intensifying screen in front of the cassette, and a lead sheet behind the cassette.
- Standing ventrodorsal projection:
  - The technique involves positioning the X-ray tube ventral to the horse's abdomen in front of its hind leg. The cassette and grid are sat on top of the animal's quarters following the inclination of the pelvis. This can be held in place with a long-handled cassette holder or alternatively with a fixed bucky which can be rotated to accommodate the incoming X-ray beam. Similar exposures as in the recumbent technique are used.
  - An angle of 20° to 25° to the vertical produces a good view of the ilial shaft. A 30–35° angled beam produces good images of the acetabulum and caudal pelvis.
  - Sedation may be necessary to control animals that are difficult to handle.
  - A limitation of this technique is the need for sophisticated technical equipment: a tilting, ceiling mounted cassette holder, a high-output generator and above all an X-ray tube which can be moved significantly sideways from the upright from which it is suspended. This is necessary to enable positioning of the tube ventral to the abdomen and sufficiently close to the midline of the body.
  - The standing view produces a more oblique view of the pubis and obturator foramen and a different projection of the femoral neck. The caudal pubis is often obscured by caudal abdominal contents. A better view of the cranial acetabular rim and the joint space is obtained while weightbearing. The technique is also cheaper, less time consuming and less hazardous to the patient.
- Oblique projections (ventral 45° left-dorsal right oblique).
  - This projection gives a better view of the rim of the acetabulum, the hip joint and the proximal femur.
  - The affected side is tilted 45° towards the cassette. The central beam is vertical, perpendicular to the cassette and centred over the head of the femur on the affected side. The cassette lies horizontal.
- Lateral projections.
  - Lateral views of the pelvis are only practical in foals and small ponies.

Radiographic anatomy

- The horse has a deep, semicircular hip joint with a prominent acetabular fossa. A small area of mild radiolucency is often present within the femoral head near the insertion of the teres ligament (fovea capitis).
- The obturator foramina should be symmetrical.
- The sacroiliac joint is difficult to image, because it slopes at approximately 30° to the horizontal. Its location is suggested by the line of superimposition between the transverse process of the sacrum and the sacral wing of the ilium.
- Physeal closure:
  - Tuber coxae, tubera ischii, femoral epiphysis all closed by 2 years.
  - Symphysis pelvis is not fully closed until end of 6th year, and may remain evident throughout the horse's life.
  - The articulations between the transverse processes of adjacent lumbar vertebrae can be irregular in outline, with occasional lucent zones and areas of sclerosis in the subchondral bone. Sometimes fusion is seen. The clinical significance of these changes is uncertain.

Radiopathology

- Luxation:
  - 90% of cases suffer craniodorsal displacement of femoral head.
  - Rarely ventral displacement: femoral head beneath pubis or in obturator foramen (rectally palpable).
  - Loss of joint congruity.
  - Often concurrent acetabular fractures.
- Round ligament rupture:
  - In the horse, the round ligament, which consists of the ligament of the femoral head and its accessory ligament, can rupture without luxation of the coxofemoral joint.
  - Radiographic signs:
    Abnormal position of femoral head in acetabulum.
    Intra-articular avulsion fracture possible.
    Widening of joint space.
    Chronic: periarticular osteophytes, remodelling femoral head and acetabular rim.
- Osteoarthritis:
  - New bone formation around acetabular rim.
  - Flattening of the femoral head.
  - Irregular joint space: narrowing, widening.
  - Subchondral lysis and sclerosis.
  - Usually secondary to intra-articular fractures or round ligament rupture.
  - Mild changes are of equivocal significance (e.g. irregular outline cranial femoral neck).
- Fractures:
  - Physeal fracture/epiphysiolysis femoral head.
  - Pelvic fractures.
    Radiography can be attempted standing with diagnostic results.
    Radiography under GA (ventrodorsal) carries risk for exacerbation of clinical signs and displacement of fragments.
    Scintigraphy method of choice to localize lesion with subsequent ultrasonography to characterize lesion further.
  - Stress fractures of the ilium.
    Spontaneous fractures that occur during normal exercise in young racing Thoroughbreds.
    Fracture line usually enters the sacral wing of the ilium from its caudal margin, courses cranially in a sagittal plane, and often remains incomplete.

Diagnosis can be made by ultrasonography showing a clear discontinuity in the upper surface of the sacral wing of the ilium, with/without callus formation.
Scintigraphy shows an intense focal increase in radiopharmaceutical uptake just lateral to the tuber sacrale.

The head

See Chapters 1 and 5.

Indications

- Nasal discharge (uni-/bilateral; mucus; blood; pus or all three).
- Soft tissue or bony swellings.
- Clinical signs of dental problems: dysphagia, weight loss, halitosis, malocclusion, bony swellings.
- Trauma.
- Post-operative check for remaining fragments of extracted teeth.

Equipment

- While portable X-ray machines are sufficient for the required exposures, head radiographs require some expertise and are much easier in a hospital setting with all the necessary equipment and experienced assistance.
- Large cassettes.
- Lead gowns, thyroid protectors, gloves.
- Short handled cassette holder if available.
- Head stand where the horse can rest its muzzle helps with keeping the head steady in a standardized position (Figure 25.25).
- Instead of handholding the cassette either a ceiling-mounted cassette holder can be used, or if not available the cassette can simply hung in a plastic bag from a drip stand.
- Blinkers or blindfolds often help to keep the horse quiet (Figure 25.25).
- Halter without metal parts are recommended to avoid superimposition of metal on area of interest (Figure 25.25).
- Sedation.

Projections

- Standard projections:
  - Different projections and exposures are required for the different anatomical structures of the head.
  - The most commonly radiographed structures are the paranasal sinuses and the teeth.
  - For these the standard projections are:
    Laterolateral (Figure 25.63).
    Dorsoventral/ventrodorsal.
    30° oblique projection for the maxillary cheek teeth.
    45° oblique projection for the mandibular cheek teeth.
- Additional studies:
  - Intraoral studies: to separate the upper and lower incisors, most commonly used for fractures and congenital abnormalities such as a wry nose.
  - Skyline projections: Lesion-oriented skyline projections may be indicated in cases of skull trauma and are especially helpful for the evaluation of depression fractures.
  - Contrast techniques: Contrast agents have been used in the investigation of discharging sinus tracts, occlusions of the nasolacrimal duct and for the identification of aneurysms in cases of guttural pouch mycosis.
  - Dynamic studies using a fluoroscope have been used in the investigation of pharyngeal and laryngeal function with or without contrast.

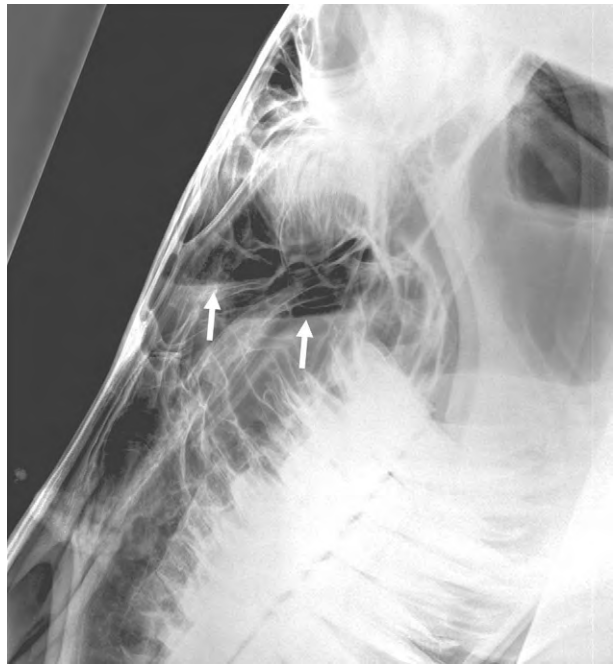


Figure 25.63 Lateral radiograph of the sinus area: there are two horizontal fluid lines with increased soft tissue opacity below it in the caudal maxillary and the dorsal conchal sinus indicating sinusitis.

Technique

- Laterolateral projection.
  - This allows visualization of fluid lines and soft tissue opacities within the paranasal sinuses (Figure 25.63) and alignment of the cheek teeth.
  - Rest the horse's muzzle on a stand, either with the nose as horizontal or as vertical as possible, this facilitates alignment of the X-ray beam.
  - Position the camera to the side of the horse. If there is a clinical indication as to which side may be affected, position the X-ray tube away from the affected side and the cassette next to the affected side.
  - Position the cassette as close as possible to the horse parallel to the sagittal plane and perpendicular to the X-ray beam.
  - Centre the X-ray beam in the middle of a line drawn from the eye to the rostral end of the facial crest, perpendicular to the sagittal plane.
  - Align your collimated area with the head and collimate to have all four margins on the cassette.
- VD or DV projections.
  - This is a technically challenging projection. Meaningful interpretation requires symmetry between left and right sides, and therefore care must be taken to avoid even slight tilting.
  - These views allow identification of the side affected by a lesion already identified in a maxillary sinus or a nasal chamber on the lateral projection and may give a better appreciation of its extent.
  - It is the only projection that allows differentiation between the rostral maxillary sinus and the ventral conchal sinus which are superimposed on a laterolateral radiograph.
  - It can be acquired as a DV or VD projection; this is often determined by how high or low the X-ray machine can be positioned.

  - In any case, position the horse's head on a head stand with the nose as horizontal as possible.
  - For a DV projection rest the mandible of the horse on the cassette.
  - Position the X-ray machine dorsal to the head.
  - Centre the X-ray beam on the midline of the nose, in the middle of a line drawn from the eye to the rostral end of the facial crest.
  - Align the X-ray beam perpendicular to the cassette.
- Oblique projections of the maxillary and mandibular cheek teeth.
  - This separates the left and right row of cheek teeth and allows the assessment of the roots of the cheek teeth of one side.
  - The cassette is positioned against the side of interest, care has to be taken to take the obliquity of the beam into account!
  - The X-ray machine is positioned on the opposite side, dorsally for the maxillary cheek teeth, ventrally for the mandibular cheek teeth.
  - The X-ray beam is angled at 30° from dorsal for the maxillary tooth roots or 45° from ventral for the mandibular tooth roots.
  - The beam is centred in the middle of the facial crest for the maxillary cheek teeth and the middle of the mandible for the mandibular cheek teeth.

Radiographic anatomy

- The equine skull is a very complex structure resulting in many superimpositions on radiographs.
- The horse has the following dentition in each arcade: 3 incisors, 1 canine (if male), 3 premolars, 3 molars. An additional small first premolar may be present as wolf tooth.
- The horse's teeth are constantly erupting throughout life, thus the reserve crown gets progressively shorter as the horse gets older.
- Dental eruption cysts in young animals (2–3 year olds) can be radiographically seen as radiolucent halos around the tooth roots of the mandibular cheek teeth and should not be confused with infection.
- The horse has the following paired paranasal sinuses:
  - Rostral maxillary.
  - Caudal maxillary.
  - Ventral conchal.
  - Dorsal conchal.
  - Frontal.
  - Sphenopalatine.
- As a guide the rostral maxillary is situated lateral to the ventral conchal sinus and they communicate.
- As a guide the rostral border of the rostral maxillary sinus is located dorsal and rostral to the third cheek tooth.
- As a guide the first two cheek teeth are not associated with a paranasal sinus. The middle two cheek teeth extend in the rostral maxillary sinus, the caudal two cheek teeth in the caudal maxillary sinus.
- As a guide the rostral maxillary and the caudal maxillary sinus are separated by a septum that is radiographically visible as a radioopaque line extending dorsally from the fourth maxillary cheek tooth.
- The facial crest is seen as a radioopaque line running rostrocaudally over the maxillary sinuses.
- The stylohoid can be appreciated overlying the radiolucent guttural pouch.
- There are several joints in the head (temporomandibular, temporohyoid, atlantooccipital joint and various non-synovial joints between the different skull bones). Their radiographic assessment is a challenge and requires special projections and other imaging techniques especially CT are usually better suited.

Radiopathology

- Fractures. These may involve the mandible, the flat bones of the skull and the hyoid bones, especially the stylohyoid.
  - Fractures of the mandible at the diastema may involve incisors or cheek teeth.
  - Lateral, DV, oblique and intraoral projections may all be required in order to accurately define the parts affected.
- Dental disease.
  - Laterolateral and oblique projections are used to identify fractures of tooth roots and also apical abscesses.
  - If the periapical abscess is associated with a discharging sinus, a metal probe can be used to confirm the relationship between the external opening and the diseased tooth root.
  - Radiographic signs of dental infection include:
    Loss of the lamina dura and widening of the lucency associated with the periodontal membrane.
    Reactive new bone around a well-defined lucency at the apex of the tooth.
    There may also be 'clubbing' of the tooth root.
  - Radiographs are not very sensitive in picking up periapical abscesses and complementary imaging techniques such as scintigraphy and CT may be required.
- Sinusitis (Figure 25.9).
  - Radiographic signs of sinusitis include: fluid lines and soft tissue opacities over the paranasal sinuses.
  - Sinusitis can be primary or secondary and the radiographs should be carefully evaluated for any signs of underlying disease such as dental problems, sinus cyst etc.
- Nasal conditions.
  - The commonest neoplasm affecting the horse's skull is the ethmoid haematoma.
  - This can sometimes be identified on the lateral view as a soft-tissue opacity in the region of the ethmoturbinates, but the outline of the orbit superimposed on this region should not be mistakenly diagnosed as a tumour.
  - Ethmoid haematoma is usually an endoscopic diagnosis, but those located relatively dorsal to the ethmoturbinates may be difficult to visualize endoscopically and occasionally these masses may be present in the maxillary sinus.
- Guttural pouches.
  - Endoscopy is usually the method of choice for evaluating the guttural pouches.
  - The main conditions affecting these structures are mycosis, empyema and tympany.
  - A mycotic lesion itself is usually not recognizable on plain films but the aneurysm may be revealed on contrast radiography.
  - Empyema may show as complete obliteration of the guttural pouch, or the presence of a fluid line, or a line with an irregular surface suggesting chondroids (solid pebble-like aggregations of pus).
  - Tympany will show as a gas-filled, enlarged guttural pouch.
  - The guttural pouches are also useful indicators of soft-tissue pathology in the retropharyngeal region. Masses in this region may cause compression of the guttural pouch.
- Diseases of the pharynx, larynx and palate. The method of choice for the assessment of these structures is endoscopy; radiography however may be helpful to determine the exact location and extend of a lesion if a soft tissue mass is suspected. The lateral projection may reveal evidence of neoplasia in the roof of the pharynx. Large soft-tissue masses may be indicative

of lymphosarcoma and erosions of the roof of the pharynx may indicate other forms of neoplasia. Defects and even complete absence of the soft palate may also be recognized in lateral projections.
- Dentigerous cyst. The presence of dental material adjacent to the base of the ear can be confirmed by a skyline view.
- Frontonasal suturitis. This condition is easily diagnosed clinically. Radiography will reveal the presence of new bone around the frontonasal and possibly adjacent sutures. It is usually a blemish which improves with time, but may, in some cases, involve obstruction of the nasolachrymal duct and epiphora.

The thorax

The equine thorax can be imaged but there are a number of limitations to radiography which make it less useful in horses than in small animals. Except for small foals, multiple films are required and, in the adult horse, this essentially means four views. The thorax has a large diameter, and therefore small lesions may be missed. In addition, it is impossible to obtain dorsoventral projections in the adult horse, so areas of suspected pathology cannot be assessed in a second view. One manoeuvre which can be used, relating to the large size of the chest, is comparative left and right lateral projections. It may be possible to establish the site of the lesion by comparison of magnification in each projection. In addition, one projection may show a lesion much more clearly than the other.

Indications

- Suspected pulmonary, mediastinal and cardiac disorders: abnormal lung sounds, coughing, nasal discharge, exercise intolerance, dyspnoea, tachypnoea.
- Suspected oesophageal disorders.
- Ultrasonography often better choice for cardiac and pleural disorders!
- Remember: Radiographic signs often lag behind the disease progress and do not correlate well with the severity of the disease!

Equipment

- High-output generator, stationary X-ray machine in conjunction with stationary grid/cassette holder.
- About 1 m of bones, muscle and lung tissue to go through:
  - High exposures needed to achieve necessary penetration.
  - Grid needed to reduce scatter (increases the exposure even more!)
  - Mounted cassette holder advisable to minimize exposure to personnel.
  - Remember: most scatter is on the side of the X-ray machine! So person taking the exposure should ideally leave the room or at least take a big step back!
- Breathing:
  - Short exposure times (and exposures go up again!) and fast rare earth screen-film combinations needed to prevent motion blur.
  - Inspiratory films to maximise air/tissue contrast.

Projections

Four projections (35 × 42 cm cassettes) per side needed to get complete picture:

- Caudodorsal: largest unobstructed view of the lung: assessment of lung fields and pulmonary vasculature, diaphragm.
- Craniodorsal: heart base, aorta, trachea, oesophagus, lymph nodes.
- Caudoventral: lung, caudal heart, vena cava, ventral diaphragm.
- Cranioventral: cranial lung, heart, mediastinum.

Technique

- Horse should stand with its legs extended forward to move some of the muscle mass out of the field of view.
- Detemine lung field: If in doubt about the size/extent of the lung field, percussion or auscultation can help!
- Place markers indicating the position of the four projections. The cranioventral view is usually achieved by centring on the middle of the triceps muscle mass.
- Procedure in the foal: much easier! Laterolateral and dorsoventral views should be performed. If foal was in lateral recumbency it should be brought in dorsal recumbency for a while (10 minutes) before radiographing to allow proper inflating of the previously squashed lung half.

Evaluating the quality of the radiograph

- Is there movement blur? This can be assessed by looking at the edges of the bones in the view, these should be sharply demarcated.
- Is the exposure adequate? This is best assessed by comparing soft tissue and bony opacities. Overexposure results in overly 'clear' impression of the lung field and may mask abnormal opacities. Underexposure may give the impression of an increased overall opacity.
- Is this an inspiratory or expiratory film? This can be assessed in the caudal films by looking at the position of the diaphragm. For the cranial films the overlapping lung area should have the same appearance as in the caudal inspiratory films.

Radiopathology

The radiographic image can be assessed in two ways. A 'geographical' approach, starting at one end of the film and working to the other, or starting peripherally and working inwards, is used for most parts of the horse. However, a systems-based approach is often more useful for the assessment of the thorax, running through the systems, as follows:

- Respiratory system – trachea, bronchi, lung, pleura/mediastinum, diaphragm.
- Cardiovascular system – heart, aorta, vena cava, pulmonary vessels.
- Gastrointestinal system – oesophagus, stomach.
- Lymphatic system – hilar lymph nodes.
- Musculoskeletal system – vertebrae, ribs, sternum.

Respiratory system. Radiographic changes can be categorized into:

- Diffuse overall.
- Diffuse localized.
- Solitary pulmonary lesions.

Diffuse disease: 4 basic lung patterns:

- Bronchial pattern.
- Interstitial pattern.
- Alveolar pattern.
- Vascular pattern.

Most commonly more than one pattern is present!
Bronchial pattern:

- Appearance: increased visibility of bronchial walls, 'Donuts', due to.
  - Mineralization of the bronchial walls, most commonly seen as an age-related change!
  - Thickening of the bronchus, associated with chronic inflammation.
  - Oedema/inflitration of the peribronchial tissues in acute allergic/inflammatory conditions.

- Early stage bronchopneumonia.
- Cardiac insufficiency.

Interstitial pattern:

- Appearance: Unstructured (hazy) and/or nodular, diffuse or localized, caused by:
  - Expiratory films – decrease in proportion of air to tissue density, common cause of misdiagnosis of interstitial pattern!
  - Interstitial pneumonia – diffuse interstitial infiltration with some discrete nodules.
  - Fungal disease; metastatic neoplasia: usually in combination with nodular lesions.
- In foals: immature patients, bronchointerstitial pneumonia.

Alveolar pattern:

- Appearance: patchy or diffuse, air bronchogram – air-filled bronchus surrounded by a relatively homogeneous soft-tissue opacity (fluid-filled alveoli), caused by:
  - Oedema: cardiogenic vs non-cardiogenic.
  - Consolidation in cases of pneumonia.
  - Haemorrhage.
  - Atelectasis: air in alveoli becomes replaced by fluid/soft-tissue opacity.
- Pleural disease. This may show as increased fluid or gas in the pleural cavity. The increased fluid is usually associated with pleurisy or pleural neoplasia. Pneumothorax may result from trauma to the thoracic walls and penetration to the pleural cavity. In the case of a pneuomothorax the edges of the lung lobes are visible on the radiograph.
- Diaphragm. Congenital abnormalities and ruptures may be seen and diagnosed from abnormal shadows created by viscera protruding into the thoracic cavity from the abdomen.
- Cardiovascular system. Cardiac failure and pericardial effusion may be recognized as an increase in size of the cardiac silhouette, and secondary pulmonary oedema may be seen as a consequence of cardiac disease.
- Gastrointestinal system.
  - Dilation of the oesophagus may be seen in cases of obstruction of the oesophagus with foreign bodies or impacted food material, and grass sickness.
  - Strictures may be a congenital problem, or associated with spasm/fibrosis following damage to the oesophagus from a foreign body.
  - Oral contrast studies using barium feed may be helpful to assess these cases further.
- Musculoskeletal system.
  - In the case of trauma to the thorax, rib fractures may be seen in association with other evidence of pulmonary and pleural pathology.
  - Congenital anomalies of vertebrae may be recognized on thoracic films, together with vertebral spondylosis and vertebral fractures.

The neck

Indication

- Horse showing signs of ataxia.
- Abnormal head or neck carriage.
- History/clinical signs of trauma to the neck.

Equipment

- Portable X-ray machines are sufficient in smaller horses and for radiographing the cranial neck in bigger horses; the more caudal projections require higher exposures and a high-output generator.
- Due to the equipment and personnel required, neck radiographs are easier to perform in a hospital setting.
- Mounted cassette holder (ceiling or drip stand).
- Grid.
- Large cassette.
- Head stand.
- Lead gowns and thyroid protectors.

Projections

- Standard projections:
  - Laterolateral C1–C3.
  - Laterolateral C3–C5.
  - Laterolateral C5–C6.
  - Laterolateral C6–T1.
- Additional projections.
  - A ventrodorsal/dorsoventral radiograph is only possible in the cranial neck.
  - Ventrolateral-dorsolateral oblique projections are helpful in the assessment of the articular facet joints.
  - Myelography is the method of choice to demonstrate spinal cord compression.

Technique

- For all of the above projections the horse should be positioned with its head on a stand with its head and neck extended.
- To reduce scatter a grid should be used.
- Palpation of the position/direction of the cervical spine and marking of the intended position of the cassettes with tape is helpful.
- The beam is orientated perpendicular to the neck for the lateral projections. True lateral projections are vital; any rotation will impair diagnostic quality.

Radiographic anatomy

- The equine cervical spine comprises seven vertebrae. Due to its complex structure it is helpful to have a neck specimen at hand to differentiate between the superimposed structures on radiographs.
- The first, second, sixth and seventh are very distinct in their anatomy, whereas the third, fourth and fifth look very similar.
- The atlas is ring shaped, the axis has a marked dorsal crest, the sixth vertebra has a double ventral crest and the seventh is much shorter than the rest.
- The vertebrae develop from several ossification centres and a thorough knowledge of the location and closure times of these is essential, so not to interpret those as fractures.
- The vertebrae articulate with each other forming the facet joints dorsally. The caudal edge of the joint is usually radiographically visible as a smoothly outlined, well defined linear lucency. These joints are orientated obliquely and overlap for the most part on the standard lateral projection, so mild osteoarthritic changes of these joints are difficult to appreciate radiographically. Most horses show some degree of OA changes, especially of the caudal facet joints, as they grow older, usually without any clinical significance.
- The dorsal and ventral border of the vertebral canal should be continuous without any step or angle formation.
- The cranial and caudal ends of the vertebrae should be similar in dimensions.

Radiopathology

- Spinal cord compression.
  - The spinal cord itself and hence spinal cord compression cannot be visualized on plain radiographs!

  - Radiographs can only show bony changes that may indicate compression of the spinal cord. Radiographic changes that may be associated with spinal cord compression are:
    Narrowing of the spinal canal caused by malformed vertebrae.
    Step and angle formation between adjacent vertebrae. Mild malalignment may be positional!
    Marked new bone formation extending into the spinal canal.
  - Inter- and intravertebral ratios have been suggested as radiographic measure of spinal cord compression: e.g. the width of the spinal canal at its widest part should be more than 50% of the width of the vertebral body.
- Osteoarthritis of the facet joints.
  - Radiographic signs include new bone formation, obliteration of the joint space, enlargement and/or deformation of the articular processes, subchondral bone sclerosis.
  - Radiographic signs of OA are often observed in the more caudal joint in older horses without clinical significance. However, extensive new bone formation extending into the spinal canal may cause spinal cord compression.
- Congenital or developmental stenosis of the vertebral canal caused by vertebral malformation.
  - 'Funnelling': narrowing of the cranial part of the vertebral foramen in relation to the caudal part.
  - 'Endplate flaring': abnormally shaped and enlarged caudal vertebral epiphyses.
  - Caudal extension of the dorsal arch of the vertebral foramen.
- Other conditions affecting the equine neck are fractures, neoplasia, abscesses and congenital abnormalities such as atlanto-occipial malformation in Arab foals.

Myelography

Myelography is at the moment considered 'gold standard' for the diagnosis of spinal cord compression in the live horse. The method of choice in humans and small animals is MRI, this is, however, not possible in the horse due to size restrictions.

Myelography is performed in the anaesthetized horse. A non-ionic iodine-based contrast medium is injected into the subarachnoid space and a series of normal, flexed and extended lateral radiographs is acquired. This results in dorsal and ventral contrast columns outlining the spinal cord. Several parameters are used to evaluate myelograms, but one commonly used indicator for spinal cord compression is a 50% reduction in height of the dorsal contrast column in relation to its greatest height.

The back

See Chapter 18.

Indication

- Clinical signs of back problem.
- Positive regional analgesia.
- Positive scintigraphic findings.

Equipment

- High-output X-ray generator.
- Mounted cassette holder.
- Grid.
- Aluminium dodger or wedge to compensate for different thickness of overlying soft tissues.
- Lead gowns and thyroid protectors.

Projections

- Laterolateral projections: spinous process and vertebral bodies.
- Ventrolateral-dorsolateral oblique projections: facet joints.

Technique

- Laterolateral projections (Figure 25.9).
  - Allows the assessment of the dorsal spinous processes and the vertebral bodies, but require different exposures and different centring. It is usually advisable to acquire separate radiographs for optimal image quality for both structures.
  - Spinous processes and vertebral bodies of the second and third thoracic vertebrae and caudal to the third lumbar vertebra and the vertebral bodies of the fourth to the eighth thoracic vertebrae cannot be imaged, hence radiography of the whole vertebra is limited to the ninth thoracic to the third lumbar vertebrae.
  - Markers are helpful to indicate which vertebra to allow identification of the individual vertebrae on the radiograph.
  - The X-ray beam is centred approximately 10 cm ventral to the skin surface for the dorsal spinous processes and 20 cm for the vertebral bodies.
  - A horizontal beam is used and collimated so that all edges of the field of view are visible on the cassette.
- Ventrolateral-dorsolateral oblique projections.
  - Allows the assessment of the facet joints.
  - The X-ray beam is centred 20 cm ventral to the skin surface in the dorsal midline.
  - The X-ray beam is angled 20° in a ventrodorsal direction.

Radiographic anatomy

- The top of the thoracic spinous processes have separate ossification centres which remain united with the rest of the process throughout life in many horses.
- The thoracic spinous processes are inclined caudally to T15 which is straight (also called the anticlinal veterbae), the remaining thoracic spinous processes are inclined cranially.
- The space between the spinous processes can vary in width, with some coming into contact with each other ('kissing spines'). This is of variable clinical significance.

Radiopathology

- 'Kissing spines' (Figure 25.9).
  - The inclination of spinous processes becomes flatter and the space between adjacent spinous processes is narrowed with the bones touching in advanced cases.
  - The spinous processes can show new bone formation, change in shape, sclerotic rims and lucencies.
  - A large proportion of horses without any clinical signs of back problems have 'kissing spines' and the clinical significance has to be established through regional anaesthesia.
- Spondylosis: new bone formation on the ventral aspect of the vertebral bodies bridging between adjacent vertebrae. These are usually incidental findings, however may become clinically significant if very severe.
- Fractures:
  - Can affect each part of the vertebrae, but most commonly the spinous processes.
  - May require lesion orientated, oblique projections to visualize.
- Facet joint OA:
  - Mild changes are difficult to appreciate radiographically.
  - Radiographic signs include osteophyte formation, obliteration of the joint space, subchondral bone sclerosis, subchondral lucencies, enlargement and deformation of the articular processes.
  - Scintigraphy can be helpful in assessing how active observed bony changes are.

- Congenital defects:
  - Vertebrae: hemivertebrae, fused or wedge-shaped vertebrae.
  - Spinal alignment: lordosis, kyphosis and scoliosis.

25.2 ULTRASONOGRAPHY

Introduction

Ultrasonography was introduced in veterinary medicine in the 1970s for the assessment of the reproductive tract, and since then it has become one of the most commonly used imaging modalities and is used in equine practice on a daily basis.

Ultrasound physics

- Ultrasound is sound with a frequency beyond the range of human hearing (greater than 20 kHz). Ultrasound frequencies used for diagnostic purpose are usually in the range 1 and 20 MHz.
- Ultrasound waves are produced by a transducer, which contains crystals that have the ability to oscillate and thereby produce waves when an electrical pulse is applied, and vice versa. This physical phenomenon is called the piezo-electric effect.
- The sound waves are emitted from the crystal similar to sound waves being emitted from a loudspeaker. The sound is focused either by the shape of the transducer (curved, linear, sector) or a set of control pulses from the ultrasound machine. This focusing produces the desired shaped sound wave from the face of the transducer.
- When a sound wave encounters a material with a different density (acoustic impedance), part of the sound wave is reflected back to the transducer and is detected as an echo (this is called the pulse-echo principle). The time it takes for the echo to travel back to the transducer is measured and used to calculate the depth of the tissue interface causing the echo. The greater the difference between acoustic impedances, the larger the echo.
- Highly reflective interfaces give rise to a strong echo which is represented on the screen as a bright (= hyperechogenic) spot, while the opposite is true of weak reflective (= hypoechogenic) interfaces. Areas without acoustic interfaces such as the lumen of vessels and other cavities containing liquid (blood, bile, ascites or urine) give no reflection and appear black (= anechogenic). If the waves hit gas or bone the density difference is so great that most of the acoustic energy is reflected and it becomes impossible to see deeper.
- The echoes returning to the transducer are converted by the crystals into electrical signals which are then processed to form the image. To form the image, the ultrasound machine needs to determine the direction of the echo, how strong the echo was, and how long it took the echo to be received from when the sound was transmitted. Once the ultrasound scanner determines these three things, it can locate which pixel in the image to light up and to what intensity.
- The frequencies used for equine imaging are generally in the range of 2 to 15 MHz. Higher frequencies have a smaller wavelength and hence provide higher resolution. However, the attenuation of the sound wave is increased at higher frequencies, so in order to have better penetration of deeper tissues, a lower frequency is used. Due to this trade-off between resolution and penetration depth one should always aim to use the highest frequency possible. As a rule of thumb: 7.5 MHz or higher penetrate 5–7 cm; 5.0 MHz penetrates 7–15 cm; 2.5–3.5 MHz penetrates >15 cm.
- Doppler ultrasonography employs the Doppler effect to assess the direction and velocity of structures (usually blood) in relation to the transducer. This technique is used to assess blood flow in the heart and blood vessels.

Indications in the horse

Orthopaedic applications (see Chapter 15)

- Diagnosis and monitoring of any soft-tissue injury, especially tendons and ligaments, but also muscles and wounds (Figure 25.64).
- Investigation of the surface of bone, especially in proximal regions where radiography is challenging, e.g. pelvic fractures.
- Investigation of joint abnormalities.

Non-orthopaedic applications

(see Chapters 2, 3, 4, 6, 7, 8 and 14)

- Reproductive tract.
- Gastrointestinal tract, especially for colic.
- Liver.
- Kidney.

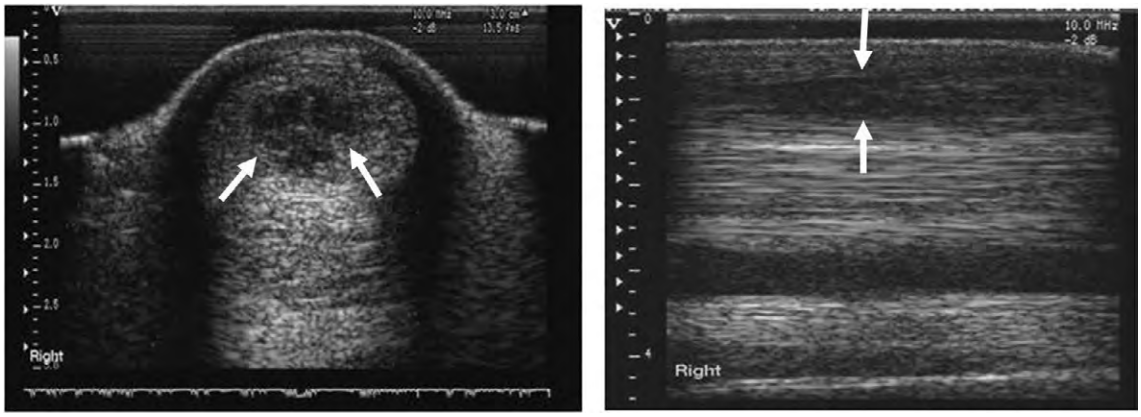


Figure 25.64 Core lesion in the superficial digital flexor tendon represented as circular hypoechogenic area in the transverse plane (left image) and as longitudinal hypoechogenic area in the longitudinal plane (right image) (white arrows). The other tendons show the normal speckled pattern in the transverse plane and striated pattern in the longitudinal plane.

- Lungs.
- Heart.

Procedure

- It is important to obtain good contact between the probe and the skin itself so the following procedure is employed before scanning:
  - The hair over the area is removed with fine-bladed electric clippers.
  - The area is then spirited to help dissolve wax and greases on the skin.
  - Surgical scrub solution is used to clean the area thoroughly.
  - Finally, the region is wiped dry.
- Contact gel (commercially available, high-viscosity type) is applied liberally to the skin, transducer and, if used, the 'stand-off'. A stand-off is a gel pad that helps to provide good contact between the transducer and the structure and also improves image quality for superficial structures: e.g. in the metacarpal/metatarsal region a stand-off is used for investigating tendon and ligament injuries.
- The procedure adopted for examination must be methodical.
- For musculoskeletal applications the left and right legs should be compared.
- Ultrasound requires a thorough knowledge not only of the anatomy of the structure examined, but also of the ultrasonographic appearance of different tissues. It is usually much easier to interpret the images, if the person doing so also has performed or was at least present at the scan.

Interpretation

Ultrasonographic parameters. The following parameters should be evaluated when interpreting ultrasound images. They should be compared between sides in paired organs or between legs. The operator, however, has to beware of bilateral lesions (especially with tendons/ligaments injuries).

- Echogenicity.
- Size.
- Shape.
- Position.
- Margination/outline.

Terminology

- Anechogenic/anechoic – not producing any echoes (therefore appearing black on the screen).
- Echogenic – the ability of a tissue to produce reflections or echoes.
- Echoic – reflections or echoes on the image.
- Hyperechogenic/hyperechoic – producing high-intensity echoes (therefore appearing brighter on the screen).
- Hypoechogenic/hypoechoic – producing low-intensity echoes (therefore appearing darker on the screen) (Figure 25.64).
- Isoechogenic/isoechoic – producing similar intensity echoes to another area.
- Normoechogenic/normoechoic – producing the expected intensity of echoes for the structure in question.
- Ultrasonogram – image produced by the ultrasound scanner.

Tissue characteristics

The following are some examples of the common appearance of various tissues; however, one should bear in mind that some tissues may have different appearances depending on their position and structure.

- Fluid is anechogenic, often with acoustic enhancement (see section on artefacts) deep to it provided the collection of fluid is sufficiently large (2–3 cm wide).
- Blood varies between hypoechogenic and hyperechogenic. Normal blood vessels are hypoechogenic, haematomas may be hyperechogenic.
- Pus has a variable appearance depending on its consistency but will have a variable number of internal echoes ± shadowing and reverberation artefacts if gas is present.
- Muscle: muscle tissue itself is hypocheogenic, but the connective tissue surrounding the fascicles and fascial planes appears hyperechogenic resulting in a speckled appearance of muscle on a transverse and a striated appearance on longitudinal images.
- Fat is relatively hyperechogenic.
- Tendons/ligaments/muscles have a dotted internal architecture when the transducer is orientated at right angles to the structure (= transverse view) and a striated pattern when the transducer is parallel (= longitudinal view) (Figure 25.64).
- Bone is highly reflective and produces acoustic shadowing deep to it.
- Gas/air will produce reverberation artefacts unless suspended in small bubbles which is characterized by bright specular reflections.
- Foreign bodies are usually strongly hyperechogenic and produce either acoustic shadowing or reverberation artefact (the so-called 'comet tail') beneath.

Common artefacts

- Operator-induced errors.
  - Poor contact between the probe and the skin due either to insufficient preparation or an air gap between the probe and the skin will result in fewer echoes returning to the probe. Consequently, either a poor quality picture or dark, anechoic bands will be produced in the image.
  - Too great a pressure exerted by the transducer not only may cause the horse to fidget but also will distort the normal anatomy.
  - Gain controls should be standardized as far as is possible. They should only be altered if the picture obtained is too dark or too light. It is common to find the gain controls set too high, which will obscure tissue detail.
  - Failure of the transducer to be positioned at 90° to the structure results in the central portion of a tendon appearing hypoechoic.
- Propagation errors.
  - The presence of air/gas (or metallic foreign bodies) either in the tissues or between the transducer and the limb will result in a 'reverberation artefact' – a series of concentric rings of echogenicity seen on the screen.
  - Refraction causes the beam to be bent as the sound travels across a boundary between two areas with different acoustic impedance. Positioning errors will be produced as a result.
  - A highly reflective surface can result in a second reflection which can produce a mirror image of the more superficial soft tissues deep to the reflective surface.
- Attenuation errors.
  - Shadowing is caused either by a highly reflective boundary (e.g. bone) or occurs at the edge of a reflective curved surface when the beam is tangential to it (so-called edge refraction or edge shadowing) – the latter is particularly evident when scanning the suspensory ligament branches in the transverse fashion when the border of the flexor tendons result in a shadow through each branch.
  - Enhancement occurs deep to an area of low attenuation (e.g. fluid) producing a more echogenic appearance to the structures beneath. With the use of sector scanners,

the central area of the beam produces more echoes than the periphery. This is known as focal enhancement.
- The presence of either shadowing or enhancement can be used to the operator's benefit to help identify areas of calcification or fluid collection.

Advantages/disadvantages of ultrasonography

Advantages

- Non-invasive: most horses tolerate ultrasound procedure very well without sedation.
- Cheap: there is a huge variety of ultrasound machines on the market which range in price from £5000 to £200 000++. A cheap ultrasound machine renders diagnostic images for the two main applications in the horse: the assessment of the reproductive organs and tendon injuries.
- Very portable: modern ultrasound scanners are often not bigger than a laptop and can easily be taken out to the client.

Disadvantages

- Ultrasound cannot penetrate air-filled structures, so cannot go deep to gas-filled intestines or beyond the surface of the lung.
- Diagnostic ultrasound also does not penetrate bone, so can only be used to evaluate bone surface.

25.3 SCINTIGRAPHY

Introduction

Following the discovery of radium by Marie Curie, the early medical uses of radioactivity were mainly for therapeutic purposes. Professor Gottlieb Ueltschi, from Bern, was the first to publish on the use of scintigraphy in the horse in 1977. Since then it has found widespread use in referral level equine hospitals.

Underlying principle

- Scintigraphy is an imaging process which detects and depicts gamma radiation emitted by the patient after injection of a radiopharmaceutical.
- It is the only imaging modality which portrays the metabolism of the system being studied, rather than the anatomy (the anatomic resolution of scintigraphy is rather poor).
- The patient is injected with a radiopharmaceutical: this is a compound of a radioisotope (usually technetium ^{99m}Tc) plus a label which is taken up by the system under investigation (e.g. phosphonates for bone).
- There is a large range of possible labels – including antibodies, erythrocytes, leukocytes – and it is possible to specifically image the liver, kidneys, brain, heart, thyroids or skeleton in animals. In humans the range of indications for scintigraphy is vast; in horses, however, it is mainly used for investigating the musculoskeletal system.

Choice of radiopharmaceutical and dose

- Technetium-99m (^{99m}Tc) is the main radionuclide used in nuclear medicine imaging investigations.
- In many ways it is almost ideal for use in scintigraphy:
  - It has a half life of 6 hours, which is enough time to perform a study, but short enough to send the horse home after 48 hours.
  - It is a pure gamma ray emitter, with an energy of 141 keV, which allows valuable diagnostic studies to be carried out while delivering radiation doses to patients that are no higher than those received from X-ray procedures.
  - Technetium is produced from the decay of molybdenum, which is a byproduct of uranium-based nuclear power plants.
  - Equine hospitals can either obtain already made-up vials of ^{99m}Tc-MDP on the day of the scan from human hospitals or generate their own from molybdenum which is delivered on a weekly basis.
  - The standard dose for horses is 1 GBq/100 kg, commonly injected as 5–10 mL.
- In horses the main indication for scintigraphy is to evaluate the musculoskeletal system, and the radiopharmaceutical of choice is ^{99m}Tc linked to phosphonates (e.g. ^{99m}Tc-methylendiphosphonate).
  - This radiopharmaceutical binds to hydroxyapatite crystals, which are a major component of bone.
  - The uptake depends mainly on blood supply, bone turnover (osteoblastic activity) and the quantity of mineralized bone.
  - Since bone as a living tissue undergoes constant modelling, there is always a mild to moderate uptake; however, this can increase considerably for pathological conditions, e.g. inflammation or fractures.

Detection of radiation and image formation

In equine hospitals there are usually two types of detector: probe detectors to monitor contamination and gamma cameras which produce the diagnostic images.

Gamma cameras have a whole grid of detectors that allow the formation of an image by localizing where the gamma rays come from. Gamma cameras can be rectangular (Figure 25.25) or circular and come in different sizes. Due to the size of the horse, multiple images are required and large field of view is of major advantage because it reduces the number of images and the time spent next to a radioactive horse and, hence, exposure.

Indications in the horse

As with any imaging modality the choice of performing scintigraphy should be based on a through clinical investigation. This is especially important since scintigraphy does involve comparatively high radiation exposure to personnel. It is also a very sensitive, but not very specific technique which usually often identifies multiple areas of increased radiopharmaceutical uptake, the clinical significance of which is often unclear and interpretation needs to be based on clinical findings or other tests. In some cases, especially if the problem is long-standing, there may be no scintigraphic abnormalities, however this still does not rule out musculoskeletal pathology.

In the horse scintigraphy ('bone scans') are performed in the following cases:

- Horses showing acute severe lameness. Some fractures are not visible radiographically in the first few days after injury, while scintigraphy will show an increased in radiopharmaceutical uptake (Figure 25.18b).
- Proximal limb/back/neck/head: areas where the site of pain cannot be determined with diagnostic analgesia and where other imaging techniques are technically impossible or less useful.

- Multiple site problems.
- Unruly horse: some horses are too uncooperative for diagnostic analgesia, but will tolerate having a camera moved round them.
- Other circumstances: scintigraphy is often employed as a 'last resort' when no diagnosis could be reached with other techniques. These are the cases where scintigraphy is the least useful.
- Horses with a suspected dental problem where the affected tooth cannot be identified radiographically.
- Non-musculoskeletal indications in the horse are not very commonly performed but the use of scintigraphy for the evaluation of kidney and liver function, tumour detection and the use of Tc-labelled white cells for the detection of abscesses and inflammatory bowel disease has been reported.

Procedure

- Ideally horses should come into the hospital on the day before the scan to allow determination of the exact weight and hence calculation of exact dosage of the radiopharmaceutical.
- Horses are lunged on the day of the procedure before injection to improve blood flow to the distal limb, if the condition of the horse allows it.
- The horse is injected intravenously with the radiopharmaceutical. The use of an intravenous catheter is recommended since it decreases exposure to personnel.
- Imaging can be undertaken in three phases:
  - Vascular flow phase – immediately post injection. The radiopharmaceutical is visible in the blood vessels. This provides information on blood flow to areas, but can also detect blockage of blood vessels. In the horse it has shown to be useful in the visualization of thromboembolism and in cases of infection.
  - Soft-tissue phase – 5 to 10 minutes: the radiopharmaceutical is entering the capillary beds of the soft tissues.
  - Bone phase – 2 to 3 hours: at this stage the radiopharmaceutical is largely cleared form the soft tissue and located in the bone. For the majority of applications this is the most useful phase.
- The amount of gamma rays is registered as counts, and images are acquired until a sufficient number of counts per pixel are collected.
- After the procedure, the horse is still radioactive and should be contained until the radioactivity has receded (usually after a maximum of 48 hours). Tc is mainly excreted in urine, and to a lesser degree in sweat and faeces, hence appropriate measures to avoid contamination of the environment and personnel have to be taken.

Interpretation

An increase in bone turn-over results in an increase in radiopharmaceutical uptake at that site, which is commonly referred to as 'hot spot'. As with all other imaging techniques, it is easier to detect an acute lesion (fracture) on scintigraphy than a low-grade chronic one (e.g. degenerative joint disease). Scintigraphy is very sensitive but fairly non-specific. That is, osteomyelitis may look just the same as a fracture on a bone scan, and other tests are usually necessary to make the final diagnosis once scintigraphy has localized an abnormality. Figure 25.65 shows a dorsal scan of the pelvis of a horse with an increase in radiopharmaceutical uptake over its left ilium. The ultrasound scan of this area clearly shows a disruption of the bone outline indicating a fracture.

Because of its high sensitivity, scintigraphy picks up changes in bone turnover that are not caused by pathology but reflect an increase in mechanical load the bone at this site experiences. Most horses'

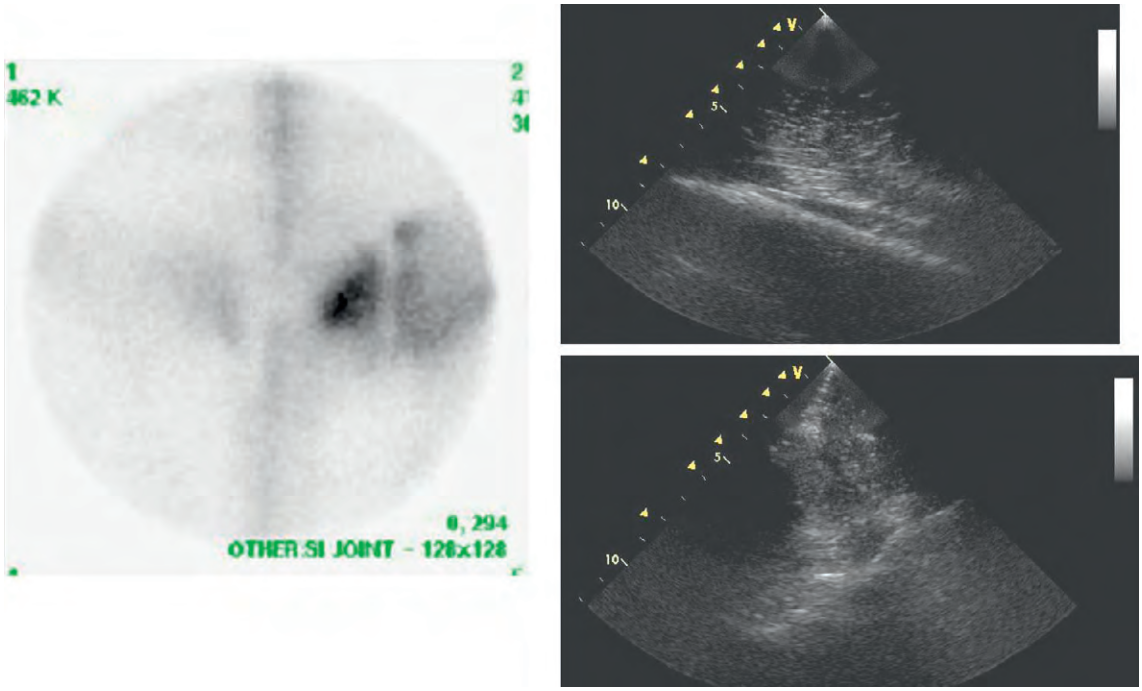


Figure 25.65 Iliial wing fracture. The dorsal scintigram of the pelvis (left) with a marked increase in radiopharmaceutical uptake over the left ilial wing (massive black spot). The ultrasound image shows a disruption of the bony outline of the ilial wing (lower image) compared to the normal image (top).

conformation causes more force to go through the medial aspect of the limb and hence the bone is more active there and a focal increase in radiopharmaceutical is often seen on the medial aspect of the proximal metatarsus.

Scintigraphy can also result in 'cold spots', areas with no radiopharmaceutical uptake, most commonly due to lack of blood supply, e.g. a sequestrum.

One of the major advantages of scintigraphy is that it allows objective analysis in addition to subjective viewing. This is done by comparing the number of counts from one area to the same area on the other leg or to a reference area by drawing region of interests (Figure 25.18b).

Advantages/disadvantages of scintigraphy

Advantages

- High sensitivity: compared to any other imaging modalities it has a very high sensitivity (which means a very low rate of false negative examinations) and (which is why one of the main indications for scintigraphy in people is the detection of metastases).
- This is the only imaging modality that portraits metabolism rather than morphology; hence, it allows detection of subtle and early changes.
- It allows objective analysis through region of interest technique.
- It can be performed in the standing horse on proximal body parts.

Disadvantages

- Exposure to ionizing radiation.
- Costs.
- Low to moderate specificity does often not allow the identification of the true nature of the disease, e.g. a hot spot could indicate a fracture as well as an infection.
- Planar imaging modality, hence summation of radioactivity from overlying structures. In humans rotating camera systems are used which allow the acquisition of tomographic images; this is not possible in the horse (yet).
- Poor anatomical resolution, which makes detailed localization often difficult.

25.4 COMPUTED TOMOGRAPHY (CT) AND MAGNETIC RESONANCE IMAGING (MRI)

Computed tomography and magnetic resonance imaging are both tomographic imaging modalities which represent a three-dimensional object as a series of two-dimensional slices and hence avoid superimposition of structures. Both modalities allow for viewing the structure in different planes after acquisition and its reconstruction in three dimensions. Both imaging modalities have become more widespread over recent years and are now available in many equine hospitals. Especially the Hallmarq system provides a relatively low-cost solution for performing MRI of the distal limb of the standing horse.

Computed tomography (CT)

Underlying principle

Conventional radiographs depict a three-dimensional object as a two-dimensional image. Their main limitation is that overlying tissues are superimposed over each other and summed up to produce an image. Computed tomography overcomes this problem by producing a series of cross sectional slices of the structure of interest, thus virtually slicing the body up. X-ray slice data are generated using an X-ray source that rotates around the object; X-ray sensors are positioned on the opposite side of the circle from the X-ray source. Many consecutive scans are taken as the object is gradually passed through the gantry. The resulting information is combined together by a mathematical procedure known as tomographic reconstruction.

Another limitation of the conventional radiograph is its inability to distinguish between two tissues with similar density. Computed tomography can differentiate between tissues of similar density because of the narrow X-ray beam and this can be made visible by the use of 'windowing'. The information acquired by computed tomography is stored on a computer as digital raw data and an image can be displayed on a video monitor or printed onto X-ray film. The image is made up of a matrix of thousands of tiny squares or pixels. Each pixel has a computed tomography number (measured in Hounsfield units, HU) attributed to it. The computed tomography number is a measure of how much of the initial X-ray beam is absorbed by the tissues at each point in the body. This varies according to the radio density of the tissues. The radiodenser the tissue is, the higher the computed tomography number or Hounsfield unit:

- Ideally the image would be displayed with a different shade of grey for every different computed tomography number. However, the human eye is limited to distinguish 256 shades of grey so a system of windowing is used. The radiographer selects the range of computed tomography numbers to be displayed and all the numbers within this range are spread over the available grey scale.
- The benefits of computed tomography do not come without a price. Firstly, computed tomography is considerably more expensive than conventional radiography. A new scanner starts at around £150 000. Used scanners, usually out of human hospitals, are considerably cheaper to buy; however, they still require relatively high maintenance costs. Secondly, the radiation dose from computed tomography is much larger than for conventional radiography. Thirdly the size of the opening of the scanner (usually between 60 and 80 cm), and the external dimensions of the gantry, limits its use in the adult, standard-sized horse to the head, cranial part of the leg and limbs up to and including the carpus and tarsus. The use of CT for the equine stifle with the aid of a specially designed table set-up has been reported, but is not (yet) widely used. CT in the horse usually requires general anaesthesia with the associated risks. Recently a CT system has been developed that allows for scanning heads in the standing, sedated horse. This system requires either for the gantry to be raised in relation to the horse or the horse lowered in relation to the gantry to allow positioning of the horse's head in the scanning field.

Interpretation

Interpretation of CT images follows the same rules as interpretation of radiographs in general and can be described using the Roentgen signs: lesions are described by their ability to attenuate X-rays, location, number, shape and outline. As with any other imaging modality comparing left and right sides or legs is very helpful. A thorough knowledge of artefacts is essential since they can interfere with images interpretation.

Indications in the horse

- Head. CT has proved to be useful for the diagnosis of head problems in the horse, especially for the evaluation of fracture

configuration, cysts, abscesses and tumours of the jaws/sinuses/nasal cavity/orbits. CT has also been used in the diagnosis of the causes of chronic sinusitis, the hyoid apparatus, the temporomandibular and atlantooccipital joints. It has been shown to allow visualization of dental disorders with relative ease. For detection of brain tumours, CT scanning with IV contrast can be used (Figure 25.66) but is thought to be less sensitive than magnetic resonance imaging (MRI).

- Cervical spine. In the horse CT is restricted to the cranial part of the neck by the size of the gantry opening. CT has been found useful in the diagnosis of osteomyelitis, fractures of vertebral body, changes in the facet joints, and contrast-enhanced CT has been shown to be useful in the diagnosis of cervical stenotic myelopathy.
- Extremities. CT is the method of choice for the evaluation of subchondral bone disease, e.g. occult subchondral osseous cyst-like lesions of the equine tarsocrural joint and osteochondritis dissecans-like lesions of the proximal phalanx. CT has also been shown to aid diagnosis in horses suspected of suffering from navicular syndrome, osteoarthritis and osteomyelitis. CT enables the thorough assessment of complex fractures because of its ability to reconstruct the area of interest in multiple planes. The use of contrast enhances its usefulness for evaluating soft tissue problems of the distal limb. It is widely used in research for bone density measurements, e.g. to assess the effect of training on third metacarpal bone in racehorses. CT angiography allows the mapping and evaluation of blood vessels in three dimensions. Figure 25.67 shows a series of three CT

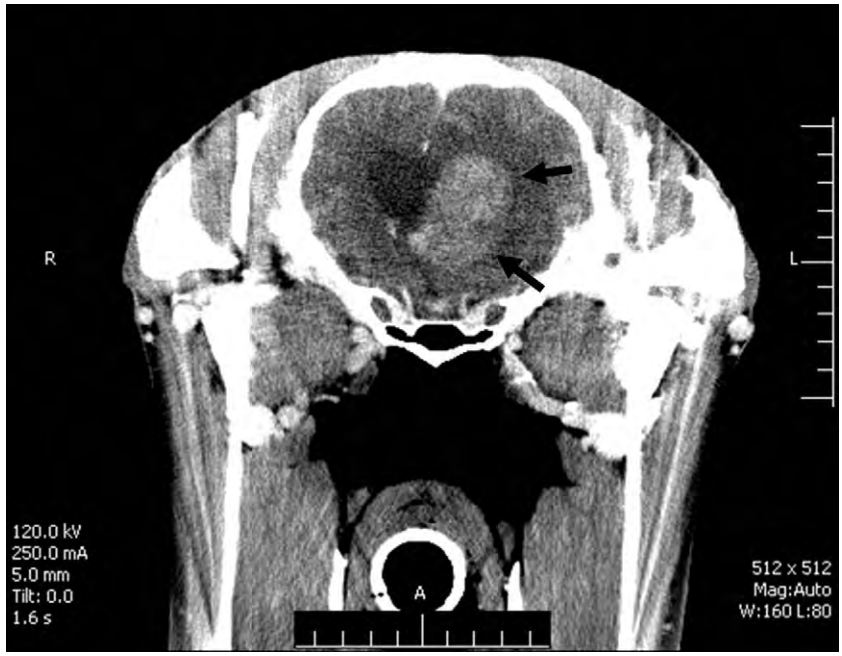


Figure 25.66 Computed tomographic scan after injection of intravenous contrast medium showing a cross-section of the head at the level of the temporomandibular joint. There is an oval mass in the left ventricle consistent with a cholesterol granuloma (black arrows).

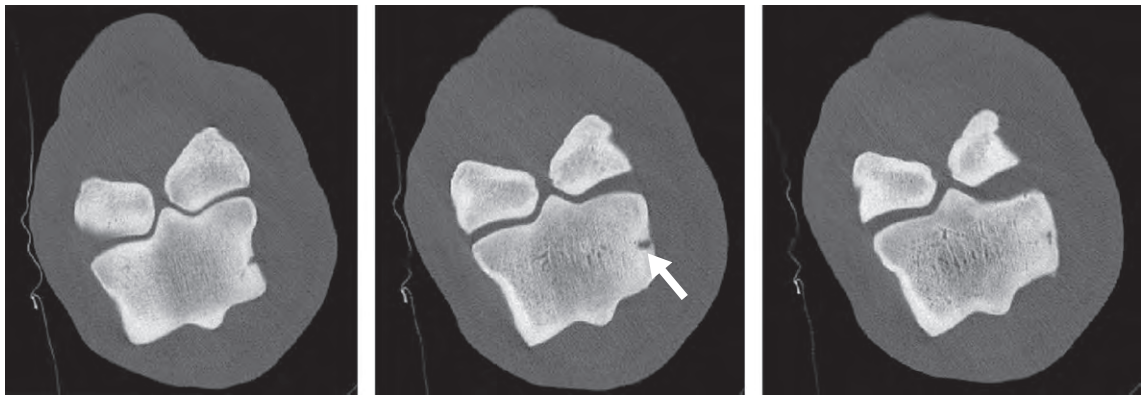


Figure 25.67 Computed tomographic scan showing a cross-section of a horse leg at the level of the proximal sesamoid bones. There is a lucent defect metatarsus with marked sclerosis of the surrounding bone and a faint periosteal reaction at the attachment site of the lateral collateral ligament on the dorsolateral aspect of the distal third metatarsus (white arrow).

scans of a horse suffering from severe desmopathy of the lateral collateral ligament indicated by lucency surrounded by marked sclerosis at the attachment site of the ligament onto the distal metatarsus.
- Others.

In foals and small breed horses the use of CT is similar to that in humans and small animals and can be extended to the proximal part of the limbs, the back, the thorax and abdomen.

Magnetic resonance imaging (MRI)

History

Magnetic resonance imaging is based on the physical phenomenon of nuclear magnetic resonance. (The original name for the medical technology is nuclear magnetic resonance imaging, NMRI). MRI became available in the medical field in the late eighties and early nineties when faster scanning times were made possible by the advancements in computer sciences. At the same time MRI has become available for small animals in some specialist centres soon followed by mainly open systems at some universities that allowed access for anaesthetized horses. A low-field system has been developed for the use in the conscious horse by Hallmarq Inc and is now widely used in numerous equine hospitals around the world (Figure 25.68).

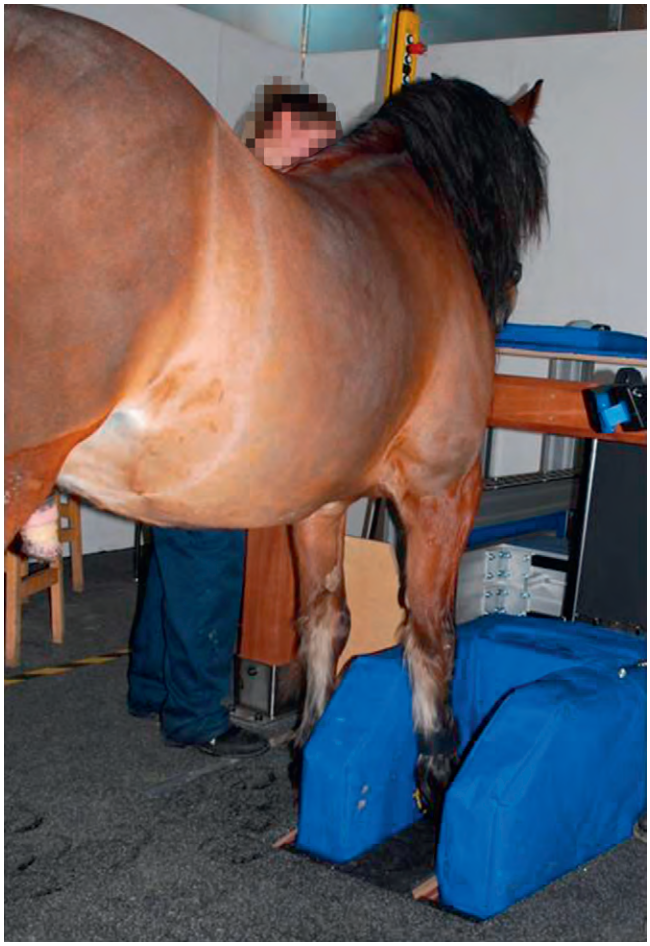


Figure 25.68 Standing MRI in the sedated horse with the right foot positioned in the magnetic scanner.

Underlying principle

Nuclear magnetic resonance describes the physical phenomenon that all atoms that contain odd numbers of nucleons have an intrinsic magnetic moment. The most common molecule in the body is hydrogen and the nucleus of hydrogen is composed of an unpaired proton. It is this proton which plays a major part in the generation of the MRI signal. The direction of the magnetic moment of these protons is randomly distributed in nature. If those protons are put in the field of a stronger magnet, the tiny spins of the hydrogen atoms align themselves with the magnetic field. If a third, perturbing magnetic field is introduced to this system, the aligned spins are aligned afresh, depending on the strength and duration of the perturbing field. When this perturbing field is suddenly turned off the spins begin to wobble before gradually realigning themselves to their original vector in the magnetic field. Whenever a magnetic vector spins, it induces a current in a nearby coil and this is the principle by which the signal is detected from the realigning magnets. The realigning of the magnets is called the relaxation and occurs in two different ways: T1 and T2 relaxation. Depending on the application, the emphasis is put on one or the other and results in 'T1 or T2 weighted' images.

Depending on the strength of the magnetic field, and the way this magnetic field is generated, one can distinguish between low-field magnets that use permanent magnets and high-field magnets that use superconducting magnets. Permanent magnets are considerably cheaper to buy and to maintain than superconducting magnets that require the permanent cooling down of the system through the use of helium. Since there is a positive relationship between magnet strength and the signal-to-noise ratio, high-field magnets produce a higher quality image.

Advantages/disadvantages of MRI

MRI has all the advantages of a tomographic imaging modality by avoiding superimposition without involving ionizing radiation. So far it has not been possible to establish any damaging effect on the body.

In the horse, the Hallmarq low-field system allows for scanning standing horses' distal limbs; conventional gantry systems have the same size limitations as CT and require general anaesthesia. MRI scanners are expensive to buy and maintain. Compared to CT, MRI requires more technical expertise in operating and trouble-shooting.

Indications in the horse

The major application in the horse is the evaluation of foot-related problems. Over the last few years a series of studies have demonstrated that MRI is an excellent diagnostic tool in the diagnosis of soft-tissue problems, especially ligaments and tendons, such as the deep digital flexor tendon or collateral ligaments in the foot (Figure 25.69). It has been used for the assessment of fractures and subchondral bone pathology; it seems, however, inferior to CT in the visualization of bone pathology. It allows for detection of cartilage damage in clinical circumstances depending on the quality of the scanner and has been used as a research tool to assess cartilage metabolism.

It has been used in the diagnosis of brain tumours and abscesses in horses.

A thorough knowledge of the MRI anatomy of the different structures and a good understanding of MRI-associated artefacts is necessary to avoid misinterpretation of findings and come to the right diagnosis.

Like CT, the scope of MRI in foals or miniature breeds is much larger and similar to that in humans or small animals.

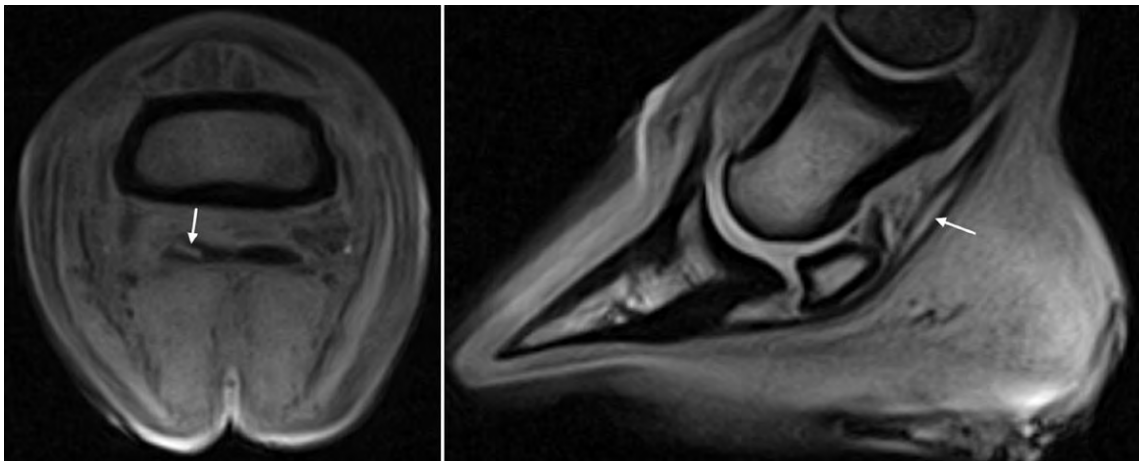


Figure 25.69 Transverse (left) and sagittal (right) image of the foot of a horse showing a lesion in one lobe of the deep digital flexor tendon (white arrows).

FURTHER READING

Radiography and radiology

Butler J, Colles C, Dyson S, Kold S, Poulos P (2008) Clinical radiology of the horse, 3rd edn. Wiley-Blackwell, Oxford

Schebitz H, Wilkens H (1986) Atlas of radiographic anatomy of the horse, 4th edn. Thieme Publishing, Stuttgart

Smallwood J, Shively M, Rendano V, Habel R (1985) A standardized nomenclature for radiographic projections used in veterinary medicine. Veterinary Radiology 26:2–9

Weaver M, Barakzai S (2009) Handbook of equine radiography. Saunders, Philadelphia

Ultrasonography

Reef V (1998) Equine diagnostic ultrasound. W.B. Saunders, Philadelphia

Scintigraphy

Berry C, Daniel G (1996) Handbook of veterinary nuclear medicine. North Carolina State University, Raleigh NC

Dyson S, Martinelli M, Pilsworth R, Twardock R (eds) (2003) Equine scintigraphy. EVJ (Wiley-Blackwell), Oxford

CT and MRI

Denoix J-M (2000) The equine distal limb: an atlas of clinical anatomy and comparative imaging. Wiley-Blackwell, Oxford

Murray R (ed) (2010) Equine MRI. Wiley-Blackwell, Oxford

Murray R, Mair T (2005) Use of magnetic resonance imaging in lameness diagnosis in the horse. In Practice 27(3):138–146

Tucker R, Sande R (2001) Computed tomography and magnetic resonance imaging in equine musculoskeletal conditions. Veterinary Clinics of North America, Equine Practice 17(1):145–157

Radiation safety

Guidance notes for the safe use of ionising radiation in veterinary practice: ionising radiations regulations 2002. British Veterinary Association publication

Radiation safety in equine practice. DVD directed by Pilsworth R, Holmes M, BEVA

Chapter 26

Common problems and techniques in equine critical care

Kelsey A. Hart, Kira L. Epstein

CONTENTS

26.1 Principles of fluid therapy 561
Reasons to administer fluids 561
Components of a fluid plan 562
Route of fluid administration 563
Rate of fluid administration 563
Management of common electrolyte and acid–base disturbances 564
Fluid therapy in neonatal foals: special considerations 565
26.2 Pathophysiology and management of shock 565
Definition of shock 565
Pathophysiology of shock 566
Classifications and types of shock 566
Stages of shock 567
Management of shock 567
Monitoring 568
26.3 Administration of blood products in equine critical care 568
Commonly used blood products 568
Blood transfusions 569
Supportive care and other management considerations in the anaemic patient 571
Plasma transfusions 571
26.4 Nutritional support in the critically ill horse and foal 572
Introduction and key points 572
Nutritional requirements 572
Nutrition in the sick foal: special considerations 574
26.5 Evaluation and management of the sick neonatal foal 574
Introduction 574
Evaluation of the neonatal foal 574
Stabilization and management of the sick neonatal foal 576
26.6 Management of acute respiratory distress 578
Causes of respiratory distress 578
Clinical signs and physical examination findings 578
Emergency treatment 579
Ancillary diagnostics 581
26.7 Cardiopulmonary cerebral resuscitation (CPCR) 582
Cardiopulmonary arrest 582
Cardiopulmonary cerebral resuscitation (CPCR) and its use in horses 582
Further reading 584

26.1 PRINCIPLES OF FLUID THERAPY

Reasons to administer fluids

- Fluid therapy is most commonly used to restore and/or maintain hydration and blood volume.
- Fluid losses and dehydration may occur as a direct result of the primary disease, such as gastrointestinal or urinary losses in colitis or renal failure or loss in acute or chronic haemorrhage, or may be due to decreased water intake in patients with anorexia or dysphagia.
- Enteral intake of feed and water is also often restricted for periods of time in the management of horses with certain types of colic, in which case intravenous fluids must be administered to prevent dehydration.
- Occasionally, specific types of fluids are administered to euhydrated patients to correct electrolyte or acid-base derangements, to provide colloidal support to hypoproteinaemic patients, or to provide oxygen carrying capacity in anaemic patients.

© 2013 Elsevier Ltd
DOI: 10.1016/B978-0-7020-2801-4.00011-0

Components of a fluid plan

- Constructing an appropriate fluid plan for a critically ill patient is both a science and an art.
- When forming a fluid plan, four key components must be addressed:
  1. fluid volume to be administered.
  2. type of fluids to be administered.
  3. route of fluid administration.
  4. rate of fluid administration.
- In addition, the patient's status and response to the initial plan must be monitored closely – sometimes hourly or even more frequently in critically ill horses – and the fluid plan revised as the patient's condition changes.

Fluid volume

In general, the amount of fluids to be administered is determined by the following formula:

$$\text{fluid amount} = \text{hydration deficit} + \text{maintenance fluid needs} + \text{ongoing fluid losses}$$

The hydration deficit is an estimation of fluid losses determined primarily by clinical assessment of the patients. It is estimated with the following formula:

$$\text{hydration deficit (L)} = \text{estimated percent (\%) dehydration} \times \text{body weight (kg)}$$

The % dehydration typically ranges from 5 to 12% and is estimated based on physical examination and clinicopathological findings. Typical clinical findings in horses with varying degrees of dehydration are shown in Table 26.1.

Maintenance fluid needs are defined as the amount of fluid needed per day to maintain euhydration, and take into account use of water in cellular metabolism and water losses in urine, faeces, and respiratory and cutaneous evaporation. The animal's age and activity level and the environment (temperature, humidity) also impact maintenance fluid needs.

Maintenance fluid requirements in adult horses range from 40 to 60 mL/kg/day. In neonatal foals (<3 to 4 weeks of age), maintenance fluid needs are higher at 80–100 mL/kg/day due to a larger percentage of free body water and immature renal urine concentrating capacity.

Ongoing fluid losses due to the patient's primary or underlying disease also must be considered when determining a fluid plan. Sometimes these are easy to quantify, such as in a horse with proximal duodenitis/jejunitis that is producing large volumes of gastric reflux that can be collected via a nasogastric tube and measured.

However, most often ongoing fluid losses are difficult or impossible to quantify, such as losses in diarrhoea or in polyuria associated with renal failure. In these cases, an estimation of the disease's contribution to increased maintenance fluid needs is typically made and then adjusted based on close monitoring of the patient's response to the initial fluid plan. For example, a horse with moderate to severe diarrhoea may need two to four times the normal maintenance fluid requirements of a healthy horse to maintain euhydration due to fluid losses in diarrhoea.

Table 26.1 Clinical signs associated with dehydration in the horse

Percent dehydration	Clinical signs
<5%	• Not clinically apparent.
5–7%	• Tacky to dry mucous membranes. • Prolonged CRT. • +/– mild depression.

8–10%	• The above, and:
	  • prolonged skin tenting.
	  • weak peripheral pulses.
	  • cool extremities.
	  • slow jugular refill.
	  • moderate tachycardia.
	  • depressed mentation.

10–12%*	• The above, and:
	  • cold extremities.
	  • obtunded mentation.
	  • severely prolonged skin tenting (does not return to normal position).
	  • severe tachycardia.

>12%	• The above, and:
	  • moribund/comatose.
	  • recumbent.
	  • severe tachycardia or bradycardia.

*Note: ≥10–12% dehydration is fatal if not addressed rapidly and aggressively.

Type of fluid

While the ultimate goal of fluid therapy in dehydrated patients is to increase total body water, administration of pure water is not possible due to tonicity issues and the potential for development of dangerous hyponatraemia. Thus, when determining a type of fluid to administer, it is important to consider the tonicity and electrolyte composition of the fluid relative to plasma, as well as whether or not the patient requires supplemental oncotic support or oxygen carrying capacity.

- Fluids with a similar electrolyte composition to plasma are described as 'balanced'.
- Crystalloids are solutions that contain both electrolyte and non-electrolyte solutes that can move freely between the extracellular and intracellular fluid compartments. Crystalloids may be isotonic, hypertonic, or hypotonic relative to plasma, and may be balanced or unbalanced. Examples of crystalloid fluids used commonly in equine medicine are shown in Table 26.2. Isotonic crystalloid fluids are administered primarily to restore and maintain hydration and correct electrolyte derangements. Hypertonic crystalloids are used to rapidly increase intravascular volume in patients with shock, but must be followed by an appropriate volume of isotonic fluids (see section on 'Pathophysiology and management of shock'). Hypotonic fluids are used infrequently; they are used in patients with increased plasma osmolality (e.g. hypernatraemia).
- Colloids are solutions that contain high-molecular-weight solutes that cannot leave the vascular space, and thus serve to increase colloidal oncotic pressure. The most common colloids used in equine medicine are equine plasma or synthetic colloids such as dextrans and hydroxyethyl starch (hetastarch). Colloids are most often used in patients with shock or severe hypoproteinaemia (see section on 'Pathophysiology and management of shock').
- Oxygen-carrying fluids include whole blood, packed red blood cells, and purified bovine haemoglobin products (e.g. Oxyglobin®). Oxygen-carrying fluids are administered to patients

Table 26.2 Types of intravenous fluids often used in equine critical care

Crystalloids	
Isotonic	***Dextrose-containing fluids:*** • 5% dextrose in water. • 2.5% dextrose/0.45% NaCl. ***Balanced polyionic fluids:*** • Ringer's solution. • Lactated Ringer's solution. • Hartmann's solution. • Acetated Ringer's solution. • Normosol-R, Normosol-M. • Plasmalyte 148, Plasmalyte 56. ***Other isotonic crystalloids*** • 0.9% NaCl.
Hypertonic	3%, 5%, or 7.2% NaCl (7.2% NaCl is used most frequently in equine medicine)
Hypotonic	0.45% NaCl
Colloids	
Non-oxygen carrying	***Natural*** • Equine plasma. • 25% Human albumin. ***Synthetic*** • Hydroxyethyl starch (hetastarch, pentastarch). • Dextrans.
Oxygen-carrying	***Natural*** • Fresh whole blood. • Washed/packed red blood cells. ***Synthetic*** • Purified bovine haemoglobin (Oxyglobin®).

with decreased oxygen-carrying capacity secondary to severe acute haemorrhage or anaemia. These products contain large macromolecules and are thus colloids, but as they do less to increase colloidal oncotic pressure, compared to plasma and synthetic colloids, they are not typically used in this manner. Administration of these products is covered in more detail below in the section on 'Administration of blood products in equine critical care'.

Route of fluid administration

When deciding on a route of fluid administration, it is important to consider the volume and type of fluid being administered, the desired administration rate (see below), and the underlying condition being treated. Economic factors may also play a role in the decision in some cases. The following routes of administration are used most often in horses:

- Intravenous fluid administration.
  - This route is used most often in equine critical care, as it permits rapid administration of large volumes of fluids.
  - In horses and foals, intravenous access is most often achieved through catheterization of one or both jugular veins. In adult horses, the lateral thoracic veins may also be used, though fluids cannot be administered quite as rapidly (Figure 26.1).
  - The cephalic veins are occasionally used if jugular or lateral thoracic vein access is limited, but cephalic catheters can be dangerous to place in some fractious horses and are typically difficult to maintain for long periods.
- Enteral fluid administration.
  - This method is typically inexpensive and is appropriate for patients that can tolerate oral fluids (e.g. patients that are not refluxing and that do not have intestinal disease that would impair absorption of oral fluids) and for those that do not require rapid administration of large volumes of fluid (e.g. severely dehydrated patients or patients in shock).
  - Enteral fluid therapy is typically administered to horses via a nasogastric tube; an indwelling nasogastric tube may be maintained for repeat administration or continuous infusions.
  - Small amounts of pure water may be administered enterally, but if large volumes or prolonged enteral fluid therapy is required then an isotonic electrolyte solution should be used to prevent the development of hyponatraemia.
  - Large volume enteral fluid therapy is rarely used in neonatal foals.
- Other routes of administration.
  - In foals requiring fluid therapy in which the above routes are not usable, intraosseus fluids may be administered. However, it is difficult to maintain intraosseus catheters for long periods of time, and rapid or large volume fluid administration through this route can be challenging.
  - Subcutaneous fluids are not well tolerated in horses and foals and should be avoided. Similarly, the risks associated with intraperitoneal fluid administration in horses (e.g. gastrointestinal puncture, peritonitis) almost always preclude the use of this method in horses or foals.

Rate of fluid administration

The rate of fluid administration is determined primarily by the underlying condition of the patient and the route of fluid administration.

- Shock-rate intravenous fluid administration (see section on 'Pathophysiology and management of shock' for more detail)
  - Severely dehydrated patients showing signs of shock require rapid intravenous fluid administration, at 40–90 mL/kg/hour, to quickly restore intravascular volume.
  - The patient should be closely monitored and the fluid rate modified accordingly as the hydration status improves.
  - In adult horses, multiple large bore catheters and fluid pumps may be required to achieve such rapid rates (e.g. ~40 L/hour for a 450 kg horse).
  - Therefore, hypertonic crystalloids and/or colloids followed by isotonic crystalloids may be alternatively or additionally used in these cases to more rapidly increase intravascular volume.
- Maintenance intravenous fluid administration.
  - In patients with less severe dehydration or in those receiving maintenance fluids only, the administration rate is determined by dividing the total volume of fluid required in 24 hours into intermittent boluses or a constant rate infusion (CRI). For instance, if the total 24-hour fluid volume required by a 40 kg foal with estimated 5% dehydration is 6 L (2 L hydration deficit and 4 L maintenance needs at 100 mL/kg/day), then the foal could receive a 1L bolus every 4 hours or a CRI of 250 mL/hour. Specialized IV sets that permit calculation of volume per hour based on drip rate or an electronic fluid pump are required for accurate administration of a CRI.

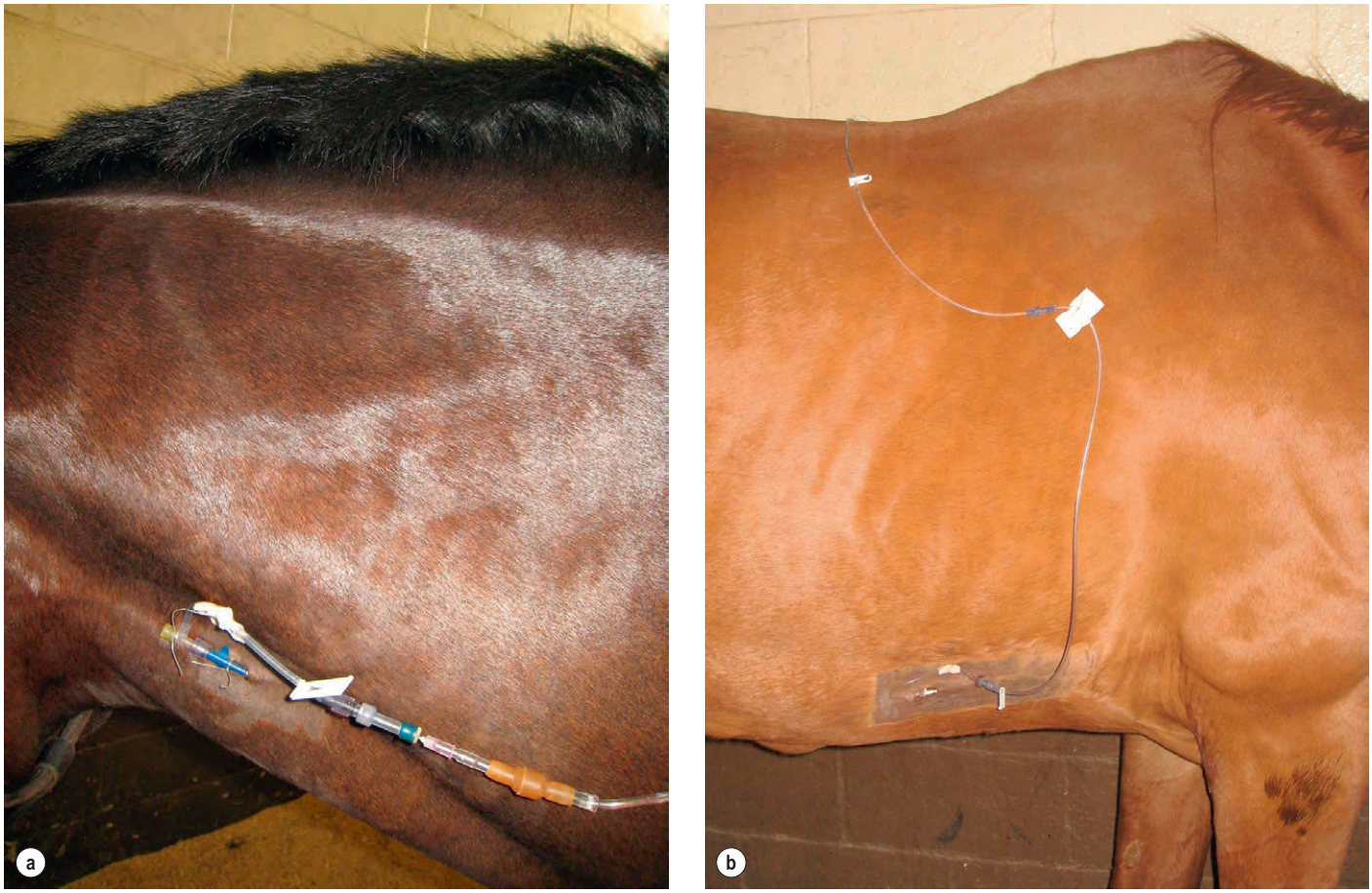


Figure 26.1 Jugular (a) and lateral thoracic (b) intravenous catheters sutured in place in adult horses.

- Enteral fluids.
  - No more than 6–8 L at one time or 2–4 L/hour of enteral fluids should be administered to an adult horse to avoid gastric distension and associated discomfort.
  - Enteral fluid therapy rates should be slowed or discontinued if signs of colic or abdominal distension are observed during or after enteral fluid administration.

Management of common electrolyte and acid–base disturbances

Sodium derangements

Hyponatraemia

CNS signs (altered mentation, recumbency, seizures) are typically seen with serum sodium concentrations <120 mmol/L, but sodium concentrations may be much lower if hyponatraemia develops slowly.

The sodium deficit is calculated as follows:

$$\text{sodium deficit} = (125\ \text{mmol/L} - \text{patient sodium concentration}) \times 0.6 \times \text{body weight (kg)}$$

- If neurological signs are present, aim to correct the sodium deficit over approximately 6 hours.
- If the horse is asymptomatic or if hyponatraemia is known to have been present for a prolonged period of time (several days or more), the deficit should be corrected over 24–48 hours (no faster than 0.5 mmol/hour).
- An appropriate fluid type, typically 0.45% or 0.9% NaCl or a balanced polyionic isotonic crystalloid, is selected based on the sodium content of the fluid and the rate at which fluids are being administered, to avoid replacing sodium too quickly.
- It is vital to monitor serum sodium concentrations closely (as often as every few hours initially) during therapy to ensure that the correction is not too rapid.

Hypernatraemia

- CNS signs are frequently observed with serum sodium concentrations >160 mmol/L.
- Hypernatraemia is corrected by administration of fluids containing free water.
  - 5% dextrose in water or 0.45% saline/2.5% dextrose are ideal because these fluids are isotonic but provide more free water than other types of fluid once the dextrose is metabolized.
- The amount of these fluids to administer is calculated by the following formula:

$$\text{L of water} = (\text{patient Na concentration}/140 - 1) \times 0.6 \times \text{body weight (kg)}$$

- It is vital that hypernatraemia be corrected slowly, no faster than 0.5 mmol/hour. If the patient is dehydrated and requires fluid administration at a faster rate, fluids containing sodium should be used to avoid too rapid correction.

Potassium derangements

Hypokalaemia

Since potassium is primarily an intracellular cation, serum potassium concentration is a relatively insensitive indicator of total body potassium status. However, severe hypokalaemia can result in muscle weakness and cardiac arrhythmias; thus, hypokalaemia should be corrected if serum potassium concentration is <3 mEq/L, and potassium should be supplemented even in normokalaemic patients with prolonged anorexia or if fluids containing dextrose or bicarbonate are administered, as these can lead to rapid intracellular movement of potassium ions.

Potassium should not be administered intravenously faster than 0.5 mEq/kg/hour. Intravenous fluids supplemented with potassium at 10–40 mEq/L and administered at maintenance rates are generally safe. Intravenous fluids that are rapidly bolused should not contain potassium supplementation.

Hyperkalaemia

Hyperkalemia can result in life-threatening cardiac arrhythmias, and thus requires rapid and aggressive treatment.

- Intravenous administration of sodium bicarbonate (1 mEq/kg diluted in 5% dextrose), 5% dextrose in water (0.5 mL/kg), or regular insulin (0.1 IU/kg) and 5% dextrose (0.5 mL/kg) may be used to drive potassium intracellularly and decrease serum potassium concentrations.
- If serum potassium concentration is >6 mmol/L or electrocardiogram abnormalities (e.g. tall peaked T waves, bradycardia, widened QRS complexes, prolonged PR intervals, or atrial standstill) are present, calcium gluconate (4 mg/kg IV diluted in 1 L 0.9% NaCl) should also be administered intravenously. This has no effect on the serum potassium concentration but has cardioprotective effects while the above therapy is initiated.
- Administration of non-potassium-containing intravenous fluids at 1–2× maintenance rates also helps to eliminate excess potassium via urinary excretion.

Other electrolyte derangements

Hypocalcaemia

Hypocalcaemia is frequently encountered in horses with GI disease, periparturient mares, and in blister beetle (cantharidin) toxicosis.

- If severe (total calcium less than ~6.0 mg/dL, ionized calcium <1 mmol/L), hypocalcaemic tetany is likely to be observed, and may progress to recumbency and seizures in severe cases.
- Specific calcium correction formulas are not established for horses. In a clinical setting, hypocalcaemia is typically corrected by intravenous administration of 1 mL/kg 23% calcium gluconate. Calcium should be diluted in isotonic fluids or administered very slowly intravenously to avoid development of cardiac arrhythmias.

Hypomagnesaemia

Hypomagnesaemia often accompanies hypocalcaemia in horses and is frequently seen in anorectic horses or those with GI disease.

- Clinical signs associated with hypomagnesaemia include tachycardia, cardiac arrhythmias, muscle weakness, and sedation. Interestingly, the clinical signs of hypermagnesaemia are essentially identical.
- Estimation of total body magnesium status is difficult. In general, horses with total serum magnesium concentration less than about 1–1.5 mg/dL may show clinical signs and should receive magnesium supplementation.
- Magnesium supplementation in severe hypomagnesaemia is usually provided with intravenous magnesium sulphate at 50–100 mg/kg (25–50g per 500 kg horse) every 12–24 hours. Magnesium sulphate should be diluted in at least 1 L of sterile fluids for intravenous use.

Metabolic acidosis

Metabolic acidosis may result from loss of bicarbonate in the urine or faeces or from excess production of organic acids such as lactate.

- Mildly to moderately dehydrated horses often exhibit a mild metabolic acidosis and hyperlactataemia associated with poor tissue perfusion that resolves without specific treatment when dehydration is corrected.
- However, in severely acidotic horses or foals, such as those with a blood pH < 7.1, serum bicarbonate concentration <15 mmol/L, or a base deficit >10–15 mEq/L, alkalinizing therapy with sodium bicarbonate is indicated.
- The bicarbonate deficit in adult horses is calculated as follows:
  - HCO_3^- deficit (mEq) = (25 – patient HCO_3^-) × 0.3 × body weight (kg).
  - In foals, use 0.5 instead of 0.3 due to increased extracellular fluid volume.
  - Half the calculated deficit should be diluted in an isotonic fluid such as 5% dextrose that does not contain calcium or sodium, and administered as a slow IV bolus over approximately 1 hour. The remainder of the deficit may then be administered in IV fluids (supplemented with potassium – see above) over the next 12–24 hours.
  - In patients with significant ongoing bicarbonate losses, administration of significantly more bicarbonate than the initial deficit is often necessary, so frequent monitoring of electrolyte and acid–base status is vital.

Metabolic alkalosis

- Metabolic alkalosis is much less common in horses than metabolic acidosis, but may be seen in horses with excessive sweating or gastric outflow obstructions due to concurrent hypochloraemia.
- The alkalosis typically resolves without specific therapy when the hypochloraemia is corrected with administration of isotonic balanced crystalloids, though administration of acidifying fluids such as 0.9% NaCl can be helpful in severe cases.

Fluid therapy in neonatal foals: special considerations

Many of the above principles can be applied to both adult horses and foals, but the unique physiology of the neonate sometimes requires modification of the above strategies. These considerations are addressed in detail in the section on 'Evaluation and management of the sick neonatal foal'.

26.2 PATHOPHYSIOLOGY AND MANAGEMENT OF SHOCK

Definition of shock

- Shock occurs when cells are unable to produce enough energy to meet the demands of the cell to function.

- Most commonly the energy production is limited by inadequate oxygen.

Pathophysiology of shock

Most shock is the result of inadequate delivery of oxygen to the cell, although in some cases the cells may not be able to use the oxygen delivered.

Failure of O_2 delivery:

- Several equations are important to understanding the factors that can result in inadequate oxygen reaching the cell. Understanding what is causing inadequate oxygen delivery allows targeted therapy to improve oxygen delivery during the treatment of shock.
- Oxygen delivery (DO_2) is the product of cardiac output (Q) and arterial oxygen content (C_aO_2):

$$DO_2 = Q \times C_aO_2$$

  - Q is the product of stroke volume (SV) and heart rate (HR). SV is affected by the preload and afterload to the heart as well as the heart's contractility:

$$Q = HR \times SV$$

  - C_aO_2 is the product of oxygen carried by haemoglobin and dissolved in the blood (P_aO_2). The amount of oxygen carried by haemoglobin is the product of the amount of haemoglobin in the blood ([Hb]) and the saturation of that haemoglobin with oxygen (S_aO_2). It is very important to recognize that most of the oxygen in the blood is carried by haemoglobin

$$C_aO_2 = 1.34 \times [Hb] \times S_aO_2 + 0.003 \times P_aO_2$$

Failure of O_2 use:

- Rarely, cells are unable to utilize the oxygen delivered by the blood as a result of metabolic disease or toxicities such as cyanide.

Classifications and types of shock

Hypovolaemic shock

Hypovolaemic shock is caused by an insufficient circulating blood volume.

- Blood volume can be lost as whole blood (haemorrhage) or free water (with or without electrolytes and/or proteins).
- The fluid can be lost outside of the body or sequestered within the body, but outside of the circulation (third space).
- Prolonged lack of fluid intake can also result in hypovolaemia.
- Examples of conditions that can be associated with hypovolaemic shock include:
  - Haemorrhage – middle uterine artery bleed, laceration of a large artery associated with a fracture or wound.
  - Fluid loss – diarrhoea associated with colitis, gastric reflux associated with enteritis, sweating with exercise, or fluid leakage associated with extensive burns.
  - Third space loss – pleural or peritoneal effusions, or fluid trapped within the large colon secondary to a large colon volvulus.

Cardiogenic shock

Cardiogenic shock is caused by a failure of an effective cardiac pump. It is uncommon in horses, but examples of conditions that can be associated with cardiogenic shock include:

- Cardiomyopathy – monensin toxicity, viral cardiomyopathy.
- Arrhythmias – third-degree AV block, ventricular tachycardia.
- Valvular dysfunction – cordae tendinae rupture.

Distributive shock

This is caused by an insufficient circulating volume due to vasodilation and decreased systemic vascular resistance. Conditions such as endotoxaemia, sepsis, and anaphylaxis lead to the release of cytokines and inflammatory mediators that can result in distributive shock. Endotoxaemia results when endotoxin (also known as lipopolysaccharide, or LPS), a component of the cell wall of Gram-negative bacteria, enters the patient's systemic circulation due to bacterial death or proliferation.

- Circulating endotoxin interacts with a circulating host protein called LPS-binding protein (LBP) and a circulating or cell membrane receptor called CD14. This LPS–LBP-CD14 complex then binds and activates a cell surface receptor on leukocytes and endothelial cells called toll-like receptor 4 (TLR-4).
- Activated TLR4 initiates an intracellular signalling cascade that activates the transcription factor NFκB. NFκB enters the nucleus and stimulates the transcription of many proinflammatory mediators such as TNF-α, IL-1 and IL-6. These inflammatory mediators stimulate leukocyte adhesion and activation and endothelial cell activation, cause increased production of PGE2, ACTH, acute phase proteins, eicosanoids, and NO, and induce the release and activation of arachidonic acid metabolites, platelet-activating factor, reactive oxygen species, histamine, kinins and complement. Many of these substances have effects on vasomotor tone and vascular permeability, which can result in altered blood flow and distributive shock.
- Endotoxaemia can occur secondary to a variety of diseases in horses, including colitis, pleuropneumonia, and retained foetal membranes.

Sepsis

Sepsis is a condition that occurs in bacteraemic patients (those with bacteria in the bloodstream) when the body's inflammatory response to the infection becomes systemic (resulting in the systemic inflammatory response syndrome – SIRS).

- The inflammatory mediators associated with sepsis/SIRS include many of the same cytokines and result in many of the same effects as are described above for endotoxaemia.
- Sepsis is most common in foals (see Chapter 20), but may also occur in adult horses secondarily to bacterial infection or compromise in the gastrointestinal mucosal barrier.

Anaphylaxis

This is a systemic hypersensitivity reaction that results in mast cell degranulation, and thus, the release of large amounts of histamine, prostaglandins, and leukotrienes. Severe vasodilation can occur as a result. Anaphylaxis is uncommon in horses but can occur with drug or blood product administration.

Obstructive shock

This is caused by an obstruction to blood flow. Examples of conditions that can be associated with obstructive shock include:

- Diseases that restrict the heart's ability to expand such as pericardial effusion and restrictive pericarditis.
- Diseases that compress large vessels such as tension pneumothorax or large colon volvulus.

Metabolic shock

Metabolic shock is not related to altered blood flow but rather is a problem with blood oxygenation, tissue uptake of oxygen, cell utilization of oxygen, or cell demand for oxygen. Examples of conditions that can be associated with metabolic shock include:

- Decreased oxygen carrying capacity or hypoxemia – anaemia, respiratory diseases resulting in poor oxygen exchange.
- Impaired tissue uptake of oxygen/blood release of oxygen – carbon monoxide toxicity.
- Impaired cell utilization of oxygen – cyanide toxicity.
- Increased cell demand for oxygen – sepsis.

Stages of shock

The stages of shock are defined by how well the body is able to respond to the conditions resulting in shock by attempting to continue to meet the energy demands of the cell. This response is focused on meeting the energy demands of the 'most vital' structures such as the brain and heart.

- It is important to remember that these compensatory mechanisms are not without consequence (such as decreased perfusion to 'less vital' organs like the gastrointestinal tract), and will eventually become exhausted.
- These compensatory mechanisms include multiple physiologic responses that are triggered when the body detects the decreases in oxygen delivery, circulating volume, and mean arterial pressure including:
  - Fluid moves from the interstitial space into the vascular space causing an increase in blood volume.
  - Decreased mean arterial pressure is sensed by baroreceptors (aorta and carotid), triggering catecholamine release, which leads to arterial and venous constriction, increased myocardial contractility, and increased heart rate. It also results in increased antidiuretic hormone (vasopressin) secretion, which stimulates an increase in water resorption in the kidney to increase blood volume.
  - Decreased arterial pressure in the kidney results in activation of the renin-angiotensin-aldosterone pathway, which results in systemic vasoconstriction to increase blood pressure and increased sodium and thus water resorption to increase blood volume.
  - Catecholamine and ACTH release leads to increased circulating cortisol, which stimulates gluconeogenesis and protein catabolism to meet increased cellular energy demands.

Stage I: Compensated (also known as hyperdynamic) shock

- Compensatory mechanisms discussed above are able to maintain blood flow to heart and brain.
- Clinical signs: tachycardia, tachypnoea, hyperaemic mucous membranes, decreased capillary refill time (CRT), and bounding peripheral pulses.

Stage II: Early decompensated shock

- Compensatory mechanisms are unable to meet energy needs of tissue resulting in increased anaerobic metabolism, lactic acidosis, and organ dysfunction.
- Clinical signs: progressive tachycardia and tachypnoea, prolonged CRT, a 'toxic line' on mucous membranes, cold extremities, decreased urine production, and altered mentation.

Stage III: Late decompensated (also known as irreversible) shock

- As anaerobic metabolism continues, sympathetic arterial and venous constriction is overwhelmed, resulting in blood pooling in venules, fluid leaking into interstitium, and activation of inflammatory cells. Eventually, this leads to organ failure and death.
- Clinical signs: marked hypotension, bradycardia, circulatory collapse, pale/grey mucous membranes, and organ failure.
- This stage is often fatal, even with aggressive treatment.

Management of shock

Treatment of shock is directed at improving oxygen delivery to tissues and correcting the underlying disease. Therapies designed to improve oxygen delivery are targeted at restoring cardiac output and maximizing oxygen content in the circulation. Due to the number of diseases that can result in shock, a wide variety of therapies may be used to correct the underlying disease

Therapies directed at restoring cardiac output

As described above, cardiac output is the product of heart rate and stroke volume. The majority of therapies utilized in most types of shock are directed at restoring stroke volume rather than altering heart rate, unless a pathological cardiac arrhythmia is present (see Chapter 7).

Intravenous fluid resuscitation, which increases preload, is the mainstay of therapy to restore stroke volume. A variety of fluids can be used for fluid resuscitation, and combinations of the fluid types may provide the best results.

- Isotonic crystalloids, which are widely available and easy to use, can be administered at a shock dose of 90 mL/kg.
  - Initially, a bolus of approximately ¼ of this dose can be given and the status of the patient reassessed.
  - However, rapid administration of the large volumes required can be difficult in horses. In addition, due to redistribution to the extravascular space, only 25% of the volume administered remains in the intravascular space after 1 hour.
- Hypertonic saline solution (2–4 mL/kg IV bolus) can also be used to rapidly increase intravascular volume (2–4 L for every 1 L of hypertonic saline solution administered).
  - Hypertonic saline solution accomplishes this by drawing fluid out of the interstitium, though this fluid eventually redistributes, resulting in only short-term (<1 hour) volume expansion.
  - Thus, to maintain the volume expansion and rehydrate the interstitium, large volumes of isotonic fluids (10 L for every 1 L administered) must be given concurrently with hypertonic saline solution.
- Colloids (10–20 mL/kg) can also be used for fluid resuscitation for rapid volume expansion.
  - Colloids are fluids that have high colloid oncotic pressure (COP) due to high-molecular-weight molecules. These molecules remain within the intravascular space to pull and maintain fluid intravascularly, providing more sustained volume expansion compared to crystalloids.
  - Colloids can be synthetic or natural (e.g. blood products). In general, synthetic colloids have higher COP than natural colloids (with the COP of normal blood), and therefore provide more volume expansion. Additionally, natural colloids carry a risk for transfusion reactions and therefore cannot be administered as rapidly as synthetic colloids. However, some synthetic colloids may interfere with coagulation at high doses.

Fluid administration is not without risk in certain conditions associated with shock.

- Leakage of fluid into the interstitial space can occur with administration of crystalloids in diseases associated with hypoproteinaemia (colitis) or impaired vascular function (endotoxaemia, lung contusions, SIRS).
- In cases of uncontrolled haemorrhagic shock, rapid volume expansion can disrupt tenuous blood clots. In these cases, low-volume fluid resuscitation coupled with the administration of blood/plasma products to support coagulation is preferable to rapid, high-volume fluid resuscitation.

If, and only if, volume replacement fails to restore adequate tissue perfusion, pharmacological cardiovascular support with pressors and inotropes may be required.

- In equine critical care, pressors and inotropes are most often used in neonatal foals at referral institutions, and are very rarely used in standing adult horses.
- However, as many of the disease conditions associated with shock in horses are associated with decreased vasomotor tone and may have effects on the myocardium, it is possible that some cases could benefit from such pharmacological support.
- Treatment with pressors and inotropes requires intensive monitoring and care, and detailed discussion of their use is beyond the scope of this text.

Therapies directed at maximizing the oxygen content in the blood

Intranasal oxygen insufflation increases the concentration of oxygen in inspired air, thus increasing the driving pressure pushing oxygen from the alveoli into the pulmonary capillaries. This increases the partial pressure of oxygen in the arterial circulation, and in turn raises the driving pressure that pushes oxygen from the blood into the tissues. It is important to remember that dissolved oxygen makes up a very small portion of the oxygen carried in blood, so the effect of intranasal oxygen may be minimal. However, even small changes can be important in cases of shock.

In cases where the oxygen-carrying capacity is limited by anaemia, administration of red blood cells (whole blood or packed red blood cells) or purified haemoglobin dramatically improves the oxygen content of the blood (see section on 'Administration of blood products in equine critical care').

Therapies directed at correcting the underlying disease

Resolution of the underlying aetiology resulting in shock is vital to the successful management of patients with any type of shock. Specific treatments are as varied as the diseases that can result in shock, and thus are addressed more completely in the chapters addressing specific primary diseases.

N.B. Therapy for cardiogenic shock differs from the management of other types of shock, and many of the treatments for other types of shock worsen cardiogenic shock. Therapy to restore oxygen delivery in cardiogenic shock is directed at improving the function of the heart (contractility), correcting cardiac arrhythmias, and decreasing stresses on the heart (volume and afterload). As noted, cardiogenic shock is uncommon in horses.

Monitoring

Assessment of patients in shock involves the use of physical examination, laboratory analyses, and monitoring physiological parameters.

Many of the clinical signs of shock are monitored best with serial physical examination.

Particularly important parameters to assess include heart rate, mucous membrane colour, capillary refill time, pulse quality, respiratory rate, rectal temperature, temperature of extremities, and mentation.

- Monitoring urination can also provide information on perfusion and hydration.
  - In properly functioning kidneys, urine production is a crude estimate of blood flow to the kidney.
  - Urine specific gravity can also be measured to provide information regarding hydration status and renal function.
- Assessment of plasma lactate concentration and blood gas parameters can also be helpful.
  - Tissues that are not receiving adequate oxygen turn to anaerobic metabolism to meet energy demands, resulting in hyperlactataemia and acidosis.
  - Tissues that are not receiving adequate oxygen extract more of the oxygen carried in the blood as it passes through, resulting in a decrease in venous oxygen saturation.
- Central venous pressure (CVP) is the pressure within the greater vessels feeding the heart and is measured via a pressure transducer attached to a catheter placed through the jugular vein into the cranial vena cava or right atrium.
  - The primary determinants of CVP are venous return and cardiac function – volume depletion causes a low CVP, and poor cardiac function results in a high CVP. In standing horses CVP should be between 4 and 12 cm H_2O.
  - CVP can be helpful in assessing whether adequate volume resuscitation has been performed and in preventing excessive volume expansion, especially in patients with heart disease.
- Arterial blood pressure can be measured directly with an arterial catheter or indirectly using oscillimetry or Doppler flow. Mean arterial pressure should be approximately 100 mmHg in a standing adult horse and above 60 mmHg in a foal.

26.3 ADMINISTRATION OF BLOOD PRODUCTS IN EQUINE CRITICAL CARE

Commonly used blood products

Fresh-frozen equine plasma and fresh whole blood are the most commonly used blood products administered in equine critical care.

Plasma

Plasma is used primarily in the following circumstances:

- Provision of colloidal oncotic support to patients with hypoproteinaemia or shock.
- Provision of coagulation factors in coagulopathic patients.
- Provision of endogenous 'anti-endotoxic' factors such as anti-LPS antibodies and provision of immune support to patients at risk for, or with signs of, endotoxaemia or sepsis.
- Provision of immunoglobulins to neonatal foals at risk for, or with evidence of, failure of passive transfer or immunity.

Whole blood

Whole blood is used to provide oxygen-carrying capacity in patients with haemorrhagic shock or severe anaemia. Fresh whole blood also provides functional platelets and active clotting factors if collected properly.

Products used in specific circumstances

Fresh plasma, washed or packed red blood cells, purified haemoglobin products, or hyperimmune serum or plasma products are sometimes used in specific circumstances.

- Fresh plasma, rather than frozen plasma or whole blood, is indicated when active functional platelets but not erythrocytes are required, such as in horses that are thrombocytopenic but not anaemic.
- Concentrated platelet preparations (platelet-rich-plasma) are often used in humans and small animals, and can be prepared with equine plasma, but are rarely used in horses due to the associated expense.
- Washed erythrocytes are most often administered to neonatal foals with neonatal isoerythrolysis (NI) (see Chapter 20). In these cases the dam's blood is most often used. The dam's plasma contains antibodies against the foal's erythrocytes, so it must be removed by washing the cells prior to the transfusion.
- Purified bovine haemoglobin preparations (such as Oxyglobin®) can be used in horses requiring oxygen-carrying capacity for which a compatible blood donor is not available.
- Hyperimmune plasma or serum products consist of the plasma or pooled serum from horses that have been hyperimmunized against a particular substance to provide disease-specific antibodies in high concentrations.

Blood transfusions

Indications for blood transfusion

Whole-blood transfusion is often required in the initial management of severe or acute anaemia (see Chapter 10 for a discussion of the causes of anaemia in horses).

Short-term fluid resuscitation with isotonic crystalloids and colloids should be initiated in cases with severe hypovolaemia and haemorrhagic shock while a blood transfusion is being organized. However, hydroxyethyl starch should be avoided or used with caution for resuscitation in active haemorrhage as it may contribute to impaired haemostasis.

- Significant rapid volume expansion with a large volume of isotonic fluids or hypertonic saline solution is controversial in patients with ongoing haemorrhage, as a rapid increase in systemic blood pressure may perpetuate bleeding. Initial fluid therapy in an anaemic patient should thus be aimed at restoration of hydration and normovolaemia with crystalloids and/or colloids to maintain peripheral perfusion, with subsequent or concurrent administration of an oxygen-carrying fluid to restore oxygen-carrying capacity if indicated.
- Whole blood transfusion should be considered when the HCT reaches <18–20% in acute or ongoing haemorrhage, or <12–14% over a longer period. It is important to note, though, that fatal acute haemorrhage can occur without a significant drop in haematocrit; thus, given the significant variations in haematocrit and plasma protein concentrations that can occur with both acute and chronic anaemia, the need for whole blood transfusion should be determined by factors other than a specific haematocrit value.
- Tachycardia, tachypnoea, pale/grey mucous membranes, and poor pulse quality are consistent with haemorrhagic shock and indicate the need for transfusion regardless of the haematocrit.
- Persistent tachycardia and/or tachypnoea in the normovolaemic anaemic patient (with slower haemorrhage, haemolysis, or erythropoietic failure) suggest ongoing tissue hypoxia due to anaemia.
- In either acute or chronic anaemia, the presence of the following laboratory abnormalities also suggest decreased oxygen-carrying capacity and indicate the need for blood transfusion:
  - Hyperlactataemia (plasma lactate >2–4 mmol/L in a euvolaemic patient).
  - Decreased *venous* oxygen pressure (P_vO_2 <30 mmHg) or saturation (S_vO_2 <50%).
    N.B. Venous samples for blood gas analysis should be rapidly collected from a large vein directly into a heparinized syringe, anaerobically sealed, and immediately analyzed to provide accurate results.
    These findings indicate the need for blood transfusion to increase oxygen-carrying capacity even in the normovolaemic patient.

General considerations for whole blood transfusion

At present, fresh whole blood obtained from a healthy equine donor remains the oxygen-carrying fluid of choice in equine medicine, as banked equine blood is not currently available to most clinicians, and synthetic haemoglobin products are usually prohibitively expensive at the large volumes needed in horses.

There is an extraordinarily large number of equine blood types, and no universal equine blood donors; however, a first-time transfusion can usually be safely performed without cross-matching in an emergency situation. In these cases, a healthy gelding of a similar size and breed as the recipient is the ideal donor.

- Broodmares are poor donor choices as they are likely to have developed alloantibodies when exposed to other blood types through breeding/foaling.
- In horses requiring multiple transfusions, and in donkeys and mules, major (donor erythrocyte, recipient plasma) and minor (donor plasma, recipient erythrocytes) cross-matching should be performed prior to transfusion to ensure compatibility.
- If a compatible donor is not available, administration of a synthetic haemoglobin product is often the only option for provision of oxygen-carrying capacity to those patients. Autotransfusion is occasionally performed in patients with aseptic haemorrhage into a body cavity, though this is not recommended in haemoperitoneum in the postoperative colic due to recent celiotomy/enterotomy.

Whole blood collection and administration

Blood should be collected from the donor from a large (12–14) gauge catheter or blood collection set, using an aseptic technique, into sterile bags or bottles anticoagulated with sodium citrate or acid citrate dextrose (140 mL of 2.5–4% solution per 1 L blood) (Figure 26.2).

- 20–25% of the donor's blood volume (~10 L for a 450 kg horse) can be safely removed.
- Whole blood can be stored for several days to weeks in plastic containers at 4°C (39.2°F) if alternative anticoagulants are used (citrate phosphate dextrose with or without supplemental adenine), but adequate platelet function is ensured only in samples stored in plastic for ≤3 days.

Calculation of an appropriate amount of blood to administer can be difficult in ongoing haemorrhage. Replacement of 20–50% of the estimated blood loss is recommended, but this is difficult to quantify in most cases. The following formula is useful for estimating the blood volume needed:

$$\frac{\text{Desired PCV} - (\text{recipient PCV} \times 0.08) \times \text{body weight in kg})}{\text{Donor PCV}} = \text{litres of whole blood}$$

Figure 26.2 (a) Supplies for collection of fresh whole blood for blood transfusion, including (from left to right) sterile blood collection bag, sterile evacuated 2-litre blood collection bottle, and blood administration set with filter. (b) Collection of fresh whole blood from an equine donor into a sterile evacuated 2-litre blood collection bottle containing acid-citrate–dextrose as an anticoagulant. A double-ended large-bore blood collection set is placed in the donor's jugular vein after aseptic preparation and distension of the vein with a tourniquet, and is then connected to the evacuated bottle so that the bottle's vacuum is maintained to facilitate blood collection.

- Whole blood should be administered via a filtered blood administration set at a maximal rate of 10–20 mL/kg/hour.
- Initial administration should be very slow, and the horse should be monitored closely for a transfusion reaction – evidenced by piloerection, agitation, tachypnoea, pyrexia, urticaria, and/or muscle fasiculations – during the entire administration period.
- If no evidence of transfusion reaction occurs in the first 20–30 minutes, the rate may be gradually increased as above, with continued monitoring of vital parameters and attitude every 20–30 minutes.
- Mild reactions may be treated by transient cessation of blood administration for 30–60 minutes, with re-initiation at a slower rate. More severe reactions may require treatment with doxylamine succinate (0.5 mg/kg i.v. slowly, monitor for agitation) and/or corticosteroids (methylprednisolone sodium succinate at 1–2 mg/kg i.v. or dexamethasone at 0.05–0.1 mg/kg i.v.). Severe reactions and anaphylaxis should be treated with epinephrine (0.01–0.02 mg/kg i.v. or i.m.).
- It is important to note that the life-span of allogenic transfused erythrocytes is just 2–4 days on average; therefore, serial transfusions may be required in patients with severe anaemia, ongoing haemorrhage, and/or impaired erythropoiesis.

Supportive care and other management considerations in the anaemic patient

- Isotonic fluid therapy should be employed to maintain hydration and normovolaemia; it is important to remember that while this may actually decrease the HCT, the mild haemodilutive effect does not decrease the oxygen-carrying capacity (the actual number of erythrocytes remains the same) and can actually improve tissue oxygenation by improving peripheral perfusion.
- Patients with haemorrhage into a body cavity should be maintained on broad-spectrum antimicrobial therapy until the haemorrhage is resolved, as blood provides an ideal medium for bacterial growth.
- Intranasal oxygen insufflation (5–10 L/min) is not required but may be helpful in the severely anaemic patient, as increasing dissolved oxygen in the blood may slightly improve tissue oxygenation.
- Stress and excitement should be minimized in all anaemic patients, especially those with ongoing haemorrhage; sedation may be required in agitated horses, though the effects of both phenothiazine tranquillizers and alpha-2 adrenergic agonists on cardiovascular parameters can be detrimental in severely anaemic patients.
- Finally, appropriate analgesic therapy to control pain associated with haemoperitoneum or intraabdominal haematoma is vital. Flunixin meglumine, lidocaine, naloxone, and/or butorphanol as indicated by the horse's clinical condition are commonly employed analgesic options. Butorphanol and naloxone should not be used concurrently.

Treatment of the underlying cause

While a whole blood transfusion is often a life-saving step in the management of the anaemic patient, treatment of the underlying cause of anaemia is also imperative. Management of specific causes of anaemia are detailed in Chapter 10.

Plasma transfusions

Indications for plasma transfusion

The primary use of plasma in equine medicine is to provide colloidal oncotic support in patients with hypoproteinaemia and decreased colloidal oncotic pressure (COP).

The most common cause of hypoproteinaemia in horses is enteric protein loss, due to:

- Intestinal mucosal damage in inflammatory intestinal disease (enteritis or colitis) or with strangulating intestinal lesions.
- Intestinal malabsorptive disease.

In addition, significant hypoproteinaemia can occur with blood loss. While whole blood transfusion does replace both plasma proteins and red blood cells, sometimes additional plasma transfusions are necessary to support COP in severe blood loss anaemia, even after adequate oxygen carrying capacity is restored with whole blood.

If enteric protein loss is ruled out, renal protein loss should be considered, though this is much less common in horses than in other species.

- Albumin provides the majority of COP; thus, the degree of hypoalbuminaemia is more important than the degree of overall hypoproteinaemia.

The most common clinical sign in patients with clinically significant hypoproteinaemia is distal limb oedema and/or ventral oedema, due to fluid leakage into the interstitium as a result of low COP. In addition, horses may show clinical signs related to hypovolaemia such as tachycardia, prolonged CRT, slow jugular refill, decreased urine production, or depressed/altered mentation. However, the presence of clinical signs is quite variable, and clinical signs may be absent in some horses with extremely low total protein and albumin concentrations.

- Colloidal support should be considered in any patient with hypoproteinaemia/hypoalbuminaemia and the above clinical signs.
  - If possible, assessment of plasma COP can be helpful for guiding therapy. Normal COP is 19–26 mmHg in adult horses and 15–23 mmHg in foals.
  - Fresh-frozen plasma or fresh plasma (if viable platelets are needed) is also indicated to provide clotting factors in patients with documented coagulopathies.

In addition, sometimes smaller volumes of fresh frozen plasma or the afore-mentioned hyperimmune serum products are used to provide immune/anti-endotoxic support in horses with endotoxaemia/sepsis, or to provide specific antibodies in horses with certain diseases such as botulism.

Plasma transfusion is also indicated in neonatal foals ≥12–24 hours old with failure of passive transfer of immunity (serum immunoglobulin concentration <800 mg/dL by 24 hours of age).

Plasma and serum products

Fresh-frozen plasma (FFP)

- This is the most commonly used plasma product in equine medicine, and is commercially available from a number of different suppliers.
- FFP provides plasma proteins and the majority of functional coagulation factors, but does not provide functional platelets.

Fresh plasma (FP)

- FP is used when viable functional platelets are necessary in thrombocytopenic patients.
- FP must be processed from fresh whole blood and administered within 24–48 hours of collection.

- Thus, fresh plasma is not commercially available and can be quite labour intensive to process.

High-gammaglobulin plasma

- High-gammaglobulin plasma is simply FFP that has had its immunoglobulin concentration quantified as greater than a specific cut-off concentration (specific cut-off concentrations vary with manufacturer).
- This is used most often to treat failure of transfer of passive immunity in neonatal foals.

Hyperimmune plasma and serum

- Hyperimmune plasma and serum products are commercially produced from horses that have been repeatedly immunized against specific agents. Thus, hyperimmune plasma/serum has high concentrations of specific antibodies.
  - Commonly used hyperimmune equine products include *Salmonella* antiserum and hyperimmune *Escherichia coli* plasma, which are often used in horses with gastrointestinal disease at risk for, or showing signs of, endotoxaemia, and polyvalent botulinum toxin antiserum, which is used to treat clinical botulism.

Administration and monitoring

- Frozen plasma should be thawed in a warm water bath just prior to administration.
- A plasma dosage of 20–40 mL/kg is recommended for initial management of hypoproteinaemia/decreased COP, and for hypogammaglobulinaemia in foals. An improvement in clinical signs may be noted with this dose, even if a significant increase in plasma protein or albumin concentration is not apparent. However, in diseases with ongoing protein loss, significantly larger volumes of plasma or repeat transfusions may be necessary.
- Plasma should always be administered via a filtered blood administration set.
- Initial administration of plasma should be slow, and the patient should be monitored closely for any signs of a transfusion reaction (as for a whole blood transfusion reaction above).
- If no evidence of a transfusion reaction is present in the first 15–30 minutes of administration, the infusion rate may be gradually increased up to 10 mL/kg/hour.
- Hyperimmune plasma products are typically administered in a manner similar to that used for administration of standard equine plasma products.
- Hyperimmune serum products are often diluted in crystalloid fluids and administered fairly slowly, but as administration methods can vary, the manufacturer's recommendations should be followed.

Alternatives to plasma – synthetic colloids

- When colloids are needed for fluid resuscitation, synthetic colloids are often preferable to plasma products due to the availability and ease of administration of these synthetic products. Administration of synthetic colloids for fluid resuscitation is described in the 'Pathophysiology and management of shock' section above.
- In severely hypoproteinaemic patients with persistently low COP, synthetic colloids may also provide better and more economically feasible oncotic support than plasma alone. In these patients, synthetic colloids are typically administered as intermittent intravenous boluses of 5–10 mL/kg q 12–24 hours, or as a constant rate infusion at 0.5–1 mL/kg/hour.

26.4 NUTRITIONAL SUPPORT IN THE CRITICALLY ILL HORSE AND FOAL

Introduction and key points

- Studies in human and small-animal critical care have shown that provision of adequate nutrition early in the course of illness is associated with decreased morbidity and mortality in critically ill patients. While similar work has not yet been conducted in horses, the same probably holds true in equine critical care.
- Nutritional support provides adequate substrate for basic cellular metabolism and for tissue healing and repair and prevents the deleterious endocrine and metabolic sequelae associated with the development of a negative energy balance.
- Nutritional support in the critically ill horse or foal may be provided enterally (via the gastrointestinal tract), parenterally (intravenously), or by some combination of the two. In most cases, enteral nutrition is preferable to parenteral nutrition for both economic and physiological reasons discussed in detail below.

Nutritional requirements

- Nutritional requirements needed to maintain body weight in healthy, inactive adult horses have been calculated to be approximately 33–40 kcal/kg/day.
- While illness can alter energy requirements, no good data exist to define a specific degree to which energy requirements are increased or decreased with a certain type of illness or trauma.
- Thus, nutritional requirements in critically ill horses are typically provided according to maintenance energy needs. This is most often expressed as digestible energy (DE).
- The digestible energy amount required to meet maintenance energy needs in a normally active, non-working adult horse is the maintenance DE (DEM) and is calculated based on the horse's body weight in kilograms (BW):
  In horses ≤600 kg: DE_M (Mcal/day) = (BW)(0.03) + 1.4.
  In horses >600 kg: DE_M (Mcal/day) = [1.82 + (0.0383)(BW)] – [(0.000015)$(BW)^2$].

In horses that are not active (e.g. confined to a stall), meeting the resting energy requirement (DE_R), which is approximately 70% of the DE_M, may be sufficient to maintain body weight. The DE_R = (BW)(0.021) + 0.975.

Increases in energy requirements attributable to illness are often offset by decreased requirements due to inactivity in critically ill horses; thus, provision of energy between the DE_R or the DE_M is adequate for most critically ill horses.

In addition, it is important to ensure that protein requirements are met to prevent protein catabolism. Daily protein requirements for an adult horse equal 0.5–1.5 g protein/kg/day. However, because protein catabolism can limit tissue healing, and can be a significant problem during critical illness, protein should be provided at 1–2 g/kg/day to the majority of critically ill horses as long as hepatic function is adequate.

Enteral nutrition

- The presence of feed components and nutrients in the intestinal lumen is vital for the health of the enterocytes and maintenance of the normal enteric flora population. Intestinal mucosal atrophy and bacterial translocation across the bowel mucosa occur rapidly within just a few days of fasting.

- Thus, some amount of enteral feeding should be provided if it is tolerated by the patient.
- Anorexic or dysphagic patients that are unwilling or unable to eat may be provided with enteral nutrition via an indwelling nasogastric tube or an oesophagostomy tube. If adequate nutrition cannot be provided enterally to these patients, the difference may be made up with parenteral nutrition.
- A variety of commercial equine diets are available for administration via an indwelling nasogastric tube.
  - Most types of complete pelleted feeds can be prepared as a slurry and administered via a medium to large bore tube, but are usually not suitable for use with smaller bore tubes.
  - Liquid diets specially formulated for equine critical care are also available and, while expensive, are easy to administer through small-bore feeding tubes.

Parenteral nutrition

Circumstances necessitating parenteral nutrition

- Patients with prolonged gastric reflux associated with ileus or proximal duodenitis-jejunitis.
- Dysphagic or anorexic patients in which enteral nutrition via indwelling nasogastric tube or in which oesophagostomy is not possible or is poorly tolerated.
- Critically ill foals in which enteral feeding at maintenance rates is not tolerated or desirable.

Types of parenteral nutrition

Intravenous fluids containing dextrose

For short periods of time (1–2 days maximum), caloric needs can be met by administration of a dextrose containing crystalloid fluid intravenously. In these circumstances, 50% dextrose (0.5 mg/mL) can be added to balanced polyionic crystalloids to create a 1–10% dextrose solution.

- The resultant solution are hyperosmolar, but the degree of hyperosmolarity is typically tolerated by both central and peripheral veins as long as the percentage of dextrose in the solution is not exceeded.
- Dextrose-containing solutions should be administered at a rate that provides dextrose at 4–8 mg/kg/minute.
- However, provision of caloric needs solely in the form of carbohydrates is unacceptable for long periods of time, as administration of amino acids and lipids is necessary for long-term nutritive support.

Partial parenteral nutrition (PPN)

- PPN formulations meet some but not all nutritional requirements, and most often contain dextrose and amino acids. Vitamin and mineral supplementation may also be included.
- PPN solutions are used most often in horses requiring parenteral nutritional support for some duration of time that either cannot tolerate lipid infusions due to hyperlipidaemia or in which provision of total parenteral nutrition (TPN, see below) is cost prohibitive.
- In general, PPN solutions are formulated to provide 3.5–5 g/kg/day of dextrose and ~1 g/kg/day of amino acids.
- PPN is rarely used in foals.

Total parenteral nutrition (TPN)

- TPN formulations contain dextrose, amino acids, and lipids, as well as vitamin and mineral supplements, and provide full nutritional requirements.
- TPN is indicated in any patient requiring parenteral nutritional support for longer than 24–48 hours (as long as that patient can tolerate lipid administration).
- Ideally, 30–40% of caloric support should be provided in the form of lipids with the remaining 60–70% of the calories provided with dextrose. A non-protein calorie-to-nitrogen (protein) ratio of 100–150 kcal per gram of nitrogen should be maintained to ensure that amino acids are spared for use as an energy source and are thus available for tissue healing and repair.
- Recommended TPN formulations include 5–8 g/kg/day of dextrose, 1–2 g/kg/day of amino acids, and 1–1.3 g/kg/day of lipid.
- 50% dextrose provides 3.4 kcal/g, 20% lipid emulsions provide 9 kcal/g, and 8.5% amino acid solution provides 4 kcal/g.

Important considerations in the administration of parenteral nutrition

Preparation and administration

- TPN and PPN are extremely hyperosmolar if not diluted. Thus, any parenteral nutrition formulation should be diluted with sterile water to a total osmolarity of ~600 mOsm/L (~ twice plasma osmolarity).
- Parenteral nutrition preparations should be administered through a non-thrombogenic catheter in a large vein.
- Large volume or rapid administration of parenteral nutrition formulation is not possible due to the osmolarity and high glucose concentrations; thus, concurrent crystalloid fluid therapy is usually necessary to maintain fluid balance during PPN/TPN administration.
- Due to the potential for bacterial growth in PPN/TPN solutions, strict asepsis should be adhered to when preparing, handling, and administering these formulations.
  - A dedicated catheter or catheter port that is used only for PPN/TPN administration should be maintained, and the vein monitored vigilantly for any evidence of thrombophlebitis.
  - Administration sets should be changed daily, and PPN/TPN solutions refrigerated until use and discarded after 24 hours of storage.

Rate of administration

- Parenteral nutrition solutions should be administered as a constant rate infusion using an infusion pump to avoid undesirable fluctuations in blood glucose from poor rate control.
- Once the PPN/TPN solution has been prepared and diluted to the appropriate osmolarity, the total volume of solution is divided by 24 hours to determine the full rate of administration in mL/hour.
  - Administration should begin at 1/4 to 1/3 of this rate for a period of 6–12 hours while the patient is monitored for the development of hyperglycaemia and glucosuria.
  - The rate may be increased by 25–33% every 6–12 hours with continued monitoring until the full rate is reached, typically 24–36 hours after initiation of parenteral nutrition.
  - If hyperglycaemia or glucosuria occurs during initial administration, the rate may be decreased by 1/3 to 1/2 for 6–8 hours before another attempt is made to increase it to the full rate.
  - If hyperglycaemia/glucosuria is persistent, concurrent insulin therapy may be required.

- When nutritional needs are able to be met with resumption of enteral feeding, the PPN/TPN rate should be gradually decreased by 1/3 to 1/2 every 6–12 hours to prevent a sudden drop in blood glucose.

Monitoring and complications

Hyperglycaemia, hypertriglyceridaemia, and electrolyte derangements are the most common complications in patients receiving parenteral nutrition.

- Blood glucose concentration should be monitored every 6 hours initially, and then every 8–12 hours once the patient is maintained at the full rate of PPN.
- Urine should be assessed once to twice daily for the presence of glucosuria if possible.
- Serum triglyceride concentration should be assessed prior to administration of PPN/TPN and then every 24–48 hours.
- Serum electrolyte concentrations should be measured daily in patients in PPN/TPN.
  - Hypokalaemia is a significant concern due to the potential for intracellular potassium flux with administration of high concentrations of dextrose. Thus, potassium should be supplemented in all patients receiving parenteral nutrition.
  - Hypocalcaemia, hypomagnesaemia, and/or hypophosphataemia may also occur in horses on PPN/TPN. Supplementation of calcium, magnesium and/or phosphorus should be considered if warranted.

Some other potential complications

- Acid–base disturbances.
- Hyperammonaemia (especially in patients with liver disease).
- Phlebitis.
- Septic thrombophlebitis or other septic complications such as pneumonia.

Nutrition in the sick foal: special considerations

Nutritional requirements in neonatal foals are different from those in adult horses, and affect both enteral and parenteral nutrition recommendations. These requirements are discussed in detail in the next section.

26.5 EVALUATION AND MANAGEMENT OF THE SICK NEONATAL FOAL

Introduction

- Management of the sick neonatal foal, which frequently presents with multi-systemic illness, encompasses many of the diverse aspects of equine critical care outlined thus far.
- In addition, a foal that initially appears stable can rapidly decompensate and die in a matter of hours.
- Thus, accurate assessment and aggressive treatment of the sick neonatal foal are vital to successful management of these most fragile equine patients.
- While specific foal diseases are discussed in detail in Chapter 20, the following section covers the general evaluation of the neonatal foal and general stabilization and management of a sick neonatal foal.

Evaluation of the neonatal foal

While a comprehensive history, systems-based physical examination, and ancillary diagnostic procedures are often similar in adult horses and foals, some important differences should be considered in evaluation of the neonatal foal.

History

- Factors related to the mare are important in assessment of potential risks to a newborn foal. Relevant history questions include:
  - Breeding date and expected parturition date?
  - Previous foals, including any with problems?
  - Any illness during gestation?
  - Recent travel or routine/diet changes?
  - Vaccinations before and during pregnancy?
  - Vaginal discharge prior to foaling?
  - Udder development and milk production, including milk dripping before parturition?
- In addition, factors related to parturition can dramatically affect the foal. Relevant questions include:
  - Parturition observed?
  - Duration of active labour from the time of amnionic rupture? (normal is <20–30 minutes)
  - Dystocia? If so, how severe and how resolved?
  - Placenta – when passed? Complete? Any abnormalities?
- Finally, the foal's progress towards normal landmarks during the first day of life and routine care after birth may be important in ruling in or out certain conditions. Relevant questions include:
  - Time to sternal recumbency? (normal is <5 minutes)
  - Suckle reflex present at or shortly after birth?
  - Time to standing (unassisted)? (normal is <60 minutes)
  - Time to nursing (unassisted)? (normal is <120 minutes)
  - Meconium passed? (within 8–12 hours)
  - Urination observed? (within 12–18 hours)
  - Routine foal care (varies from farm to farm):
    - Umbilical stump treatment?
    - Enema?
    - Tetanus antitoxin or other treatments?
    - IgG concentration assessed?
    - Other routine bloodwork?

Physical examination

- General appearance and attitude.
  - A healthy foal is bright and active, nurses aggressively and frequently with a strong suckle reflex, and typically resents restraint and handling. Any foal that is depressed or lethargic, spends long periods of time recumbent, has difficulty rising or nursing without assistance, or does not nurse frequently or well is NOT normal and should be evaluated carefully.
  - Poor body condition is often evident in premature or dysmature foals, and may indicate in utero growth restriction associated with placental insufficiency.
  - Other signs of prematurity or dysmaturity may include small size, a silky hair coat, floppy ears, a domed skull, and increased flexor tendon laxity.
- Vital parameters.
  - Normal rectal temperature = 37.5–38.8°C (100–102°F).
  - Normal heart rate = 80–120 bpm.
  - Normal respiratory rate = 20–40 bpm.

- Mucous membranes and integument.
  - Hyperaemic, injected mucous membranes are often seen in septicaemic foals. Hyperaemia of the skin in lightly pigmented areas may also be noted.
  - Petechial or ecchymotic haemorrhages on the oral mucosa, sclerae, vulva, or pinnae are also often present in foals with septicaemia, and are most likely due to a sepsis-associated vasculitis or alterations in peripheral blood flow rather than a concurrent coagulopathy.
  - Foals with neonatal isoerythrolysis (NI) often present with markedly icteric mucous membranes, though the absence of icterus does not exclude the potential of NI. Liver disease is another potential cause of icterus in the newborn foal, though this is substantially less common than NI.
  - The skin of neonatal foals is very fragile, and decubital ulcers over bony protuberances are quite common in foals that have been predominantly recumbent.
- Cardiovascular system.
  - Cardiac murmurs are commonly auscultated in neonatal foals and are most often non-pathologic physiologic flow murmurs. These typically are grade 1–3/5 systolic murmurs with a point of maximal intensity over the left heart base. Physiological flow murmurs typically disappear after the first few weeks of life.
  - While a continuous murmur associated with a patent ductus arteriosus is commonly heard immediately after birth, persistence of such a murmur beyond the first few days of life warrants further evaluation.
  - Other cardiac murmurs that are louder on the right than the left are accompanied by tachycardia, weakness, collapse, or cyanosis, those that persist beyond the first few weeks of life warrant further echocardiographic evaluation.
  - Distal limb temperature and peripheral pulse quality should also be assessed in neonatal foals, as cool limbs and/or weak peripheral pulses indicate poor perfusion and are often seen in foals with septicaemia/septic shock.
- Respiratory system.
  - Abnormal lung sounds are frequently absent even in severe pulmonary disease in foals, so further evaluation of the respiratory system with imaging or sampling is warranted in any foal with tachypnea or increased respiratory effort.
  - Premature foals or foals with neonatal encephalopathy may exhibit brief periods of apnea or irregular breathing.
- Gastrointestinal system.
  - The mouth and palate should be examined for congenital malocclusion or cleft palate.
  - Neonatal foals have a limited gastrointestinal capacity and normally appear 'thin-waisted' with a tucked-up flank during the first few weeks of life. Thus, any gross abdominal distension in a neonatal foal is cause for concern. The most common causes of abdominal distension in the neonatal foal include intestinal obstruction (most often meconium retention) uroperitoneum, or intestinal ileus associated with inflammatory intestinal disease (enterocolitis).
  - Foals may exhibit significant signs of abdominal pain with any of these conditions. In foals, abdominal pain is most often manifest by decreased nursing and more frequent recumbency, rolling into dorsal recumbency, bruxism, flagging of the tail, and straining to defecate.
- Urogenital system.
  - Neonatal foals with uroperitoneum may be observed to strain to urinate or may never be seen to urinate normally. However, many foals with a ruptured bladder continue to produce a normal urine stream. Thus, uroperitoneum should be considered in any neonatal foal with colic and/or abdominal distension regardless of the presence or absence of dysuria.
  - The umbilical stump should be evaluated for evidence of a patent urachus, which may be most evident by the presence of urine at the umbilicus only when the foal urinates.
  - In colts, both testicles are frequently descended at the time of parturition. However, the timing of testicular descent can be quite variable, and may occur as late as 12 months of age in certain breeds.
- Musculoskeletal system.
  - The limbs should be examined carefully for any evidence of flexural or angular limb deformities.
  - In addition, particular attention should be paid to the presence of any lameness, joint effusion, physeal enlargement or long bone pain, as these signs frequently indicate the presence of septic arthritis or physitis in a neonatal foal.
  - Palpate the ribs carefully for any evidence of rib fractures. Non-displaced rib fractures may be missed on palpation but can be identified with ultrasonography.
- Umbilical structures.
  - The umbilicus should be evaluated for the presence of any swelling, drainage (urine or purulent exudate), or associated hernias.
  - Normal appearance of the external umbilical stump does not eliminate the possibility of disease or infection affecting the deeper umbilical structures; complete evaluation of the umbilicus thus requires ultrasonography.
- Eyes.
  - Neonatal foals do not exhibit the (learned) menace response until they are ~3 weeks old. Thus, vision should be assessed by the ability of the pupils to respond to light and the foal's ability to orient in its environment and track the mare.
  - Corneal sensitivity may be decreased in neonatal foals; thus, fluorescein staining should be performed in any recumbent foal to evaluate for corneal ulceration.
  - Uveitis frequently occurs in septicaemic foals and may be evidenced by miosis, fibrin accumulation in the anterior chamber, hypopyon, and/or hyphaema. Ophthalmoscopic examination should be performed in every neonatal foal with suspected sepsis.

Ancillary diagnostics

The following should be considered as the minimum database for complete evaluation of any sick neonatal foal:

- Rapid blood glucose concentration and PCV/TS should be performed immediately in any recumbent, weak, or seizuring foal.
- Complete blood count and fibrinogen concentration.
  - Both leucopenia and leucocytosis may be seen in foals with septicaemia. In general, the presence of leucopenia and/or an increase in band neutrophils suggests acute infection, while neutrophilia and hyperfibrinogenaemia are consistent with an infection of longer-standing duration. If the latter are present within the first 24–48 hours of life, the possibility of in utero infection (perhaps due to placentitis in the mare) should be considered.
- Serum biochemistry profile.
  - In general, biochemical abnormalities with specific organ dysfunction in foals are comparable to abnormalities seen in adult horses, with a few important distinctions.

  - Neonatal foals may exhibit a significant degree of azotaemia in the first 24–48 hours of life due to placental insufficiency in late gestation. Thus, this should be considered as a cause of azotaemia in addition to pre-renal, renal, and post-renal causes in newborn foals.
  - Profound hypoglycaemia can develop rapidly in sick neonatal foals due to immature capacity for glucose synthesis and limited glycogen reserves.
  - Electrolyte derangements, especially sodium derangements, are common and can be quite severe in neonatal foals; thus, evaluation of serum electrolyte status should be performed before IV fluid administration if at all possible.
  - Serum total protein and globulin concentrations are lower in neonatal foals even if passive transfer of immunoglobulins is adequate.
  - Liver enzyme activity and serum bile acids are often higher in neonatal foals than in adult horses; thus, mild increases should not be a cause for concern unless they are persistent or occur with other signs of liver disease.
- Serum IgG concentration.
  - Should be performed in sick foals, even in foals previously shown to have adequate IgG concentrations, as active infection may result in consumption of immunoglobulins.
- Aerobic and anaerobic bacterial blood cultures.
  - Blood should be collected aseptically from the jugular or cephalic vein and immediately and aseptically placed into blood culture bottles containing bacterial culture media as directed by your laboratory.
  - Ideally, blood cultures should be collected prior to initiation of antimicrobial therapy, but if this is not possible, they should still be performed at the earliest convenience.

The following are frequently performed during initial evaluation of a foal with any localizing signs of disease, or in foals in which an infectious focus is suspected but not identified on physical examination:

- Plasma lactate concentration.
- Indirect blood pressure.
- Thoracic imaging – radiographs and/or ultrasonography.
- Abdominal imaging – radiographs and/or ultrasonography.
- Umbilical ultrasonography.
- Arterial blood gas analysis.
  - Should be performed in any foal with suspected or confirmed respiratory tract diseases, and in recumbent foals.
- Faecal diagnostics (in foals with diarrhoea).
  - PCR or culture for *Salmonella* spp.
  - Assays for *Clostridium difficile* and *C. perfringens* toxins.
  - ELISA or latex agglutination test for rotavirus antigen.
  - In some cases, evaluation for *Giardia* or *Cryptosporidium*.
- Trans-tracheal wash.
  - Performed less often in foals than in adult horses, as haematogenous pneumonia secondary to septicaemia is the most common cause.
- Radiographic evaluation of any effusive joints or enlarged physes.
- Synoviocentesis.

Stabilization and management of the sick neonatal foal

The primary goals for initial stabilization and management of a sick neonatal foal include the following key aspects:

- Restoration and maintenance of normovolaemia and normotension.
  - Fluid therapy of the neonatal foal is in general comparable to that of adult horses, as discussed in the fluid therapy and shock sections above.
  - Special considerations in fluid therapy in the neonate include:
    A higher maintenance fluid requirement (80–100 mL/kg/day).
    Inclusion of milk consumption and any plasma administered in the calculation of daily fluid requirements to avoid overhydration.
    Difficulty handling an excessive sodium load. Foals may become significantly hypernatraemic on some balanced polyionic crystalloids suitable for adult horses. In these foals, 0.45% sodium chloride/2.5% dextrose is usually a suitable maintenance fluid choice.
  - Vasopressor therapy is used more often in foals than in adult horses and is indicated in any foal with persistent hypotension following fluid resuscitation. Pressor therapy requires intensive monitoring and is beyond the scope of this text.
- Correction of hypoglycaemia, electrolyte disturbances, and acid–base derangements.
  - Administration of 2.5–20% dextrose solutions may be necessary for correction of hypoglycaemia, depending on the severity.
    Rapid or large volume administration of dextrose in water, even if isotonic, can result in significant electrolyte derangements and should be avoided. Small to moderate amounts of dextrose may be added to isotonic balanced polyionic crystalloids, but if large amounts or long duration dextrose supplementation are necessary, parenteral nutrition should be considered.
  - Foals that cannot nurse or be fed enterally should be maintained on parenteral nutrition or IV fluids supplemented with dextrose at 4–8 mg/kg/minute, as neonates have very limited glucose reserves.
  - Hypovolaemic or hypoxic foals are often hyperlactataemic; fluids containing lactate such as lactated Ringer's solution should be avoided in these cases.
  - Electrolyte and acid–base derangements should be corrected as described in the fluid therapy section above.
- Correction of failure of transfer of passive immunity.
  - Foals with partial or complete failure of transfer of passive immunity should receive equine plasma, as described in the above section on 'Administration of blood products in equine critical care'.
  - Due to ongoing immunoglobulin consumption during sepsis, repeat plasma transfusions may be required to fully resolve hypogammaglobulinaemia.
- Broad-spectrum antimicrobial coverage for suspected or confirmed sepsis.
  - In general, any sick neonatal foal should be considered at significant risk for sepsis. Broad-spectrum antimicrobial therapy should be initiated in any foal with an abnormal leukogram or hypogammaglobulinaemia, localizing signs of infection (diarrhoea, pneumonia, umbilical infection), and in any recumbent foal due to risks of aspiration pneumonia or urinary tract infection.
  - In addition, antimicrobial coverage should also be considered for foals with NI or neonatal encephalopathy, as well as seemingly healthy foals born to mares with placentitis, dystocia, or caesarean section, due to the increased risk of concurrent septicaemia in these groups of foals.
  - Intravenous administration is preferable to oral or intramuscular routes if septicaemia is highly suspected.

  - In general, first-line antimicrobials commonly selected for use in neonatal foals with suspected sepsis include:
    - Combination of a beta-lactam (e.g. penicillin) with an aminoglycoside (e.g. amikacin). Hydration status and renal function should be considered and monitored carefully in foals on aminoglycosides.
    - 3rd generation cephalosporin.
- Provision of adequate nutrition.
  - Foals that are willing and able to nurse and have no signs of colic should be allowed to nurse the mare freely. Orphan foals can be fed mare's milk or equine milk replacer via a bottle or pan every 2–4 hours.
  - Weak or recumbent foals that are unable to nurse adequately should be fed via an indwelling nasogastric tube. Small-bore tubes that allow the foal to nurse around them after the foal is stronger can be maintained for long periods of time.
  - Foals should receive 20–25% of their body weight in milk per day divided into ~12 feedings to grow adequately.
  - However, sick foals may have impaired gastrointestinal motility as well as compromised gastrointestinal tract defences. Thus, some clinicians elect to rest the GI tract in foals with severe FPT, leukopenia, diarrhoea, or suspected sepsis for 12–24 hours, as milk can be an excellent medium for pathogenic bacterial growth in a poorly defended GI tract. Small-volume feedings (2–5% of body weight in milk/day) are then initiated after appropriate serum IgG concentrations are established with plasma transfusion, and broad-spectrum antimicrobial therapy is initiated. If these small-volume feedings are well tolerated, the foal is then gradually increased to a full milk ration over the next several days.
  - Foals that have not been nursing for any other reason should also be gradually reintroduced to feeding in this manner.
  - Parenteral nutrition should be considered in any foal that cannot tolerate enteral feeding for 24–48 hours or more. TPN is preferable to PPN in most cases and is usually well tolerated in foals.
  - Osmolarity, non-protein calorie to nitrogen ratio, and administration recommendations for parenteral nutrition in foals are comparable to that of adults (see section on 'Nutrition in the critically ill horse and foal').
  - However, neonatal foals have higher energy requirements than do adult horses, at 40–60 kcal/kg/day. Thus, TPN formulations containing (as for adult horses) 5–8 g/kg/day of dextrose, 1–2 g/kg/day of amino acids, and 1–1.3 g/kg/day of lipid need to be administered at higher rates to foals to meet this requirement. (Reminder: 50% dextrose provides 3.4 kcal/g, 20% lipid emulsions provide 9 kcal/g, and 8.5% amino acid solution provides 4 kcal/g.)

General supportive/nursing care

- Recumbent foals should be maintained on clean, dry bedding or a soft, dry mattress to minimize the development of decubital ulcers.
- Recumbent foals, especially those with respiratory tract disease, should be maintained in sternal recumbency as much as possible to improve ventilation and minimize atelectasis.
- In addition, the foal should be assisted to stand and then returned to the opposite recumbency every 2 hours to aid musculoskeletal development and prevent decubital ulceration.
- Corneal lubrication should be applied several times a day to recumbent or obtunded foals to prevent corneal ulceration.

Gastric ulcer prophylaxis

Some evidence suggests that foals may be at an increased risk for development of gastric ulceration during hospitalization, and some clinicians routinely administer acid suppressants (proton pump inhibitors and/or H_2-blockers) and/or gastroprotectants (e.g. sucralfate) to all sick neonatal foals.

- However, other studies have not documented convincing evidence for acid-induced gastric ulceration or shown benefits for gastroprotectant therapy in sick neonatal foals.
- In addition, maintenance of an acid gastric pH is an important gastrointestinal defence mechanism.
- Thus, current recommendations generally suggest limiting gastroprotectant use to neonatal foals with evidence of gastrointestinal disease or colic, foals on NSAIDs, and older foals with evidence of gastroduodenal ulcer disease (see Chapter 20).

Additional management

Additional management may be required for foals with certain specific problems, including the following (see Chapter 20 for more details):

Colic

- Tube feeding/nursing should be withheld and hydration and normoglycaemia maintained with IV fluids and/or parenteral nutrition.
- Gastric decompression should be maintained with an indwelling or intermittently passed nasogastric tube.
- Specific causes of colic such as meconium impaction or intussusception should be treated accordingly (see Chapter 20).

Respiratory disease

- Intranasal oxygen insufflation should be provided in hypoxaemic foals.
- Mechanical ventilation may be necessary if hypoxaemia persists and/or hypercapnia develops due to ventilatory compromise.
- Nebulization with saline solution is sometimes used to aid in clearance of lower respiratory tract secretions. Antimicrobials, bronchodilators, and/or mucolytics may also be nebulized to deliver these medications directly to the lungs in foals with severe pneumonia.

Neonatal isoerythrolysis (NI)

Administration of whole blood or the mare's washed red blood cells is often necessary to correct anaemia and provide oxygen-carrying capacity in foals with NI.

Neonatal encephalopathy

- Anti-inflammatory medication such as flunixin meglumine or corticosteroids, free radical scavengers such as dimethylsulfoxide and antioxidant vitamins (vitamins C and E), and/or diuretics such as mannitol are often administered to foals with neonatal encephalopathy to decrease CNS inflammation and cerebral oedema.
- However, our current understanding of the specific pathophysiology of neonatal encephalopathy is quite limited, and there is no clear evidence that these drugs are effective or necessary in these foals.

Septic arthritis/septic physitis

- Septic arthritis should be managed with frequent and large volume lavage of affected joints with sterile isotonic fluids and

administration of intra-articular antimicrobials in addition to systemic antimicrobials.
- Intravenous regional perfusion with antimicrobials is also often performed in an attempt to increase antimicrobial penetration into affected joints/physes.
- Intraphyseal antimicrobial injection is sometimes utilized.
- Surgical debridement of affected joints/physes is often necessary in severe cases.

26.6 MANAGEMENT OF ACUTE RESPIRATORY DISTRESS (See also Chapters 5 and 6)

Causes of respiratory distress

Causes of respiratory distress can be separated into diseases of the upper and lower respiratory tract.

Upper respiratory tract

The upper respiratory tract (URT) includes the nares, nasal passages, pharynx, larynx, and trachea.

- The nasal passage must be obstructed bilaterally to produce respiratory distress. Choanal atresia is a congenital disease where tissue (bone or soft tissue) obstructs the caudal aspect of the nasal passage. Masses (infectious, sinus cysts, neoplasia) within the nasal passage or sinus can become large enough in some cases to obstruct the nasal passage.
- Mechanical or functional obstruction of the pharynx can occur. Swelling due to bleeding into the musculature of the pharynx, as occurs with longus capitus rupture, and of the guttural pouches secondary to tympany or empyema, are examples of diseases that result in mechanical obstruction of the pharynx. Pharyngeal collapse due to neurological dysfunction is an example of functional obstruction.
- Alternatively, mechanical and functional obstruction of the larynx can occur. Mechanical diseases that can obstruct the larynx include arytenoid chondritis and subepiglottic cysts. Functional obstruction is most commonly associated with inability to abduct the arytenoid cartilages (both cartilages must be affected to result in respiratory distress at rest).
- Tracheal disease is uncommon in the horse. Diseases such as tracheal collapse and intratracheal masses have been reported and can result in respiratory distress.

Lower respiratory tract

The lower respiratory tract (LRT) includes the bronchi, alveoli, interstitium, and pleural space.

- Recurrent airway obstruction (RAO) and summer pasture associated obstructive pulmonary disease (SPAOPD) are inflammatory/allergic diseases that primarily affect the bronchi and may produce respiratory distress.
- Infectious (pneumonia) and non-infectious (pulmonary oedema secondary to heart disease) conditions may affect the alveoli and interstitium, as well as the bronchi, and can also result in respiratory distress.
- Diseases that are associated with material accumulating in the pleural space limit the ability of the lungs to expand, resulting in respiratory distress. Most frequently, this is associated with relatively rapid accumulation of pleural fluid (haemothorax, neoplastic or infectious pleural effusion) or air (pneumothorax). Soft-tissue masses (e.g. tumours, abscesses) can also occur in the pleural space.

Diseases of the upper and lower respiratory tract can be distinguished on the basis of clinical signs and physical examination. Determining which portion is affected guides emergency treatment and further diagnostics.

Clinical signs and physical examination findings

Upper respiratory tract

- The hallmark clinical sign of URT obstruction is respiratory noise. This occurs as a result of increased URT turbulence during periods of negative intra-airway pressure, and results in airway collapse and exacerbation of the obstruction.
- With the exception of the intrathoracic trachea, negative intra-airway pressure occurs in the URT during inspiration, so inspiratory respiratory noise and inspiratory dyspnoea is a hallmark of URT obstruction (except for diseases affecting the intrathoracic trachea). It is important to remember that depending on the degree of obstruction and the rigidity of the cause of the obstruction, noise can also occur throughout the remainder of the respiratory cycle.
- Airflow from the nares is likely be decreased with URT obstruction. Obstructions caudal to the nasal passage result in a symmetric decrease, whereas rostral obstruction causes asymmetric decrease in airflow if the disease is more severe on one side of the nasal septum.
- Nasal discharge may be present with URT obstruction and is most commonly seen with diseases affecting the nasal passages, sinuses, and guttural pouches. Depending on the disease process, nasal discharge may have been present for a long time prior to the development of respiratory distress. Nasal discharge is typically but not always unilateral with URT disease.
- External swelling may be detected on examination. Swelling in the region of the throat latch can occur with retropharyngeal lymphadenopathy or guttural pouch tympany. Swelling adjacent to the trachea may indicate an extraluminal mass compressing the trachea. Swelling over the sinuses can indicate a mass within the sinus.
- Auscultation along the URT with a stethoscope reveals the point of maximal intensity of the respiratory noise, which indicates the location of the obstruction. Auscultation of the thorax reveals referred upper airway sounds.
- Percussion of the sinuses is dull when the sinuses are filled with fluid or soft tissue rather than air.

Lower respiratory tract

- Horses with LRT disease often present with alterations in their respiratory pattern. Diseases within the pleural space decrease lung capacity resulting in a rapid, shallow breathing pattern.
- Within the thoracic cavity, intra-airway pressures are negative during expiration. As a result, horses with lower airway disease often exhibit expiratory dyspnoea.
- Nasal discharge associated with lower respiratory tract disease, if present, is bilateral. Discharge is typically purulent with infectious disease whereas pulmonary oedema results in foamy, white discharge.
- Coughing is a frequent clinical sign of lower respiratory tract disease.
- Abnormalities are frequently detected on thoracic auscultation in patients with lower respiratory disease. Crackles are heard

when fluid filled airways snap open. They occur when alveoli are filled with fluid. Wheezes occur when the airway is narrowed. Regional increases in bronchovesicular sounds (harsh or tubular lung sounds) can occur in areas of consolidated lung. Regional decreases or absence of lung sounds can occur when portions of the lung are obliterated by abscesses or masses or, more commonly, secondary to accumulation of fluid (ventral) or air (dorsal) in the pleural space.

- Percussion of the thorax can be used to detect air/fluid or air/tissue interfaces, and is most useful for detecting the extent of fluid in the pleural space in horses with pleuropneumonia. With experience, some clinicians can detect changes associated with areas of pulmonary abscesses, masses, or consolidation.

Emergency treatment

Upper respiratory tract (see also Chapter 5)

Few horses with obstruction of the upper portion of the respiratory tract require tracheostomy. It allows airflow to bypass any obstruction of the nares, nasal passages, pharynx, larynx, and trachea proximal to the tracheostomy site. However, horses with lesions of the trachea distal to the temporary tracheostomy site do not respond to this treatment.

- The most common site for temporary tracheostomy is at the level of the junction of the proximal 1/3 and distal 2/3 of the cervical trachea (Figure 26.3a). This is just proximal to the point where the sternocephalicus muscle divides on midline, leaving less musculature overlying the trachea. The location may need to be modified if the horse has had a tracheostomy in the past or if it is likely that the horse will be having a permanent tracheostomy in the near future.
- The area should be clipped and prepared aseptically. A line block with local anaesthetic should be performed on the ventral midline approximately 12–15 cm in length. However, in horses with severe distress or collapse, these steps may need to be abbreviated or even omitted.
- A 1- to 12-cm incision through the skin and cutaneous coli muscle is made on the ventral midline.
- The underlying paired sternothyrohyoideus muscles can be separated with a scalpel or scissors along the pale, fibrous septum that separates them on midline to minimize bleeding. These muscles should be separated for a length of approximately 8 cm to expose several tracheal rings and the connecting annular ligaments.
- The loose connective tissue should then be bluntly dissected from the trachea for better visualization, but this step may be skipped in horses with severe respiratory distress.
- A scalpel blade is then used to make a transverse stab incision through an annular ligament between two tracheal rings, and the incision is extended with a scalpel or scissors. The incision should be between 1/3 and 1/2 of the circumference of the trachea to avoid injury to the vessels and nerves adjacent to the caudal half of the trachea and to avoid stenosis of the trachea upon healing.
- A variety of temporary tracheostomy tubes are available (Figure 26.3b). Tubes can be made of plastic or metal and may be self-retaining or may need to be sutured to the surrounding skin or secured with elastic adhesive tape placed around the neck. If an inflatable cuff is present on the tube, it SHOULD NOT be inflated because pressure necrosis of the tracheal mucosa is possible. The largest tracheostomy tube that fits should be used. If no tube is available in an emergency, a shortened plastic syringe or syringe case (as large as possible) can be substituted temporarily.
- If tracheal collapse distal to the site of the tracheostomy is the cause of respiratory distress, a long endotracheal tube can be placed to prevent collapse.
- Sutures encircling the tracheal rings adjacent to the tracheostomy site can be placed to make placement and replacement of the tube easier, but are not generally required.
- Because temporary tracheostomy is often an emergency procedure, it is helpful to have a kit readily available with all of the necessary instruments, including local anaesthetic, gloves, a scalpel handle and blade, scissors, hemostats, sterile gauze pads, and several sizes of tracheostomy tubes.
- Severe upper respiratory obstruction can result in pulmonary oedema due to alterations in intrathoracic pressure, capillary permeability, and pulmonary vasculature tone. If present, foam may be evident in the trachea, crackles may be heard on thoracic auscultation, and the respiratory distress may not respond as expected to the tracheostomy. Treatment for pulmonary oedema secondary to upper respiratory obstruction includes intravenously administered furosemide (1–2 mg/kg) (use with caution in horses that are dehydrated) and intranasal oxygen insufflation if available.

Lower respiratory tract (see also Chapter 6)

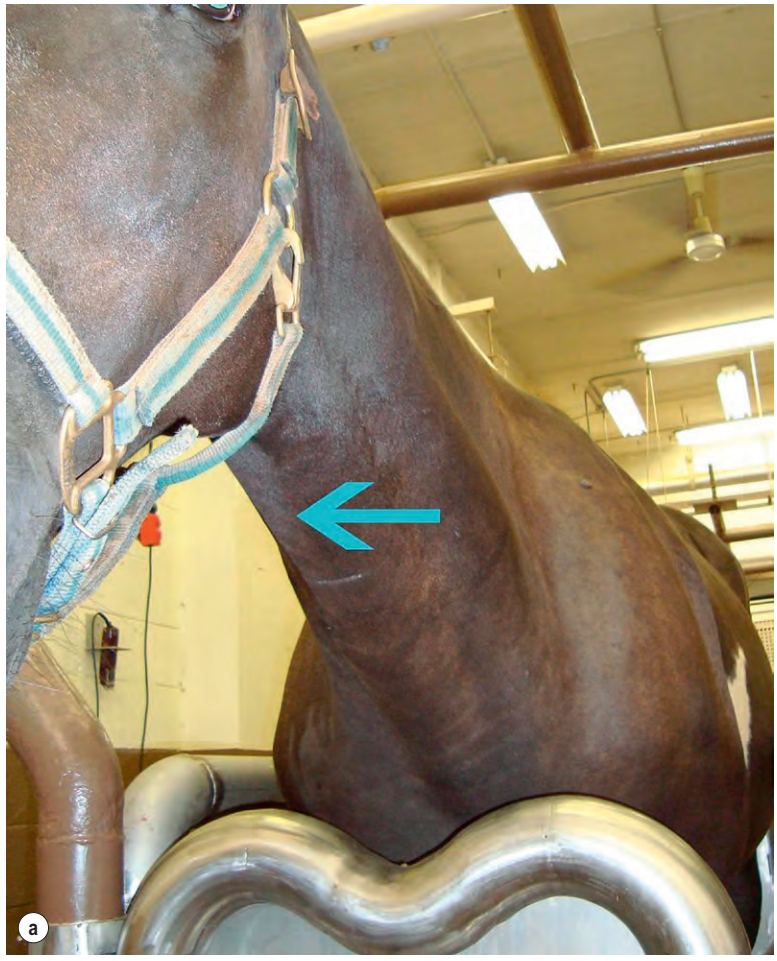
When respiratory distress results from LRT disease, intranasal oxygen insufflation may improve oxygenation and clinical signs (Figure 26.4).

The remainder of emergency treatment for LRT disease varies with the region affected and the cause of the distress.

Inflammatory bronchial disease

Respiratory distress associated with inflammatory bronchial disease is treated with bronchodilators, corticosteroids, and environmental management.

- Bronchodilators can be administered orally or by inhalation from a metered dose inhaler with the aid of commercially available masks.
  - Inhaled bronchodilators act quickly and are therefore likely to be more useful in emergency situations. However, they need to be administered frequently due to a short duration of action.
  - Commonly administered bronchodilators for horses with acute respiratory distress include β-2 agonists clenbuterol (0.8–3.2 µg/kg PO q12 hour) and albuterol (0.8–2 µg/kg inhaled q 1 hour) or the anticolinergic ipratropium bromide (0.4–1 µg/kg inhaled q 6 hour).
  - It is important to remember that bronchodilators provide symptomatic relief and do not treat the underlying cause.
- Corticosteroids are used in inflammatory LRT disease for their potent anti-inflammatory effects.
  - During an acute episode, rapid onset of action is important so parenteral (intravenous or intramuscular) or inhalant corticosteroids are preferred over oral corticosteroids.
  - Inhalant corticosteroids avoid many of the complications reported with systemic corticosteroids (such as laminitis or increased risk of bacterial pneumonia).
  - Commonly administered corticosteroids include dexamethasone (0.04–0.1 mg/kg IV q 24 hour), fluticasone proprionate (2000 µg/kg inhaled q 12 hour), or prednisolone (0.5–1.5 mg/kg PO q 24 hour).
- Environmental control is vital for long-term management of inflammatory bronchial disease and may benefit horses during an acute episode of distress.
  - For horses with RAO, minimizing dust in bedding and feed and maximizing ventilation are recommended. Pasture is the



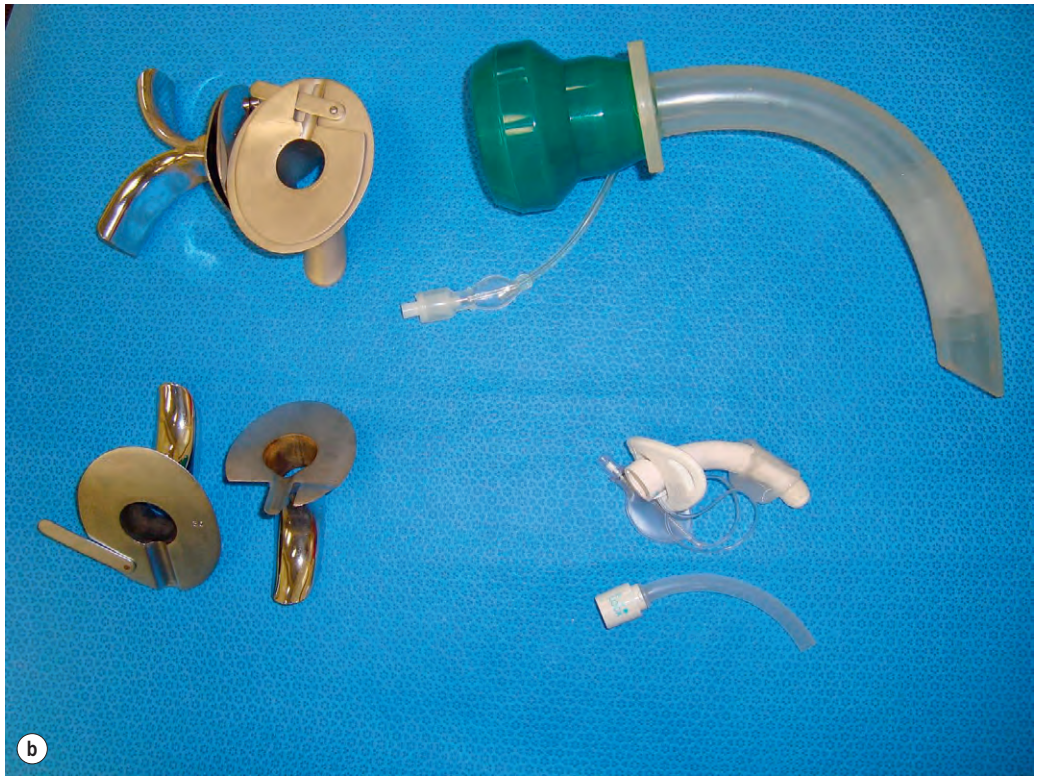


Figure 26.3 (a) Appropriate site for tracheostomy placement in an adult horse. (b) Examples of types of stainless steel and plastic temporary tracheostomy tubes used in adult horses and foals.

Figure 26.4 An intranasal cannula secured in place with elastic tape for oxygen insufflation in an adult horse. Alternatively, the cannula may be sutured in place. A similar set-up may be utilized in foals.

ideal environment, but may not be practical during an acute episode of distress.
- For horses with SPAOPD, moving the horses to a stall with minimal dust in bedding and feed is recommended.

Alveolar and interstitial disease

No standard treatment protocol consistently relieves respiratory distress associated with alveolar and interstitial disease.

- As mentioned above, oxygen therapy may improve oxygenation and decrease clinical signs.
- Treatment specific to the underlying aetiology (e.g. furosemide for pulmonary oedema, anti-inflammatories for inflammatory disease, antimicrobials for bacterial disease) should be initiated. However, these treatments may or may not improve the horse's level of distress depending on the extent of lower airway disease.
- Mechanical ventilation can be used to improve ventilation and oxygenation in some foals, but application in adult horses is extremely limited.

Diseases of the pleural space

Respiratory distress associated with diseases of the pleural space frequently improve after evacuating the material in the pleural space.

- This is more easily achieved in cases of fluid or air accumulation compared with space-occupying soft tissue masses. Due to the effect of gravity, fluid accumulates ventrally, and air accumulates dorsally.
- The site for fluid removal is the cranioventral thorax, avoiding the region of the heart, and is most often selected with ultrasound guidance.
- The site for air removal is generally chosen empirically, dorsally in the 13th intercostal space.
- Removal of fluid or air can be achieved with intermittent thoracocentesis using a teat cannula or with continuous or intermittent drainage using an indwelling chest drain (8- to 10-French catheter).
- Removal can be active (applying suction to the end of the tubing) or passive.
- Placement of a teat cannula or indwelling drain is performed in the same manner.
  - Placement at the cranial aspect of a rib avoids the intercostal vessels that run along the caudal edge of each rib.
  - The area selected is clipped and prepared aseptically and local anaesthetic solution is infused in the subcutaneous tissues and intercostal muscles.
  - An incision just long enough to accommodate the teat cannula or drain is made in the skin, subcutaneous tissue and external fascia of the intercostal muscles.
  - The teat cannula or drain is then inserted and advanced into the pleural space bluntly.
  - Care should be taken to assure slow and controlled advancement of the cannula or drain into the pleural cavity to avoid laceration of the underlying lung.
  - In addition, precautions should be taken to avoid air entering the pleural space through the cannula/drain during inspiration (due to negative pressure within the pleural space). When using a teat cannula, extension tubing should be connected to a closed three-way stop-cock, and the skin should be closed when the teat cannula is removed. When using an indwelling chest drain, haemostats, a one-way valve (Heimlich), or active suction can be applied to prevent air entering the pleural space. The tubing should be secured with purse-string and Chinese finger trap sutures, which prevent leakage around the tube.
- Distress associated with bilateral accumulation of fluid or air may or may not respond to unilateral drainage depending on the amount of fluid or air that has accumulated and whether the mediastinum is incomplete or complete.
- Complications can be associated with evacuation of the pleural cavity, particularly if rapid drainage is performed.
  - 'Re-expansion pulmonary oedema' is reported secondary to rapid correction of pneumothorax in people. Thus, evacuation of the air with suction applied at no more than 20 cm H_2O is recommended.
  - Hypovolaemia and cardiovascular compromise associated with rapid removal of fluid from the pleural space (a 'third space') can occur when large quantities of pleural fluid are removed. Intravenous fluid support and controlled drainage should minimize this.

Ancillary diagnostics

Following emergency treatment to resolve respiratory distress and initiation of supportive care, further diagnostics are necessary to

identify the underlying cause and guide treatment. These are discussed in detail in Chapters 5 and 6.

Upper respiratory tract

- Upper airway endoscopy is the single most useful diagnostic test to identify the cause of URT obstruction. The location and nature of the obstruction can often be evaluated with direct visualization.
- Depending on the nature of the obstruction found, additional diagnostics that may be indicated include radiographs, ultrasound, histopathology, haematology, and microbiology.

Lower respiratory tract

- Diagnostic imaging of the LRT frequently involves the combination of radiographs and ultrasound. Ultrasound can be performed rapidly with portable ultrasound machines whereas radiographs of an adult horse's chest requires large X-ray units generally available only in referral hospital settings. Ultrasound provides information about the pleural space and the periphery of the lung, whereas radiographs provide information about the lung parenchyma.
- Other diagnostics that may be indicated include haematology, cytological evaluation and/or bacterial culture on samples obtained from the airways via transtracheal wash and/or bronchoalveolar lavage or from the pleural space via thoracocentesis, or histopathological analysis of lung biopsy samples.

26.7 CARDIOPULMONARY CEREBRAL RESUSCITATION (CPCR)

Cardiopulmonary arrest

- Cardiopulmonary arrest is the cessation of spontaneous ventilation and perfusion.
- Unlike in humans, cardiopulmonary arrest in horses is most often secondary to systemic disease rather than a result of primary cardiac disease. Any systemic disease that depresses the circulatory and/or respiratory system can result in cardiopulmonary arrest.
- Insufficient oxygen delivery to tissues as a result of respiratory and/or circulatory failure causes severe hypoxic acidosis. Hypoxic acidosis induces respiratory arrest and non-perfusing bradycardia that progresses to cardiac arrest. Successful resuscitation is more likely if treatment of respiratory arrest is initiated prior to cardiac arrest.
- Cardiac arrest can be associated with one of four non-perfusing or pulseless cardiac rhythms: pulseless electrical activity, asystole, pulseless ventricular tachycardia, or ventricular fibrillation.
  - During pulseless electrical activity, electrical activity of the heart continues (an electrocardiogram reveals a rhythm), but the myocardium does not respond/contract, and no pulse is palpable.
  - During asystole, there is no electrical activity of the heart and no myocardial contraction.
  - Pulseless ventricular tachycardia and ventricular fibrillation are most frequently associated with primary cardiac disease and are therefore uncommon in horses.

Cardiopulmonary cerebral resuscitation (CPCR) and its use in horses

The goal of CPCR is to provide ventilatory and circulatory support and to aid in re-establishment of spontaneous cardiorespiratory function.

- Information on appropriate technique for CPCR in horses is remarkably limited; thus, recommendations for CPCR in horses are primarily based on guidelines for humans that have been established from human and animal studies.
- It is uncommon to perform CPCR in horses. It is most frequently attempted in neonatal foals (Figure 26.5). Application to adult horses is limited and efforts are unfortunately rarely successful.

Components of CPCR

- Effective CPCR requires support of the cardiovascular and respiratory systems, with cardiac compressions, pharmacologic support, defibrillation (if indicated), and ventilation.
- Current recommendations in human CPCR stress the importance of cardiovascular support over respiratory support.
- Early initiation and minimal interruption of cardiac compressions is believed to be central to success of CPCR during cases of cardiac arrest secondary to circulatory failure.
- It should be noted, however, that in cases where respiratory failure precedes cardiac arrest (as is common in foals), early ventilatory support may be more important and beneficial.
- Ventilation.
  - Ventilation is best achieved via a nasotracheal, orotracheal, or transtracheal (tracheotomy) tube. The largest diameter cuffed endotracheal tube that is easily passed (diameter: adult – 18–22 mm nasotracheal, 26–30 mm orotracheal; foal – 7–12 mm nasotracheal, 10–14 mm orotracheal) should be placed as soon as available with minimal interruption of cardiac compressions. Once tracheal intubation is confirmed the cuff should be inflated to the point where no air leaks are detected during positive pressure ventilation.
  - Recommended ventilation rate is 8–10 breaths/minute in foals and 4–6 breaths/minute in adults.
  - Ventilation should be performed with 100% oxygen if available. Breaths can be delivered via a self-inflating resuscitation bag in foals or a demand valve in adults. When using a demand valve, the valve should be taken away from the end of the tracheal tube during expiration to avoid high resistance. A positive-pressure ventilator is also appropriate and is often available if the arrest occurs under anaesthesia.
  - The volume of air administered should be enough to cause a visual rise in the chest. However, inspiratory pressure should not exceed 20 cm H_2O in foals or 30 cm H_2O in adults. Inspiration should last no more than 1 second in foals and 2–3 seconds in adults.
- Cardiac compressions.
  - It is estimated that optimal closed-chest compressions can generate 25–33% of normal cardiac output and a peak systolic arterial pressure of 60–80 mmHg. Compressions should be initiated as soon as one of the above non-perfusing rhythms is detected, regardless of whether any cardiac contractions are present. Early initiation and minimal interruption (maximum of 10 seconds interruption with at least 2 minutes of continuous compressions between) is vital.
  - Recommended compression rate is 80–120 compressions/minute in foals and 60–80 compressions/minute in adult horses.

Figure 26.5 Hand ventilation of a neonatal foal with respiratory arrest following dystocia. An orotracheal tube has been placed, and ventilation is being provided 100% oxygen via an ambu bag. The foal is being supported in sternal recumbency to maximize ventilation of both lungs.

- Each compression cycle should have a compression and chest recoil/relaxation phase. The compression phase is responsible for forward blood flow. During the recoil phase the negative intrathoracic pressure stimulates venous return. Each compression should result in 30% decrease in chest height. The chest wall should be allowed to have full recoil. Each cycle should be at least half recoil/relaxation.
- For cardiac compressions, the patient should be placed on a firm, dry surface in lateral recumbency. Compression should be focused just caudal to the elbow or triceps. Compressions should be delivered by placing one hand on top of the other, with the person administering compressions above the patient's chest with elbows locked to allow the upper body to power the compression and minimize fatigue. Some clinicians have suggested using one or both knees to compress the chest in larger patients. If more than one person is available for CPCR then they should rotate regularly (every 2–5 minutes) to minimize fatigue.
- If external chest compressions as described are unsuccessful, there are reports of internal or direct cardiac compression following thoracotomy in horses. If arrest occurs during abdominal surgery, an incision in the diaphragm can allow direct cardiac compression. However, complications post-resuscitation with this method are likely.

- Drug therapy.
  - Ideally, resuscitation drugs should be administered through a central vein. If it is not possible to obtain vascular access, many drugs can be administered intratracheally at higher doses. In foals, intraosseous administration, using intravenous doses, may be attempted.

Specific drugs used in CPCR

1. Epinephrine is a mixed adrenergic agonist that is used primarily for its α-adrenergic (vasoconstrictive) effects during CPCR to help maintain coronary perfusion.
   - It can be administered at a dose of 0.01–0.02 mg/kg IV or 0.05–0.1 mg/kg intratracheally every 3–5 minutes.
   - If unsuccessful, a higher dose can be administered (0.1 mg/kg IV). However, high-dose epinephrine can be associated with complications including myocardial necrosis, post-resuscitation hypertension and tachyarrhythmias, and refibrillation.
2. Vasopressin is a potent non-adrenergic vasoconstrictor.
   - It can be administered at a dose of 0.2–0.8 U/kg IV (0.4–1.2 U/kg intratracheally).
   - The recommended dosing interval is variable, with some sources indicating that a single dose is adequate due to the long duration of action (10–20 minutes) with other protocols recommending administration every 3–5 minutes with or without alternation with epinephrine.
   - In some studies, vasopressin has had superior results for resuscitation of patients with asystole (humans) and ventricular fibrillation (animal models).
   - Potential benefits of vasopressin include maintenance of coronary and cerebral perfusion, ability to cause

vasoconstriction in an acidic environment, and decreased likelihood of post-resuscitation arrhythmia.
- Potential disadvantages of vasopressin include dramatic splanchnic vasoconstriction with potential for ischaemia post-resuscitation (especially in the face of hypovolaemia), increased liver enzymes, hyperbilirubinaemia, increased urine production, decreased cardiac output due to increased afterload, decreased heart rate, decreased platelet count, and skin necrosis.

3. Atropine is a parasympatholytic drug that may be useful in treating vagally mediated asystole or non-perfusing bradycardia, which may occur with GI disease and distension.
   - It can be administered at a dose of 0.02 mg/kg IV twice (5-minute interval).
   - It is important to remember that, in foals, hypoxia rather than high vagal tone is generally responsible for bradycardia and asystole. Treatment with atropine in this situation is contraindicated due to resultant increased oxygen demand and hypoxic insult to the myocardium. In this situation, ventilation with 100% oxygen combined with epinephrine/vasopressin and external cardiac compression is appropriate.
4. Anti-arrhythmic agents are used to treat pulseless ventricular tachycardia or ventricular fibrillation.
   - Specific drugs include amiodarone (5 mg/kg IV or IO over 10 minutes with one repeat dose of 2.5 mg/kg after 3–5 minutes), lidocaine (1 mg/kg IV or IO with additional doses of 0.5–0.75 mg/kg at 5–10 minute intervals up to a total dose of 3 mg/kg), and $MgSO_4$ (25–50 mg/kg diluted in 1L 5% dextrose in water IV over 5–20 minutes).
   - Amiodarone may be superior to lidocaine if defibrillation is planned because lidocaine increases defibrillation threshold.
   - $MgSO_4$ is probably most effective in cases of torsades de pointes.
5. Judicious fluid administration is part of many resuscitation protocols.
   - In euvolaemic patients rapid administration of a low volume bolus (10–20 mL/kg) is recommended.
   - In patients that are hypovolaemic due to dehydration and/or vasodilation, additional volume expansion with isotonic or hypertonic crystalloids or colloids (as described in the section on the 'Pathophysiology and management of shock') may be required.
   - Excessive volume administration should be avoided because increased volume may result in decreased coronary perfusion pressure due to increased right atrial pressure relative to aortic pressure, and decreased cerebral perfusion pressure due to a disproportionate increase in intracranial pressure relative to mean arterial pressure.

Defibrillation

Defibrillation is indicated for pulseless ventricular tachycardia and ventricular fibrillation, but given the specialized equipment necessary is not routinely performed in horses. In addition, due to high impedance, defibrillation is not possible in large horses (>350 kg). Thus, detailed discussion of defibrillation technique is beyond the scope of this text.

Patient monitoring during CPCR

- Palpating a carotid or femoral pulse is frequently used to assess effectiveness of chest compressions. However, this method may not be reliable because venous pulses in adjacent veins can be present without adequate arterial blood flow.
- Identification of blood flow with a Doppler ultrasound transducer on the cornea may be used to assess cerebral blood flow.
- Pupil size and response to light can also be used to assess cerebral blood flow. A fixed and dilated pupil is consistent with inadequate cerebral blood flow, whereas with adequate cerebral blood flow the pupil size is more normal, and a pupillary light reflex is present.
- Capnometry (measurement of the partial pressure of CO_2 in exhaled gases – the end tidal CO_2 or $ETCO_2$) allows assessment of the effectiveness of compressions at producing an appropriate cardiac output. Because CO_2 is produced by all cells in the body, when circulation ceases, no CO_2 is transported from the tissues to lungs for expiration. Thus, no $ETCO_2$ is detected. If chest compressions are effective in restoring circulation, $ETCO_2$ increase. The goal for $ETCO_2$ during CPCR is 12–18 mmHg. An $ETCO_2$ of 18–24 mmHg is consistent with return of spontaneous circulation.
- Evaluation of an electrocardiogram is very important to correctly classify the rhythm in order to initiate the appropriate therapy.
  - Early defibrillation improves the chances of reversing ventricular fibrillation, whereas defibrillation can decrease survival in cases of asystole.
  - Assessment of the ECG rhythm should occur early during resuscitation and be repeated after any treatment that may change the rhythm, but compressions should only be interrupted briefly, with at least 2 minutes of compressions between interruptions.

FURTHER READING

Carr EA, Holcombe SJ (2009) Nutrition of critically ill horses. Veterinary Clinics of North America, Equine Practice 25(1):93–108

Holcombe SJ (2006) Shock: pathophysiology, diagnosis, and treatment. In: Auer JA, Stick JA (eds) Equine surgery. Saunders/Elsevier, St Louis

McGorum BC, Dixon PM, Robinson NE, Schumacher J (eds) (2007) Equine respiratory medicine and surgery. Saunders/Elsevier, St Louis

McKenzie HC, Goer RJ (2009) Feeding management of sick neonatal foals. Veterinary Clinics of North America, Equine Practice 25(1):108–119

Muir WW, Hubbel JAE (eds) (2009) Equine anesthesia: monitoring and emergency therapy. Saunders/Elsevier, St Louis

Orsini JA, Divers TJ (eds) (2008) Equine emergencies: treatment and procedures. Saunders/Elsevier, St Louis

Palmer JE (2007) Neonatal foal resuscitation. Veterinary Clinics of North America, Equine Practice 23:159–182

Index

NB: Page numbers in *italics* refer to boxes, figures and tables.

A

AAEP (American Association of Equine Practitioners) scale, lameness, 311
Abaxial sesamoid nerve block (ASNB), 313, *313*
Abdomen
  auscultation, 34
  distension, 34
  examination, *33*, 34
  muscles, rupture, 295
  obstructive/strangulating lesions, 444
  pain, acute *see* Colic
  palpation, 34
  penetrating wounds, 470
  radiography, 502
Abdominal cavity, 67–75
  abscesses, 73–74, 443–444
  drainage/lavage, 73
  hernias *see* Hernias
  neoplasia *see* Abdominal neoplasia
  peritonitis *see* Peritonitis
Abdominal neoplasia, 74–75
  gastrointestinal, 74–75, *74*
  urogenital tract, 75
Abdominal testis, 171, *172*
Abdominocentesis, 198, 440, 446
Abducens nerve (VI), 212, *213*
Abortion, 307–308
  equine herpesvirus 1 (EHV1), 402–403
  infectious causes, 294
  non-infectious causes, 294–295
Abrasion, tooth, 10–12
Abscesses
  abdominal, 73–74, 443–444
  brain, 220–221
  cervical injection, 105
  corneal stromal, 251, *251*
  Corynebacterium pseudotuberculosis, 278, 455, *456*
  dental, 14
  foot, 334–335, *335*
  globe/orbit, 243
  liver, 445
  lung, *118*
  nodular skin, 278–279
  strangles, 5, 412, *412*
  uterine, 290
Absolute polycythaemia, *199–200*
Accessory carpal bone fractures, 371, 535
Accessory ligament, deep digital flexor tendon, 367
Accessory nerve (XI), 213, *213*
Acepromazine, 26, 490, 492
Acer spp, 466
Acer rubrum, toxicity, 466
Acetazolamide, 255, 453
Acetic acid (vinegar), 54
Acetylcysteine, 121, *240*
Acetylpromazine, 333
Acid-base balance, 464–465
  disturbances, 564–565
Acidosis
  metabolic, 464
  respiratory, 464–465
Acinetobacter spp, 248–249
Acquired conditions
  cardiac defects, 429
  cataracts, 256
  conjunctiva, 246–247
  cornea, 248–252
  eyelids, 244–246
  globe/orbit abnormalities, 243–244
  heart murmurs, 429
  occlusion, 247
  palatal defects, 5, 103
  stenosis, 247
  tracheal obstructions, 103
  vitreal anomalies, 257
Acquired valvular heart disease, 142–148
  congenital malformations, 142–143
  myocardial disease, 147–148
  pericardial disease, 147
Acremonium coenophalum, 178
Actinobacillus spp, 146, 278
Actinobacillus equuli, 441
Actinobacillus mallei (glanders), 220
Actinomyces spp, 276, 278
Actinomyces pyogenes, 279
Activated partial thromboplastin time (APTT), 198
Acute conditions
  abdominal pain *see* Colic
  diarrhoea, 58, 65
  enterocolitis, 63–64
  grass sickness, 55
  haemorrhage, *199–200*
  hepatic necrosis, 52
  interstitial pneumonia, 128
  iridocyclitis, 254
  obstructive pulmonary disease, 121–122
  oesophageal obstruction, *3*
  renal failure (ARF), 162
  renal tubular necrosis (RTN), 162
  sacroiliac injury, 394
  sudden onset coughing, 131
  toxic metritis, 290
Acute respiratory distress, 578–582
  causes, 578
  clinical signs, 578–579
  emergency treatment, 579–581
  investigations, 581–582
Adenovirus, equine, 404, 432, 442
Adhesions
  cervical, 291
  colic, 47
  intrauterine, 290
Adrenal insufficiency (hypoadrenocorticism), 185, 447–448
β-adrenergic drugs, 343
Adrenocorticotropin hormone (ACTH) (corticotropin), 182–184
  stimulation test, 184
Advanced assisted reproduction, 306–307
Aflatoxin, 468
African horse sickness (AHS), 405–406
  syndromes, 405–406
Agammaglobulinaemia, 207, 450
Agenesis, 244
Airway obstruction, 92–94
  causes, 103–104
  collapse, causes, 102–103
  differential diagnosis, 105
  endoscopy, 93–94, *94*
  history, 93
  inflammatory airway disease (IAD), 122–123
  palpation tests, 93
  radiography, 93
  upper respiratory tract (URT), 95
  see also Recurrent airway obstruction (RAO)

Alar cartilage necrosis, 84
Alar folds, hypertrophy, 83
Albinism, 257, 281–282
Albuterol, *120*
Albuterol sulfate, 121
Alfalfa hay, 193
Alimentary system disease, 439–444
  alimentary (true) colic, 22
  congenital conditions, 441
  endoscopy, 440
  examination, 439–441
  lymphoma, 61–62, *62*
  radiography, 440
  ultrasonography, 440
  see also Upper alimentary system
Alkalosis
  metabolic, 465
  respiratory, 465
Allergy, food, 268
Allografts, 487
  defined, 481
Alloimmune haemolytic anaemia, 449–450
Alopecia, 262, 268–274
Alopecia areata, 271–272, *272–273*
Alpha$_2$-agonists
  anaesthesia, 491–492, 494
  clinical use, 490
  epidural, *495*, 496
  sedation, 490
α-agonist, 303
Alphavirus spp, 219–220
Alternaria alternata, 276
Alternaria dermatitis, 276
Alternaria spp, 248–249, 276
Alveolar disease, 581
Alveolar pattern, lung, 550
Amikacin, *242*
Aminoglycosides, 447
Aminophylline, 121
Amphotericin-B, 128, *240*
Ampicillin, 442
Ampullae, 302
Amsinckia spp (riddleneck), 51–52
Amyloidosis, 280–281
  nasal, 281
  systemic, 281
Anaemia
  blood transfusions, 571
  chronic disease, *199–200*, 203
  equine infectious (EIA), *199–200*, 294, 405
  haemorrhage, 200–201, *201*
  heinz body haemolytic, 202
  immune-mediated, *199–200*
  immune-related haemolytic, 201–202
  iron deficiency, 203
  isoimmune/alloimmune haemolytic, 449–450
  microangiopathic, 202
  non-regenerative, 210
  rickettsial disease, 203
Anaesthesia, 37–38
  epidural, 494–496, *495*
  foals, 493
  general
    complications, 496–497
    induction, 491–492, 496–497
  inhalation, 493
    advantages/disadvantages, 493
    recovery, 493
  injectable, 492–493
  local, *495*, 496
  maintenance, 493
  patient preparation, 489–490
  postoperative complications, 45
  prolonging, 492
  risks, 489
  total intravenous, 492
  see also Sedation
Analgesia, 494, 571
  epidural, 494–496, *495*
  intrasynovial, 314–315
  postoperative, 44
Anaphylaxis, 129
Anaplasma phagocytophilum, 203, 415
Anastomosis, 40, *40*
  intestinal segments, 40, *41*
Anencephaly, 216
Angiotensin-converting enzyme (ACE) inhibitors, 149
Angular limb deformities, 372–374, *373*, 439, 537, *537*, 543
Anhidrosis (dry coat syndrome), 181–182
Aniridia, 252
Anisocytosis, 197
Ankyloblephron, 244
Annular ligament syndrome, 356, *356*
Anoestrus, 307
  lactational, 304
Anophthalmos, 243
Anoplocephala magna, 420
Anoplocephala perfoliata, 420
Anovulatory follicles, 288
Antebrachiocarpal joint, 315
Anterior chamber, examination, 237
Anterior (cranial) epidural technique, 496
Anterior enteritis, 30–32, 410–411
Anthelmintics, 266, 417–419, 467
Anthrax, 279, 415
Anti-acids, 443
Anti-arrhythmic agents, 584
Antibacterial sprays, 269
Antibiotic-associated diarrhoea, 442
Antibiotics
  bacterial pseudomycetoma, 276
  bacterial pyoderma, 270
  cervical laceration, 291
  diarrhoea and, 442–443
  endocarditis, 146
  jugular thrombophlebitis, 157
  ocular disease, *240*
  pericardial disease, 147
  postoperative, 45
  septic arthritis, 323
  sole penetrating wounds, 337
  sporadic lymphangitis, 209
  toxicity, 457, 468
  Tyzzer's disease, 445
Anticoagulants, 147, 467
Anti-collagenase therapy, 250
Anticollagenolytics, *240*
Antidiuretic hormone (ADH) (vasopressin), 182, 583–584
Anti-endotoxin therapy, 443
Antifungals, *240*, 250, *252*, 276–277
Antihistamines, 267, 280
Anti-inflammatories
  iridocyclitis, 255
  ocular disease, *240*, *252*
  postoperative, 44
  see also Non-steroidal anti-inflammatory drugs (NSAIDs)
Antimicrobials
  abdominal abscesses, 444
  corneal stromal abscess, 251
  iridocyclitis, 255
  keratomalacia, 250
  neonatal foal, 576–577
  ocular disease, *252*
  ulcerative keratitis, 250
  wound healing, 481
Antioxidants, 121, 435
Anti-proteinase therapy, 250
Antiseptics, 476
Antithrombin III (ATIII) activity, 198
ANTU (alpha-naphthylthiourea), 467
Anus, examination, 214
Aortic insufficiency (AI), 144–145
Aorto-iliac thrombosis, 157–158
Aplasia cutis, 281
Arboviruses, 220
Arrhythmias *see* Cardiac arrhythmias
Arsenic, 467
Arterial blood gas analysis, 429
Arterial blood pressure, 142
Arteritis
  cranial mesenteric, *36*
  viral *see* Equine viral arteritis (EVA)
Arthritis, 346
  septic *see* Septic arthritis
  traumatic, 319–320, 346, 370
Arthrocentesis, 315–316
Arthrodesis
  chemical, 379–380
  surgical, 379
Arthroscopy, lameness, 318–319, *319*
Artificial insemination (AI), 305–306
  low sperm count, 307
Arytenoid chondropathy, 100–101, *101*
Arytenoidectomy, 97
Ascarids, 420
Asclepias spp, 466
Aspartate aminotransferase, 452
Aspergillus spp, 128, 248–249, 416, 468
Aspergillus fumigatus, 117–118
Aspirin, 397
Asteroid hyalosis, 257
Astragalus spp (locoweed), 217, 465
Ataxia, 214, 222–225, *223*, *224*
  foals, 437
  neurological signs, 234
  Sorghum grass, 228
ATD (auditory tube diverticulum) *see* Gutteral pouches
Atelectasis, 116
'Atheroma', false nostril, 83
Atlanta-occipital (AO) space, 215
Atopic dermatitis, 267, *267*
Atrial sound, 135
Atrial conditions
  fibrillation (AF), 151–153, *152*

premature complexes (APC), 151, *151*
septal defect (ASD), 428
tachyarrhythmias, *136*
tachycardia, 151, *151*
Atrioventricular (AV) block, *136*, 150, *150*
Atropine, 26, 121, 230, *240*, *242*, 255, 584
Attrition, tooth, 10–12
Atypical myoglobulinuria (myopathy), 457–458
Auditory tube diverticulum (ATD) *see* Gutteral pouches
Autoagglutination, 198
Autograft, defined, 481
Autonomic nervous system, 212, 233
Autumn transition stage, 285
Avulsion fractures, 511
Axial deviation of ary-epiglottal folds (ADAF), 102
Azaleas, 466
Azathioprine, 204
Azithromycin, 126–127, 414
Azotaemia, 189

B

Babesia caballi, *198*, 421–422
Babesiosis, equine (piroplasmosis), *199–200*, 202–203, 421
neonatal, 208
Bacillus anthracis, 279, 415
Bacillus Calmette–Guérin (BCG), 278
Bacillus piliformis see Clostridium piliforme
Bacitracin, *240*
Back
anatomy, 389, *390*
diagnostic approach, 389–392
disorders, 392
examination, 390–391
fractures, 551
history, 389–390
investigations, 391–392, *391*
treatment, 395, *395*
see also Spinal cord
Back, radiography, 551–552
anatomy, 551
projections, 551
radiopathology, 551–552
technique, *506*, 551
Bacterial infections, 131, 415–416
abortion, 294
cystitis, 165–166
diagnosis, 400–401
foals, 432–433
meningoencephalitis, 435
orchitis, 302
pseudomycetoma, 275–276
pyoderma, 270, *271*, 279
Bacteroides spp, 278
Band neutrophils, 197
Basal ganglia, 234
Basidiobolus spp, 276
Basidiobolus haptosporus, 276
Basophilia, 197
'Bastard strangles', 278, 411
Beclamethasone diproprionate, 120, *120*
Behaviour, abnormal reproductive, 303
Benzimidazoles, 418
Benzodiazepines, 435, 490–492
Bermuda grass staggers, 228, 466
$Beta_2$ agonists, 121
$Beta_2$ receptors, 121
Biceps tendon, ossification, 378
Bicipital bursitis, 378
Bilateral purulent nasal discharge, 131
Biliary atresia, 445
Biliary calculi, 52–53, *53*
Biliary obstruction, 445
Biological dressings, 481
Biomechanical theory, navicular disease, 342
Bipolaris speciferum, 276
Birdsville horse disease, 218
Black walnut (*Juglans nigra*), 466
Bladder
neoplasia, 75
ruptured, 447
Blastomyces spp, 277
Blastomycosis, 277
Bleeding tendency, 50
Blindness
bilateral, 260
unilateral, 260
Blister beetle toxicosis, 166, 463
Blood
gas analysis, 464
loss *see* Haemorrhage
products, 568–572
typing, 199
Blood transfusions, 201, 569–571
anaemia, 571
indications, 569
Blunt trauma, 243
'Bog spavin', 380
Bone disorders, 327–328
bone spavin, 379–380
contusion, distal/middle phalanx, 344
cysts, 526
fragments, *505*, *507*, 510
X-rays, 501
see also Fractures; Joint diseases
Bone healing, 324
complications, 324
primary, 324
secondary, 324
Bone lucency, 510
Bone marrow
evaluation, 198
suppression/failure, *199–200*, 204
Bordetella bronchiseptica, 125
Borna disease, 220
Borrelia burgdorferi, 415
Bots, 420–421
Botulism (shaker foal syndrome), 229, 438
Bowel disease, chronic inflammatory, 59–61, *60–61*
Brachial plexus injuries, 437–439
Bracken fern, 466
Brain, 216–222
abscess, 220–221
disease, 327
foals, 216–217
infectious conditions, 219–221
lesions, neurological signs, 234
neoplasia, 221
parasitic lesions, 221
toxic conditions, 217–219
trauma, 217
Brainstem
dysfunction, 234
lesions, 234
Breeding
management, 305
season, 304
soundness examination (BSE), 285–287, 298–300
Bronchial disease, inflammatory, 579–581
Bronchial pattern, lung, 549–550
Bronchoalveolar lavage (BAL), 115, *115*, 417
blind technique, 115–116
endoscopy, 115
Bronchodilators, *120*, 121, 579
Brucella abortus, 415
Brucellosis, 415
'Bucked shins', 360
Buckwheat, photosensitization, 467
Bullae, 262
'Bullet-hole' lesions, 259
Buphthalmos, 243
Burkholderia mallei, 415
Burn carcinoma, 473
Butorphanol, 26, 494, 571
'Butterfly' lesions, 259

C

Caecum
impaction colic, *28*
resection, 42
Caesarean section, 297
'Cake-frosting', 249, *249*
Calcinosis, 543
Calcinosis circumscripta (tumoral calcinosis), 328, 387, 545
Calcitonin, 462
Calcium disodium ethylene diamine tetra-acetic acid (calcium EDTA), 218
Calcium gluconate, 453
Calcium homeostasis, 462
Calcium metabolism, 462
Calcium sulphate (plaster of Paris), 13
Caley pea, 466
Callus, 515
formation, 501, *502*
Candida spp, 128
Canine teeth, extractions, 18
Canker, 336
Cantharidin toxicosis, 463
Capillary refill time, 34
Capped elbow, 376
Capped hock, 383
Capsulitis, 537
Carbamazapine, 222
Carbohydrate malabsorption, 58
Carbon tetrachloride, 467
Cardiac arrhythmias, *136*, 149–156
pathological, 151–156
physiological, 150–151
Cardiac catheterization, 142
Cardiac compressions, 582–583
Cardiac glycosides, 466
Cardiac murmurs *see* Heart murmurs
Cardiac output, restoring, 567–568

Cardiogenic shock, 426
Cardiopulmonary arrest, 582
Cardiopulmonary cerebral resuscitation (CPCR), 582–584, *583*
  components, 582–583
  defibrillation, 584
  drugs, 583–584
  patient monitoring, 584
Cardiovascular system, 133–158, 550
  arterial blood pressure, 142
  auscultation, 134–138, *135*, *136*
  cardiac catheterization, 142
  diseases, 428–429
  echocardiography, 140, *141*
  electrocardiography, 138–140
  evaluation, 133–142
  examination, *33*, 134–138, 428–429, 575
  exercise testing, 140–141
  history, 133–134
  laboratory tests, 141
  poor performance, causes, *459*
  radiography, 142
  signalment, 133
  vascular disease, 156–158
  see also Heart disease
Caries, dental, 13–14, *14*
Carpal canal syndrome, 374
Carpitis, 537
Carpus, 370–375
  anatomy, 370
  bone cysts, 537
  slab fractures, 511, *512*, 535
  stabilization, 324–325
Carpus, radiography, 316, 533–537
  anatomy, *512*, 534–535, *535–536*
  projections, *528–530*, 534
  technique, 534
Caslick vestibuloplasty, 176, *176*
Caslick vulvoplasty, 177, 291
Castor bean (*Ricinus communis*), 465
Castration, 169–170
  cryptorchid, 171, *172*
Cataract, 256, *256*
Catheter care, 44
Cauda equina neuritis, 232–233, *232*
Caudal aortic/iliac artery thrombosis, 397
Caudal epidural technique, 495–496, *495*
Cefalosporins, 443
Cefotaxime, 443
Ceftazidime, 443
Ceftiofur, 443
Ceftriaxone, 443
Celiotomy, 38
Cellular immune function, 199
Cellulitis, 243
  nodular skin diseases, 278–279
Centaurea repens (Russian knapweed), 218
Centaurea solstitialis (yellow star thistle), 218
Central nervous system (CNS) disorders, 434–436
  causes, 436
  toxic plants, 465–466
Cephalothin, *242*
Cephazolin, *242*
Cerebellar abiotrophy, Arabs, 216, 436–437
Cerebellar disease
  congenital, 216
  foals, 436–437
Cerebrospinal fluid (CSF) analysis, 215
Cervix, 290–291, 293
  cervical adhesions, 291
  cervical injection abscessation, 105
  cervical laceration, 291
  cervical spine, radiological examination, 224–225
  cervical tear, 298
  cervical vertebral stenotic myelopathy (CVSM), 225–226
  cervicitis, 290–291
Cheek teeth, extraction, 18–19, *19*
Chemical arthrodesis, 379–380
Cherries, 466
Chip fractures, 349–350, 371, 511, *512*
Chloramphenicol, *240*, *242*
Chlorhexidine, 476
Chlorinated hydrocarbons, 467
Choanal stenosis, 103
Cholangiohepatitis, 52–53, *53*, 445
Choledocholithiasis, 52–53, *53*
Chondroids, gutteral pouch, 81, 412, *412*
Chorioptes bovis, 264
Chorioptic mange, 264
Chorioretinopathies, 259
Chronic conditions
  active hepatitis, 53
  anaemia, *199–200*
  ATD diverticulitis, 81–82
  ATD empyema/chondroids, 81
  cough, 119, 131
  diarrhoea, 58, 62, 65
  grass sickness, 55, *56*
  haemorrhage, *199–200*
  inactive chorioretinopathies, 259
  infections, 183
  inflammatory bowel disease, 59–61, *60–61*
  interstitial inflammation, 127, *127*
  interstitial lung disease, *118*
  iridocyclitis (uveitis), endstage, 254–255
  keratinization plaques, 2
  lower airway inflammation, *118*
  obstructive pulmonary disease (COPD) *see* Recurrent airway obstruction (RAO)
  pulmonary disease (CPD) *see* Recurrent airway obstruction (RAO)
  pyelonephritis, 163
  renal failure (CRF), 162–163
  sacroiliac disease, 394, *394*
  small airway disease (SAD) *see* Recurrent airway obstruction (RAO)
  weight loss, 49, 58
Chrysops spp, 266
Cicuta spp (cowbane), 465
Cimetidine, 443
Ciproflaxin, *240*
Cirrhosis, 52
Cisapride, 27
Cladosporium spp, 248–249
Clarithromycin, 127, 414
Clenbuterol, *120*, 121
Clinical biochemistry, 391–392
Clitoris, enlarged, 292, *292*
Cloning, 307
Closed fracture, *502*
Closed pneumothorax, 129
Clostridial diseases, 410–411
  enterocolitis, 410–411
  myonecrosis, 411, 455
Clostridium spp, 31, 278, 410, 439–440
Clostridium botulinum, 229, 410
Clostridium difficile, 31, 127, 410–411, 441
Clostridium perfringens, 31–32, 410–411, 441
Clostridium piliforme, 415
Clostridium septicum, 411
Clostridium sordellii, 411
Clostridium sporogenes, 411
Clostridium tetani, 229–230, 410
Clovers, photosensitization, 467
Cloward technique, ventral fusion, 226
Coccidia spp, 422
Coccidioides immitis, 128, 277, 416
Coccidioidomycosis, 277
Coital exanthema, 302
Coital vesicular exanthema, 292, 403
Colic
  alimentary (true), 22
  anterior enteritis, 30–32
  classification, 22, 35
  clinical examination, 33–35, *33*
  common types, 27–30
  complications, postoperative, 45–47
  diagnosis, 32–35
    history, 32–33, *33*
    signalment, 32
  false, 21–22
  foals, 577
  imaging techniques, 35
  monitoring protocol, 44–45
  observation, 33
  overview, 21–22
  pharmacological management, 25–27
    aims, 25
    types, 25–27
  postoperative care, 44–45
  recurrent/chronic, *36*
  surgery, 35–44
  see also Grass sickness; Intestinal obstruction
Colitis, *409*
Collarettes, 262
Collateral cartilages, ossification, 526, *526*
Collateral desmitis of distal interphalangeal (DIP) joint, 345–346, *346*
Collateral ligament, 352, 382, 386
Colloids, 147, 562, *563*, 567, 572
Coloboma (dysgenesis), 244, 247
  fundus, 258
  uveal tract, 252–253
'Comet tails', *116*
Comminuted fractures, 349, 371, *506*, 535
Compartmental syndrome, 453
Compensated (hyperdynamic) shock, 567
Complete blood count (CBC), 196
Complexed calcium, 462
Compton effect, 500
Computed tomography (CT), 556–558
  indications, 556–558, *557*
  interpretation, 556
  principles, 556
Conception, failure, 307
Conchal necrosis/metaplasia, 91

Condensing lens, 236
Conducting airways, functions, 92
Conduction disturbances, 155–156, *157*
Condylar fractures, 511, *512*
Congenital conditions
  alimentary system, 441
  cerebellar disease, 216
  conjunctiva, 246
  cornea, 248
  cysts, 282
  eyelids, 244
  globe/orbit abnormalities, 243
  heart malformations, 142–143
  hepatobiliary, 445
  monorchidism, 170
  muscular system, 452–453
  palatal defects, 5, 103
  pigmentation disorders, 281–282
  scrotum, 301
  skin diseases, 281–282
  stationary night blindness, 258
  tracheal collapse, 103
  urogenital system, 446
  uveal tract, 252–253
  vitreal anomalies, 257
Congestive heart failure, 429
Conidiobolus spp, 276
Conidiobolus coronatus, 276, 416
Conium maculatum, 466
Conjunctiva, 246–247
  amyloidosis, 247
  biopsies, 238–239
  foreign bodies, 246
  pedicle graft, 243, *243*
  pseudotumour, 247
Conjunctivitis, 246
Contact dermatitis, 267–268
Contact lenses, 242
Contagious equine metritis (CEM), 290
  code of practice, 300
Continuous perfusion devices, 242
Continuous rate infusion (CRI), 190
Coombs test, 198
Copper sulphate, 481
Cor pulmonale, 429
Core vitrectomy, 255
Cornea, 247–252
  biopsy, 238
  examination, 237, *237*
  non-inflammatory disease, 248
  perforation, *251*
  reflex, 237
  stromal abscess, *251*
Coronavirus, 442
Cortical thickening, 501
Cortical/basal ganglia, 234
Corticosteroids
  equine protozoal myeloencephalitis (EPM), 227
  granulation, 479
  inflammatory bronchial disease, 579
  iridocyclitis, 255
  myocardial disease, 148
  navicular disease, 343
  recurrent airway obstruction (RAO), 120–121, *120*
  strongyles, 419
  thrombocytopenia, 204
  wound healing, 481
Corticotropin-like intermediate peptide (CLIP), 183–184
Corticotropin-releasing hormone (CRH), 183
  see also Adrenocorticotropin hormone (ACTH)
Corynebacterium spp, 299
Corynebacterium pseudotuberculosis, 279, 415
  abscesses, 278, 455, *456*
Cough, 112, 119, 131
Cowbane, 465
Crackles (rales), 113
Cranial (anterior) epidural technique, 496
Cranial cruciate ligament (CrCL), 386
Cranial mesenteric arteritis, *36*
Cranial nerves, 212–213, *213*
Cranial nerve disorders, 436
  evaluation, 433–434
  neurological signs, 234
Cranial thoracic mass, 125
Creatine phosphokinase (CK), 451–452
Crib-biting, 104
Crico-pharyngeal relaxation, deglutination, 2
Critical care
  blood products, 568–572
  see also Nutritional support
Cross-matching, 198
Crotalaria spp (rattlebox), 51–52, 217
Crust, 262
Cryogens, wound healing, 481
Cryosurgery, sarcoids, 278
Cryptococcosis, 277
Cryptococcus spp, 277
Cryptococcus neoformans, 128, 416
  var *gattii*, 277
  var *neoformans*, 277
Cryptorchidism, 170–171, 446
  cryptorchid castration, 171, *172*
Cryptosporidium spp, 422, 442, 576
Crystalline fluids, 147
Crystalloids, 562, *563*
Cuboidal bone, 439
Culicoides spp, 265–267, 405, 407
Culicoides brevitarsus, 266
Culicoides insignis, 266
Culicoides pulicaris, 266
Culicoides spinosus, 266
Culicoides stellifer, 266
Culicoides variipenis, 266
Cunean tenectomy, 379
Curb, 381
Curvularia geniculata, 276
Cushing suture, *40*
Cushing's syndrome, 167, 182–185, *183*, 188, 282
Cutaneous asthenia, 274
Cutaneous habronemiasis ('summer sores'), 174–175, *175*, 275, 473
Cyanoacrylate spray, 481
Cyanogenic glycosides, 466
Cyathostominosis, 419
Cyclicity, aberrant patterns, 304
Cycloplegics, 254–255
Cyclosporine A, *240*, 255
Cyproheptadine, 185, 222
Cysplatin, 278
Cystitis, 163
Cysts
  bone, 526
  congenital, 282
  cystic calculi, 164
  cystic glandular distension, 290
  cystic lesions, 501, *508*, 533, 543
  dentigerous, 549
  endometrial, 290
  epithelial inclusion, 289, *289*
  hydatid, 54, 243
  intra-mural oesophageal, 6
  mesonephric, 289
  osseous *see* Osseous cyst-like lesions
  palatal, 5, 103
  paramesonephric, 289
  pharyngeal, 5, 103
  sinus, 89
  sub-epiglottal, 5–6, *6*, 100
  subchondral *see* Subchondral cystic (cyst-like) lesions (SCLs)
  synovial, 226
  uveal, 253
Cytology, 238

D

Dakin's solution, 480
Dallas grass staggers, 228, 466
Darling pea (*Swainsona* spp), 217, 465
Dazzle reflex, 237
Dead men's fingers, 465
Debridement
  lavage, 475–476
  phase, wound healing, 474
  surgical, 475
Decompensated shock
  early, 567
  late (irreversible), 567
Deep cutaneous nodular mycoses, 276
Deep digital flexor tendon (DDFT), 337
  tendinitis, 364–365, *365*
  distal tendinitis, 344–345, *344*
  accessory ligament, desmitis, 367
Defibrillation, cardiopulmonary cerebral resuscitation (CPCR), 584
Degenerative disease
  bone lysis, fetlock, 532
  corneal, 248
  distal interphalangeal (DIP) joint, 346
  joint *see* Arthritis; Osteoarthritis
  retinopathies, 258–259
  testis, 302
Deglutination
  crico-pharyngeal relaxation, 2
  laryngeal protection, 2
  lingual function, 2
  mastication, 2
  oesophageal peristalsis, 2
  oesophageal phase, 2, 6–7
  oral phase, 2, 4–5
  palate, elevation, 2
  pharyngeal constriction, 2
  pharyngeal phase, 2, 5–6
  prehension, 2
Dehydration, signs, *562*
Dembrexine, 121
Demodex caballi, 270

Demodex equi, 270
Demodicosis, 270
Dental anatomy, normal, 8–9
  dental cup, 8
  dental star, 8, *9*
Dental disorders, 5, 548
  abrasion/attrition, 10–12
  caries, 13–14, *14*
  clinical signs, 15–16
  diagnosis, 15–19
  endodontic, 14
  extractions, 17–19, *19*
  investigations, 17
  oral examination, 16, *16–17*
  periodontal disease, 12–13, *12*
  radiography, 16–17, *17*
Dentigerous cysts, 549
Dentition, ageing, 15
Depigmentation, 281
Dermanyssus gallinae (red/poultry mite), 265
Dermatitis
  alternaria, 276
  contact, 267–268
  equine papular, 275
  equine ventral midline, 266
  exfoliative eosinophilic, 273–274
  exudative, 274
  pastern, 282
Dermatology *see* Skin diseases
Dermatophilosis, 268–269, *269*
Dermatophilus congolensis, 268, *269*
Dermatophytosis (ringworm), 269–270, 416
Dermoids, 244, 246
Desmitis
  accessory ligament, 367
  collateral ligament, 352
  distal sesamoidean ligaments, 350–351, *350*
  distal/proximal annular ligament, 351
  intersesamoidean ligament, 352
  metacarpophalangeal joint, 352
  suspensory ligament (SL), 365–367, *366*
Detomidine, 490–491, *495*, 496
  anaesthesia, 492
  colic, 26
Developmental disorders, 435
  cataracts, 256
  orthopaedic disease, 321–322
  skin diseases, 281–282
Dexamethasone
  atopic dermatitis, 267
  hypoadrenocorticism, 185
  insect hypersensitivity, 267
  ocular disease, *252*
  pemphigus foliaceus, 271
  purpura haemorrhagica, 282
  RAO, 120, *120*
  suppression test (DST), 184
  thrombocytopenia, 204
Dexamethasone HCl, *240*
Dextrose, 573
Diaphragm, 550
  hernias, 71
  ruptured, 130
  synchronous flutter, 455–456
Diaphyseal components, fractures, *502*, *512*, 532
Diarrhoea
  acute, 58, 65
  chronic, 58, 62, 65
  foal, 441–443
  postoperative, 47
Diastema, 8
Diastemata, *12*
Diastolic filling sound, 136
Diastolic heart sound, 135
Diastolic murmurs, 137
Diazepam, 217, 303, 435, 447, 491–492
Dictyocaulus arnfieldi (lungworm), 114, *118*, 124, 421
Diffuse chorioretinopathy, 259
Digital fat cushion, puncture, 337
Digital flexor tendon, longitudinal tears, 357
Digital flexor tendon sheath (DFTS), 337
  tenosynovitis, 357
  septic tenosynovitis, 357–358
Digital synovial sheath, 315
Digitalis spp, 466
Digoxin, 149
1,25-Dihydroxycholecalciferol, 462
Dimecaprol (BAL), arsenic and, 467
Dioctyl sodium succinate (DSS), 27
Dioestrus, 284, *284*
  prolonged, 304
Diphenhydramine, 267
Diprenorphine, 492
Dipyrone, 25, 27–29
Direct hernia, 67
Direct ophthalmology, 236, *238*
Dislocation, tarsus, 543
Disseminated intravascular coagulation (DIC), 198, *198*, *199–200*, 204–205, *204*
Distal annular ligament, 351
Distal humerus, stress fracture, 375
Distal interphalangeal (DIP) joint, 337
  analgesia, 314
  collateral desmitis, 345–346, *346*
  flexural deformity, 346–347, *347*
  septic arthritis, 346
  traumatic/degenerative arthritis, 346
Distal intertarsal joint, 315
Distal limb blocks, 312
Distal metacarpus, stabilization, 324–325
Distal phalanx
  bone contusion, 344
  fractures, 339–340, *339*
  osseous cyst-like lesions, 340–341
  puncture, 337
Distal radius, stabilization, 325
Distal sesamoidean ligaments, 350–351, *350*
Distributive shock, 426
Diuretics, 149
Diverticulitis, gutteral pouch, 81–82
DLPMO radiograph, *503*, *505*
DMPLO radiograph, *501*, *505*
DMSO (dimethyl sulfoxide), *252*, 435
'Dog-sitting', *454*
Domperidone, 27
  test, 184
Donor site, skin grafting, 482, 487
Dorsal displacement of soft palate (DDSP), 97–99
  aetiopathogenesis, 97–98
  clinical signs, 98
  diagnosis, 98
  prognosis, 99
  treatment, 98–99
Dorsal metacarpal disease, 360
Dorsal spinous processes, impingement, *391*, 393, *394*
Dorsal tarsus, *473*
Dorsopalmar imbalance, 338, 524
Dorzolamine, 255
Dourine, 422
Doxycycline, *240*, *252*, 270, 414
Dracunculiasis, 275
Dracunculus spp, 275
Drains
  disadvantages, 478
  types, 476, *477*
  wound closure, 476–478
Draschia megastoma, 422, 473
Dropped elbow, *453*
Drowning, lower respiratory tract (LRT), 129
Dry coat syndrome, 181–182
Dry eye syndrome, 248
Duodenitis jejunitis, 410–411
Duodenum, impaction colic, *28*
Dusts, hay/straw, 117–118
Dutonella spp, 421
Dyschondroplasia, 321–322
Dysgenesis *see* Coloboma
Dysmaturity, 424–425
  defined, 424
Dysphagia, 2–4
  aetiology, 4–7
  differential diagnosis, 106
  endoscopy *per nasum*, 3–4, *3–4*
  examination, 3
  history, 2–3, *3*
  oral examination, 4
  radiography/fluoroscopy, 4
Dyspnoea
  adult horse, 132
  obstructive, 93
Dystocia, 178
  reproduction, 297

E

Ear *see* External ear; Middle ear
Eastern equine encephalitis (EEE), 219
Ecchymoses, 210
Echinococcus granulosus, 422
Echium plantagineum (Patterson's Curse), 51–52
Ectoparasitic skin diseases, 264–265
Ectopic junctional rhythms, *136*
Ectopic ureter, 446
Ectropion, 245
Ehlers–Danlos syndrome, 473
Eimeria leuckartii, 422
Eisenmenger's physiology, 142
Ejaculatory dysfunction, 303, 308
  pharmacological aids, 303
Elbow, 375–376
  analgesia, 315
  anatomy, 375
  fractures, *506*, 538
  penetrating joint injury, 538
  stabilization, 325

Elbow, radiography, 316, 537–538
  anatomy, 538
  projections, *506*, 538
  radiopathology, 538
Electrolytes
  derangements, 565
  disturbances, 564–565
Electromyography, 452
Embryo transfer, 306–307
Embryonic period, 293
Emphysema, sub-cutaneous, 91
Empyema
  chronic ATD, 81
  primary/secondary, *86*, 87–88, *88*
Enalapril, 149
Enamel mark, 8
Encephalopathy
  hepatic, 49–50, 217
  neonatal, 577
Endocarditis, 146–147
Endocrinology, 181–193, 447–448
  anhidrosis (dry coat syndrome), 181–182
  equine metabolic syndrome (EMS), 186–188, *187*
  hyperlipaemia, 188–190, *189*
  hyperparathyroidism, 192–193, *192*
  hyperthyroidism, 191
  hypoadrenocorticism, 185
  hypothyroidism (goitre), 190–191
  insulin resistance (IR), 186–188, *187*
  phaeochromocytoma, 185–186
  pituitary pars intermedia dysfunction (PPID), 182–185, *183*
  thyroid adenoma, 191–192
Endodontic disease, 14
Endogenous serum/plasma, ocular disease, *240*
Endometrial conditions
  biopsy, 287, *287*
  cups, 293, 296
  cysts, 290
  cytology, 286, *286*
  haemorrhage, 298
  swabbings, 286, *286*
Endometritis, 289
Endometrosis (chronic degenerative endometritis), 290
Endoparasitic skin diseases, 265–266
Endophthalmitis, 244
Endophthalmos, 244, 260
Endosteal new bone, 501
Endotoxaemia, colic, 45
'Endplate flaring', 551
Enilconazole, 270
Enrofloxacin, 270
Enteral nutrition, 189, 572–573
  fluids, 563–564
Enterobacteriaceae, 248–249
Entheseopathies, radiography, *504*, 510
Entheseophytes, 371, 501, *503–504*
Entheseophytosis, fetlock, 531
Entropion, 244
Enucleation, 243
Environmental mites, 264–265
Enzootic lymphangitis, 277, 415
Enzymatic debridement, 480
Enzyme levels, interpretation, 452
Eosinopenia, 197
Eosinophilia, 197
Eosinophilic granuloma, equine, 280
Epaxial muscles, *390*
Epididymis, 302
Epidural injections, 494–496, *495*
  caudal technique, 495–496, *495*
  cranial (anterior) technique, 496
  indications, 495
Epiglottal entrapment (EE), 99–100, *99*
Epiglottal lesions, 5–6, *6*
Epiglottal retroversion, 103
Epilepsy, 221–222
  juvenile idiopathic, 435
Epinephrine, 583
Epiploic foramen entrapment (EFE), 70–71
Episclera, 246–247
Epistaxis, 132, 210
Epithelial inclusion cysts, 289, *289*
Epithelialization, 480
Epitheliotropic lymphoma, 274
Epizootic lymphangitis, 277
Equine Aeromask™, 120
Equine Cushing's disease (ECD) (pituitary pars intermedia dysfunction (PPID)), 167, 182–185, *183*, 188, 282
Equine degenerative myeloencephalopathy (EDM), 228
Equine encephalosis (EE), 407
Equine Haler™, 120
Equine herpesviruses (EHV), 402–403
  myeloencephalopathy, 227
Equine herpesvirus 1 (EHV-1), 402–403
  control, 403
  foals, 432, 445
  pregnancy, 294
  vasculitis, 227
  viral encephalitis, 435
Equine herpesvirus 3 (EHV-3), 403
Equine herpesvirus 4 (EHV-4), 403
Equine infectious anaemia (EIA), *199–200*, 294, 405
  immune-mediated haemolytic anaemia, 201
Equine metabolic syndrome (EMS), 186–188, *187*
Equine motor neuron disease (EMND), 228–229, 258
Equine protozoal myeloencephalitis (EPM), 221, 225, 227
Equine viral arteritis (EVA), 275, 294, 403–404, 432
  code of practice, 300
Equisetum arvense, 466
Erection failure, 308
Erosion, 262
Erythema, 262
Erythema multiforme, 280
Erythrocytes, 196–197, *198*
Erythromycin, 27, 126–127, 414
Escherichia coli, 125, 437, 441
Ethmoidal haematoma, 243
Etorphine, 492
Eumycotic mycetoma, 276
Eupatorium rugosum, 466
Everlasting pea, 466
Excoriations, 262
Exercise
  endoscopy, 94, *94*
  intolerance *see* Performance problems
  respiratory noises, 93
  tests, 93
Exercise-induced haematuria, 166
Exercise-induced pulmonary haemorrhage (EIPH), 114–115, 117, *118*, 123–124, *123*
Exertional rhabdomyolysis (ER), 454–455
Exfoliative eosinophilic dermatitis, 273–274
Exhausted horse syndrome, 458–460
Exophthalmos, 243, 260
Expectorants, 121
Expiratory dyspnoea, 112, *112*
Extensor process fractures, *526*
External carotid artery, rupture, 158
External ear, diseases, 78–79
  anatomy, 78
  investigations, 78
External genitalia, examination, 285, *285*
External hernia, 67, *68*
External nares, 83–84
  anatomy, 83
Extractions, tooth, 17–19, *19*
Exuberant granulation tissue ('proud flesh'), 277
Exudate, dried, 262
Exudative dermatitis, 274
Exudative nodular skin diseases, 275–278
Eyelids, 244–246
  lacerations, 242
  swelling, 260
Eyes, examination, 575
  see also Ophthalmology

F

Facet joints, 551
Facial nerve (VII), 212–213, *213*
Facial palsy, 4, 105
Facial paralysis, 83, 231
Facial trauma, 91
Faeces, 409
  faecal analysis, 440–441
  faecal egg count reduction test (FECRT), 416
  faecal larvae, 416
  faecal occult blood, 198
  faecal worm egg count (FWEC), 416
Faenia rectivirgula, 117–118
Fagopyrum spp, 467
Failure of passive transfer (FPT), 208, 448–449
  causes, 448–449
Fainting disease, 436
False colic, 21–22
'False rigs', 170
'False strangles', 278
'Farcy', 277, 415
Far-near sutures, 478
Fascial flap overlay technique, *70*
Fasciola hepatica, 52, 422
Feed-associated poisonings, 468
Fell pony syndrome, *199–200*, 207
Femoral nerve, damage, 231
Femoropatellar joint, 315
Fenoterol, *120*
Fertilization, 292

Fetlock joint disorders, 347–359
anaesthesia, 314–315
angular deformity, 359
differential diagnosis, 368
flexural deformity, 358–359, *358*
luxation, 351
osteoarthritis (OA), *503*
osteochondrosis, *507*
radiography, 316
Fetlock, radiography, 527–533
anatomy, *507*, 529–531, *529*
projections, 527–528, *528–530*
radiopathology, 531–533
technique, 528–529
Fibrin degradation products (FDPs), 198
Fibroblastic phase, wound healing, 474
Fibrotic myopathy, 387
Fine-needle aspiration, 238
Fissure fractures, 371, *513*, 515
Flair nasal strip, 124
Flatulent colic, 29–30
Flavivirus spp, 219–220
Flax, 466
Flexion tests, 311
Flexural limb deformities (FLD), 439
Flow cytometry, 198
Fluconazole, 128, *240*, *252*, 270
Fluid accumulation, skin grafts, 482
Fluid therapy, 561–565
balanced fluids, 562
cardiopulmonary cerebral resuscitation (CPCR), 584
colic, 27
electrolyte/acid-base disturbances, 564–565
neonatal, 565
plan components, 562–563, *562*
postoperative, 44
rate of administration, 563–564
reasons for, 561
route of administration, 563
types, 562–563, *563*
Flunisolide, 120
Flunixin, 32, 45, 278
Flunixin meglumine, *252*, 435, 494, 571
colic, 25, 29
diarrhoea, 443
Fluorescein sodium, 239
Fluorosis, 328
5-Fluorouracil, 278
Fluphenazine toxicity, 218–219, *219*
Flurbiprofen, *240*
Fluticasone propionate, 120–121, *120*
Fly irritation, 266
Foals
brain, 216–217
foaling mares, 304
neurological examination, 215
Focal chorioretinopathy, 259
Focal light source, 236
Focal pneumonia, *118*
Foetal circulation, reversal, 431
Foetal membranes, retained, 297
Foetotomy, 297
Food allergy, 268
Foot disorders, 330–347
bone contusion, 344
bruising/abscess, 334–335, *335*
canker, 336
distal tendinitis, 344–345, *344*
imbalance, 338–339, *338*
keratoma, 335–336
lameness, differential diagnosis, 368
nailbind, 335
'pedal osteitis', 340
quittor, 338, *338*
'sidebone', 341
sole, penetrating wounds, 336–337
thrush, 336
treatment, 334
see also Distal interphalangeal (DIP) joint; Distal phalanx; Hoofwall; Laminitis; Navicular bone
Foot flight
arc, 311
path, 311
Foot placement, 311
Foot, radiography, 316, 519–527
anatomy, 522–524, *522–524*
equipment, 519
indications, 519–527
preparation, 519
projections, 519
radiopathology, 524–527, *524*
technique, 519–522, *520–522*
Forage mites, 265
Forced extraction, obstetric, 297
Forebrain syndrome, 217
Foreign bodies
conjunctival, 246
external ear, 2
inhaled, 127
intra-nasal, 91
oral, 5
pastern wound, *472*
wound healing, 474
Forelimbs
examination, 214
lameness, bilateral, 234
stabilization, 324–325, *325*
Fourth branchial arch defects (4-BAD), 6, 101–102, *102*
Foxglove, 466
Fractures, 323–327
accessory carpal bone, 535
aftercare, 326–327
avulsion, 511
back, 393, *393*, 551
carpus, 370–372, *371*, 535–536
chip, 349–350, 371, 511, *512*
classification, 323–324
closed, *502*
comminuted, 349, 371, *506*, 535
complications, 324
condylar, 511, *512*
diaphyseal components, *502*, *512*, 532
distal humerus stress, 375
distal phalanx, 339–340, *339*
elbow, *506*, 538
evaluation, 324
fissure, 371, *513*, 515
foot, *514*, 525–526, *526*
fracture lines, 511, *513*
hairline, *513*
head, 548
healing, 324
hip, 387–388
ilial wing, *555*
intra-articular, 383, *383*
long-bone, 45, 511, *511*
longitudinal frontal, 349
medication, 325
metacarpal/metatarsal bone, third, 359–360
middle phalanx, 348
navicular bone, 341, *342*, *514*
palmar, 350
patellar, 386
pelvic, 395–397, *396*, 546–547
plantar process, 350
proximal phalanx, 348–350
proximal sesamoid bone, 354–355, *355*, 531
radiography, 325, 510–516
ribs, 439
sagittal, 349
shoulder, 377–378, 539
slab, 371, *383*, 511, *512*, 535
splint bones, 361–363
stabilization, emergency, 324–325
stifle, 545
surgical techniques, 325–326, *326*
tarsus, *512*, 543
transport, emergency, 325
ulnar, 375
Free grafts
classification, 481–482
defined, 481
Free-radical scavengers, 435
Fresh plasma (FP), 571–572
Fresh-frozen plasma (FFP), 571
Frog clefts, 511, *514*
Frog pads, 333, *334*
Frontonasal suturitis, 549
Frusemide (furosemide), 123, 147, 149
Full-thickness grafts, 479
defined, 481
sheet, 482, *483*
Functional dynamic stenosis, 226
Functional murmurs, *137*
Functional obstructions, 444
Fundus, 257–259
Fungal infections, 416
abortion, 294
pneumonia, 128, *128*
'Funnelling', 551
Furosemide, 123, 147, 149
Fusarium spp, 248–249
Fusobacterium spp, 278

G

Gait
abnormalities, *214*
evaluation, lameness, 310–311
examination, 213–214, 434
Gamete intrafallopian transfer (GIFT), 307
Gastric impaction, 57–58, *58*
Gastric neoplasia, 58, 74
Gastric ulceration (gastric ulcer syndrome), *36*, 56–57, *57*, 577
Gastrocnemius tendonitis, 381–382
Gastroduodenal jejunitis, 30–32
Gastroduodenal ulcers, *36*, 441, 443

Gastroenterology, 64–65
  colic *see* Colic
  differential diagnosis, 65
  liver *see* Liver disease
  pancreatic disease, 54
Gastrointestinal system, 550
  blood loss, 210
  examination, 575
  neoplasia, 74–75, *74*
General anaesthesia
  complications, 496–497
  induction, 491–492
Generalized bone marrow suppression, *199–200*
Genital tract
  abnormalities, 300–303
  internal, examination, 285–286
Genitalia, external, 285, *285*
Gentamicin, *240*, *242*, *252*, 323
Gestation
  late, 295
  prolonged, 295
Giardia spp, 422, 576
Glanders ('farcy'/enzootic lymphangitis), 220, 277, 415
Glaucoma, 255
Globe, 243–244
  size/asymmetry, 260
Glossopharyngeal nerve (IX), 213, *213*
Glucocorticoids, 226, 267–268, 280, 282
Glucose-insulin test, 187
Gluteal muscle atrophy, *232*
Goitre, 190–191
'Golden period', wound management, 478
Gomen disease, 218
'Gonitis', 386
'Goose-stepping', 214, *214*
Gramicidin, *240*
Granula iridica, hypertrophy, 253
'Granular dermatitis', 174–175, *175*, 275, 473
Granulation tissue, 479–481, *481*
Granulocytic ehrlichiosis, equine, 415
Granulomatous enteritis, *61*
Granulosa cell tumour, 287, *287*
Grass sickness, 7, 54–55
  acute, 55
  sub-acute, 55
  chronic, 55, *56*
Greasy heal, 282
Griseofulvin, 270
Groundsel (*Senecio* spp), 51–52, 217
'Grunt-to-the-stick' test, 96
Guaiphenesin, 492
Gut lumen, occlusion, *36*
Gut viability, 39
Gutteral pouches (auditory tube diverticulum (ATD)) disorders, 80–81, 83
  anatomy, 80
  differential diagnosis, 106
  diverticulitis, 81–82
  empyema, *412*
  endoscopy, 80
  foals, 432
  mycosis, *3*, 82–83, *82*
  physical examination, 80
  radiography, 80
  radiopathology, 548
  signs, 80
  surgery, 80–81
  topical treatment, 80
  tympany, 81

H

Habronema larvae, *175*
Habronema spp, 174, 277
Habronema majus, 422, 473
Habronema muscae, 422, 473
Haematobia irritans, 266
Haematocoele, 300, *300*
Haematology values, 197, *197*
Haematoma, 288, *288*
  anaesthesia, 496
  prepuce/penis, 302
  progressive ethmoidal (PEH), 88–89, *89*
  testis, 301
Haematopoietic system, 195–210
  diagnostic approach, 195–199
  differential diagnosis, 210
  disorders, features, 199, *199–200*
  see also Anaemia
Haematopota spp, 266
Haematuria, 165–166, 210
Haemochromatosis, 53
Haemoglobin, renal disease, 447
Haemolymphatic disorders, 448–450
Haemolysis, 210
β-Haemolytic streptococci, 480
Haemorrhage, 196
  acute, *199–200*
  anaemia, 200–201, *201*
  chronic, *199–200*
  endometrial, 298
  exercise-induced pulmonary (EIPH), 123–124, *123*
  intravitreal, 257
  postpartum, 297–298
  retinal, 258
Haemorrhagic shock, 427, 568
Haemostasis, hereditary defects, 206
Haemostatic function, evaluation, 198, *198–199*
Haemothorax, 129
Hairline fracture, *513*
Halothane, 493
Hay dust, 117–118
Head
  big, 327
  fractures, 548
  loupe, 236
Head, radiography, 547–549
  anatomy, 548
  projections, 547, *547*
  radiopathology, 548–549
Headshaking, 91–92, 222
Heart disease
  defects, acquired, 429
  heart failure, 148–149
    congestive, 429
  overview, 133
  structural, 142–148
Heart murmurs, 137–138, *137*
  acquired, 429
  foals, 428
Heart rate, 33, *136*, 138, 428
Heart rhythm, 138, 428
Heart sounds, 134–136, *135*, *137*, 138
Heat stroke, 458
Heavy metals, 467
Heinz bodies, 197
Heinz body haemolytic anaemia, 202
Helminth dermatoses, 266
Hemicircumferential periosteal transection, 374
Hemlock, 466
Hendra virus, 406
Heparin, 205
Heparin sulphate, 190
Hepatic disorders
  chronic active hepatitis, 53
  damage, 189
  encephalopathy, 49–50, 217
  lipidosis, 445
  see also Liver disease
Hepatobiliary diseases, 444–445
  congenital, 445
  examination, 444
  infectious, 444–445
  toxic, 445
Hereditary equine regional dermal asthenia (HERDA), 274
Hereditary multiple exostoses, 328
Hernias, 67–71, 444
  congenital, 446
  definitions, 67, *68*
  diaphragmatic, 71
  incisional, 69–70
  types, 67, *68*
  umbilical *see* Umbilical hernias
  ventral, 69
Herpes coital exanthema, 275
Herpesvirus *see* Equine herpesviruses (EHV)
High 4-point nerve block (H4-PNB), 313, *313*
High speed treadmill endoscopy (HSTE), 94
High-gammaglobulin plasma, 572
Hindbrain syndrome, 217
Hindlimb
  examination, 214
  incoordination, 392
  lameness, 392
  stabilization, 325
Hip, 387–388
  anatomy, 387
  luxation, 387, 397
  radiography, 316
Hippomanes, 293
Hirsutism, 182–183, *183*
Histoplasma spp, 277
Histoplasma capsulatum, 128
Histoplasma farciminosi, 277
Histoplasmosis (epizootic lymphangitis), 277
'Hives' (wheals), 262
Hock, 378–384
  anatomy, 378–379
  radiography, 316
Hoest–Degen body, 220
Hoof pastern axis, 524
Hoofwall
  avulsion, 331
  cracks, 330
  separation, 330–331

Horizontal mattress tension sutures, 478
Hormones
  analysis, 287
  assays, 168
  low libido, 303
  stimulation tests, 191
Horner's syndrome, 105, 233
Horse pox, 274
Horsetail, 466
Hospitalization, complications, 45–47
Howell–Jolly bodies, 197
Hyalomma anatolicum, 220
Hyaluronic acid (HA), 315–316
Hydatid cysts, 54, 243
Hydrangea spp, 466
Hydrangeas, 466
Hydration, *33*, 35
Hydrocephalus, 216, *216*
Hydrocoele, 300–301, *301*
Hydrocortisone acetate, 267
Hydromorphone, *495*, 496
Hydrops, 295
Hydrotaea spp, 266
Hydroxyzine, 267
Hyoid disorders, 4–5
Hyoscine, 26–29
Hyovertebrotomy, 80
Hypercalcaemia, 462
  causes, 462
  of malignancy, 192
Hypercapnia, 497
Hyperdynamic shock, 567
Hyperhidrosis, 184
Hypericum perforatum, 467
Hyperimmune plasma/serum, 572
Hyperkalaemia, 156, *157*, 464, 565
Hyperkalaemic periodic paralysis (HYPP), 156, 452–453
Hyperlipaemia, 188–190, *189*
Hyperlipidaemia, 189
Hypermagnesaemia, 463
Hypermetria ('goose-stepping'), 214, *214*
Hypernatraemia, 463, 564
Hyperphosphataemia, 463
Hyperpigmentation, 262
Hypersensitivities, 266–268
Hyperthermia, malignant, 456
Hyperthyroidism, 191
Hypertonic fluids, *563*, 567
Hypertrichosis, 282
Hypertrophy
  alar folds, 83
  granula iridica, 253
  osteopathy, 328
  pulmonary osteoarthropathy, 328
Hyphomyces destruens, 276, 416, 473
Hypoadrenocorticism, 185, 447–448
Hypocalcaemia, 462–463, 565
  causes, 463
  idiopathic, 230–231
Hypoderma spp, 221, 422
Hypoderma bovis, 275
Hypoderma lineatum, 275
Hypodermiasis, 275
Hypogammaglobulinaemia, transient, 208, 450
Hypoglossal nerve (XII), 213, *213*
Hypoglycaemia, 576
Hypokalaemia, 464, 565
Hypomagnesaemia, 463, 565
Hyponatraemia, 463, 564
Hypophosphataemia, 463
Hypoplasia, testis, 302
Hypotension, 497
Hypothyroidism (goitre), 190–191
Hypotonic fluids, *563*
Hypovolaemic shock, 427
Hypoxaemia, 497
Hypoxaemic shock, 427

I

Idiopathic conditions
  chronic diarrhoea, 62
  headshaking, 91–92
  hypocalcaemia, 230–231
  renal haemorrhage, 165–166
  seizures, 216–217
  synovitis, 319
  tachypnoea, 431
IgM deficiency, selective, 207
Ileocolonic aganglionosis, 441
Ileum
  impaction colic, *28*
  resection, 40–41, *41*
Ileus, 444
Ileus, postoperative (POI), 45–46
  categories, 46
Ilial wing, fracture, *555*
Imidocarb, 203
Imiquimod, 278–279
Immobilon, 492
Immune system, 195–210
  diagnostic approach, 195–199
  differential diagnosis, 210
  disorders, features, 199, *199–200*
  function, evaluation, 199
  see also Anaemia
Immune-mediated conditions
  anaemia, *199–200*
  skin diseases, 270–273
  thrombocytopaenia (IMTP), *199*, *199–200*, 204, *204*, 450
Immune-related haemolytic anaemia, 201–202
Immunoglobulin assays, 199
Immunological disorders, 448–450
Impaction colic, 28–29, *28*
Impar ligament, 337
Impression smears, 238
Incarcerated hernia, 67
Incisional hernias, 69–70, *70*
Incisor teeth, extraction, 18
Indirect hernia, 67
Induction, parturition, 296–297
Infectious conditions
  arthritis, *500*, 538, 543, 545
  brain, 219–221
  hepatobiliary, 444–445
  investigations, 400
  myopathy, 455
  nodular papular skin diseases, 279
  osteitis, 510
  osteoarthritis (OA), *500*, 510
  recurrent, 210
  skin grafts, 482
  ulcerative/exudative nodular skin diseases, 275–277
  wound healing, 480
  see also Bacterial infections; Fungal infections; Viral infections
Inflammatory conditions
  airway disease (IAD), 122–123
  bowel disease, chronic, 59–61, *60–61*
  bronchial disease, 579–581
  inflammatory phase, wound healing, 473–474
  nictitating membranes, 245
  palpebrae, 245
  spinal cord, 227
  vitreal, 257
Influenza, equine, 401–402
Inguinal hernia, 171–173, *172*
Inguinal rupture, 171–173, *172*
Inguinal testis, 171
Inhalation
  anaesthesia, 493
  foreign bodies, 127
  smoke, 130–131
  therapy, 120–121
Insect irritation, 266
  hypersensitivity, 266–267
Insecticides, 467
Inspiratory dyspnoea, 112, *112*
Insulin resistance (IR), 186–188, *187*
  management, 188
Insulin response, dexamethasone suppression test (DST), 184
Integument, examination, 575
Interdental space, 8
Internal carotid artery, rupture, 158
Internal fixation techniques, fracture, *326*
Internal hernia, 67, *68*
Interneural articulations, arthropathy, 226
Intersesamoidean ligament, 352
Intersex, 292, *292*
Interstitial disease, 581
  pneumonia, 127, *127*, 433
Interstitial pattern, lung, 550
Intestine, motility, 27
Intestinal disorders, 58–65
  diagnosis, 59
  function tests, 59
  lesions, gross, 31
  neoplasia, *74*
  pancreatic disease, 54
Intestinal obstruction, 22–25
  simple, 22–24
  strangulating, 24–25
Intra-abdominal haemorrhage, 210
Intra-articular fractures, 383, *383*
Intracameral injection, 242
Intracarotid injection, 222, 496–497
Intracytoplasmic sperm injection (ICSI), 307
Intralesional medication, 242
Intra-mural oesophageal cysts, 6
Intra-narial larynx, 92–93
Intra-nasal foreign bodies, 91
Intranasal oxygen insufflation, 571
Intraosseous fluids, 563
Intrasynovial analgesia, 312, 314–315
Intrathoracic haemorrhage, 210

Intrauterine adhesions, 290
Intravenous fluids
  administration, 563, *564*
    rate, 563–564
  colic, 27
  dextrose, 573
  diarrhoea and, 442
  maintenance, 563
  types, *563*
Intravitreal cyclosporine A implant, 242
Intravitreal haemorrhage, 257
Intussusceptions, 444
Ionized calcium, 462
Ionophore antibiotic toxicity, 457
Ipratropium, 121
Ipratropium bromide, *120*
Iridocyclitis (uveitis), 253–254, *253*, *254*
  acute, 254
  chronic/end-stage, 254–255
Iris
  examination, 237–238
  hypoplasia, 253
Iron
  deficiency anaemia, 203
  fumarate toxicosis, 445
  overload (haemochromatosis), 53
  status, 198
Ischaemic retinopathy, 258–259
Island grafts, 482, 485–487
Isoerythrolysis (NI), neonatal, 208, 449–450, 577
Isoflurane, 493
Isoimmune haemolytic anaemia, 449–450
Isotonic crystalloids, 567
Isotonic fluids, *563*, 571
Isoxsuprine hydrochloride, 333, 343, 354
Itch (pruritis), 263–268
Itraconazole, *240*, *252*
Ivermectin, 124, 264–266, 275, 418
  mites, 265
  resistance, 418
Ixodes spp, 415

J

Japanese B encephalitis (JE), 219
Jaundice, 65
Jaw tumours, 15
Jejunocaecal anastomosis, *41*
Joint diseases, 319–323
  idiopathic synovitis, 319
  osteoarthritis, 320–321, *321*
  osteochondritis dissecans (OCD), 321–322
  septic arthritis, 323
  traumatic arthritis/synovitis, 319–320
Juglans nigra, 466
Jugular thrombophlebitis, 156–157
Junctional arrhythmias, 153–155, *154*
Juvenile idiopathic epilepsy, 435

K

Keratitis, 248–252
  parasitic, 251–252
  traumatic, 248, *249*
  ulcerative, 248–250, *249*
  viral, 251
Keratoconjunctivitis sicca (KCS), 247
Keratoma, 335–336, 525, *525*
Keratomalacia ('melting cornea'), 250–251, *250–251*
Ketamine, 435, 491, *495*
  anaesthesia, 491–492, 494
Ketoconazole, 128
Ketoprofen, 443, 494
Kimzey splint, *325*, 349
'Kissing spines', *391*, 393, *394*, 395, *506*, 551
Klebsiella spp, 125, 441
Klebsiella pneumoniae, 300
Klossiela equi, 422
'Kunkers', 276, 473

L

Lactate dehydrogenase (LDH), 452
Lactation tetany, 230–231
Lactational anoestrus, 304
Lactulose, 54
Lameness, 309–328
  arthrocentesis, 315–316
  arthroscopy, 318–319, *319*
  bone diseases, 327–328
  computed tomography (CGT), 318
  diagnostic approach, 309–319
  differential diagnosis, 368
  examination, 310
  foot imbalance, 338–339, *338*
  gait evaluation, 310–311
  hindlimb, 392
  history, 309–310
  magnetic resonance imaging (MRI), 318
  nuclear scintigraphy, 318
  provocative tests, 311
  radiography, 316
  regional analgesic techniques, 311–315
  scales, 311
  thermography, 318
  ultrasonography, 316–318, *317*
  see also Fractures; Joint diseases
Laminitis, 183, 331–334, *332*, *504*, 524–525
  aetiology, 331–332
  classification, 332
  clinical signs, 332–333
  colic, 46–47
  definition, 331–334
  pathogenesis, 331
  prognosis, 334
  radiology, 333
  treatment, 333–334
Laparoscopy, 176
Laparotomy (celiotomy), 38
  closure midline incision, 43–44, *43*
Large intestine (colon)
  displacement/volvulus, 444
  examination, 38–39
  gut lumen, occlusion, *36*
  impaction colic, *28*
  resection, 42–43, *42*
  simple obstruction, 23–24, *24*
  strangulating obstruction, *24*, 25
Larynx, 432
  abnormalities, 6
  adductor response test ('slap test'), 213, *213*, 225
  advancement ('tie forward'), 99
  diseases of, 548–549
  intra-narial, 92–93
  neoplasia, 103
  protection, deglutination, 2
Lasalocid, toxicity, 457
Lateral buccotomy extraction, 19
Lateral femorotibial joint, 315
Lateral palmar nerve block, 314
Lateromedial imbalance, 339, 524
Lathyrus hirsutis, 466
Lathyrus latifolius, 466
Lathyrus odoatus, 466
Lavage
  debridement, 475–476
  solutions, 476
Lavender foal syndrome (LFS), 435–436
Lawsonia intracellularis, 413–414, 441
Laxatives, 26–27
Lead poisoning, 218
'Leeches', 276, 473
Lens, 256–257
  examination, 238
Leptospira spp, 162
  serovar Pomona, 414
Leptospirosis, 414
Lethal white syndrome, 441
Lethargy, 184
Leucoderma, 281
Leucoencephalomalacia (LEM) (mouldy corn poisoning), 217–218
Leucotrichia, 281
  reticulated, 281
  spotted, 281
Leukocytes, 197
Levothyroxine sodium, 188, 191
Libido, low, 303, 308
  hormonal causes, 303
  musculoskeletal causes, 303
Lice (pediculosis), 264
Lichenification, 262
Lidocaine, 155, 494, *495*, 496, 571
Ligaments
  injuries, 386
  thoracolumbar/sacral spine, *390*
Limbus, 246–247
Lime sulphur, 270
Linear keratopathy, 248
Lingual abnormalities, 5
Lingual function, deglutination, 2
Linum spp, 466
Lip lesions, 4
Liver disease, 49–54, 435
  abscesses, 445
  causes, *50*
  diagnosis, 50–51, *50*
  liver failure, 54
  liver fluke, 52
  uncommon, 54
Local anaesthesia, *495*, 496
Local analgesia, 312
Locomotion, respiration and, 92
Locoweed (*Astragulus*/*Oxytropus* spp), 217, 465
Long-bone fractures, 45, 511, *511*
Longitudinal frontal fractures, 349
'Long-toe/low-heeled foot', 338

Low 4-point nerve block (L4-PNB), 313, *313*
Lower limb
  differential diagnosis, 368
  perineural analgesia, 313–314
  peripheral nerve blocks, 312, *313*
Lower respiratory tract (LRT), 111–132, *459*
  acute respiratory distress, 578
  auscultation, 112–113, *113*
  diagnosis, 111–117, *112*, *118*
  diaphragm, ruptured, 130
  drowning, 129
  emergency treatment, 579–581, *581*
  endoscopy, 113–114, *114*
  examination, 578–579
  foreign bodies, inhaled, 127
  haematology, 117
  haemothorax, 129
  investigations, 582
  lungworm, 124
  palpation, 112
  Parascaris equorum infection, 124
  percussion, 113, *113*
  pleural transudates, 130
  pneumothorax, 129–130, *130*
  radiography, 116, *116*
  smoke inhalation, 130–131
  ultrasound, 116–117, *116*
  see also Airway obstruction; Pneumonia; Pulmonary conditions; Recurrent airway obstruction (RAC)
Lumbosacral (LS) space, 215
Lungs
  abscess, *118*
  biopsy, 430
  metastases, 124
  sounds, 429
  tumours, 124
Lungworm (*Dictyocaulus arnfieldi*), 114, *118*, 124, 421
Lupus erythematosus (LE), 272–273
Luteal insufficiency, 295
Luxation
  fetlock joint, 351
  hip, 387, 397
  lens, 257
  pelvis, 546
  shoulder, 378
  tarsal, *511*, 543
  tarsocrural joint, 383
Lyme disease, 415
Lymphangitis, sporadic, 209
Lymphocyte typing, 199
Lymphocytosis, 197
Lymphoma
  epitheliotropic, 274
  gastric, 74–75
  haemolymphatic, 208
  peripheral nerve, 233
Lymphopenia, 197
Lymphosarcoma, 281

M

Mach lines, *501*, 511
McMaster method (modified), 416
Macule, 262
Magnesium
  metabolism, 463
  supplementation, 155
Magnesium sulphate, 435
Magnetic resonance imaging (MRI), 556, 558
  advantages/disadvantages, 558
  history, 558, *558*
  indications, 558, *559*
  principles, 558
Magnification, 236
Maintenance fluid, 562
Malignant hyperthermia, 456
Mandibular fractures, 7
Mannitol, 435
Maple (*Acer* spp), 466
Marestail, 466
Marie's disease, 328
Mastication, deglutination, 2
Maturation (remodeling) phase, wound healing, 474
Maxillary artery, rupture, 158
Mayo sutures, 68, *69*
Mean corpuscular haemoglobin (MCH), 196
  concentration (MCHC), 196
Mean corpuscular volume (MCV), 196
Meconium
  aspiration, 431
  impaction/retention, 443
Medetomidine, 490–491
Medial femorotibial joint, 315
Median nerve block, 314
Mediastinal tumours, 125
Megoesophagus, 6
Melanomas, 281
  parotid, 104
'Melting cornea', 250–251, *250–251*
Menace response, 237
Meningitis, 221
  septic, 435
Meniscal injury, 386–387
Meniscotibial ligament tears, 386–387
Meperidine, 494
Mepivacaine, *495*, 496
Mesentery
  closure, 40
  neoplasia, *74*
Mesh prostheses, 68–69, *69–70*
Meshing sheet grafts, 484, *484*
Mesonephric cysts, 289
Metabolic disorders, 447–448
  acidosis, 464, 565
  alkalosis, 465, 565
  myopathy, 455–457
  poor performance, *459*
Metacarpal bone
  third, fractures, 359–360
  ultrasonography, 317, *317*
Metacarpophalangeal joint, 311, 314–315, 352
Metacarpus
  diseases, 359–367
  stabilization, 324–325
Metals, heavy, 467
Metatarsal bone, third, fractures, 359–360
Metatarsal pain, 360–361
Metatarsophalangeal joint, 311, 314–315, 352
Metatarsus diseases, 359–367
Metformin, 188
Methadone, *495*, 496
Methylprednisolone acetate, *242*
Metoclopramide, 27
Metritis
  acute toxic, 290
  contagious equine (CEM), 290
  endometritis, 289
  toxic, 298
Metronidazole, 32, 443
Miconazole, *240*
Microangiopathic anaemia, 202
Microbial culture, 263
Microbiology, 238
Micronema deletrix, 221, 422
Microphthalmos, 243
Microsporum canis, 269
Microsporum equinum, 269
Microsporum gypseum, 269
Mid radius, stabilization, 325
Midazolam, 435, 491–492
Midbrain syndrome, 217
Middle carpal joint, 315
Middle ear, diseases, 79–80
  anatomy, 79
Middle phalanx
  bone contusion, 344
  fracture, 348
Middle uterine artery, rupture, 158
Migrating helminths, 227
Milkweeds, 466
Mineral oil, 26, 54
Mineralization, corneal, 248
Minimum contamination technique, 305
Minoxidil, 271–272
Mites
  environmental, 264–265
  rare, 265
Mitral regurgitation (MR), 144
Modified Whitehouse approach, 80–81
'Monday morning leg (disease)', 209
Monensin, 457
Monocytosis, 197
Monorchidism, congenital, 170
Morphine, 26, 494, *495*, 496
Mouldy corn poisoning, 217–218
Moxidectin, 266, 275, 418
Mucokinetic agents, 121
Mucolytics, 121
Mucometra, 290
Mucor spp, 128
Mucous membranes, 196
  examination, 34, 575
Multifidus dorsi, *390*
Multiple body systems, systemic disease, 425–428
Multiple cartilaginous exostoses, 328
Musca spp, 266
Muscular system, 451–452
  biopsy, 452
  clinical pathology, 451–452
  congenital disease, 452–453
  electromyography, 452
  examination, 451
  muscle wastage, 390–391
  pedicle grafting, 97
  see also Myopathy

Musculoskeletal disease, 439
examination, 439
low libido, 303
poor performance, *459*
radiography, 509–516
Musculoskeletal system, 550
examination, 575
immaturity, 424, *425*
Mutation, 297
Mycobacterium spp, 415
Mycoplasma felis, 125
Mycotic rhinitis/sinusitis, 89–90
Mycropolyspora faeni, 117–118
Mydriatics, *240*, 250, 254–255
Myelination, variations, 258
Myocardial disease, 147–148, 429
Myopathy
atypical, 457–458
fibrotic, 387
infectious origin, 455
metabolic origin, 455–457
nutritional origin, 457
ossifying, 387
physical causes, 453–455
polysaccharide storage (PSSM), 456–457
postoperative, 45, 497
Myotonia, 452

N

Nagana, 421–422
Nailbind, 335
Naloxone, 571
Nannomonas, 421
Narcolepsy, 222
Narcolepsy-cataplexy, 436
Narcotics, 26
Nasal passages, 431–432
Nasal conditions, 548
amyloidosis, 281
reflux, *3*, 131
sinus disease *see* Sinu-nasal disorders
Nasal discharge, 119
differential diagnosis, 106
lower respiratory tract (LRT) disorders, 112
Nasal septal disorders, 90–91
deviation, 90
resection, 91
Nasogastric intubation, 34–35
Nasolacrimal system, 247
drainage assessment, 239
lavage, 241
Natamycin, *240*
Navicular bone
disease, 341–344, *342*, 526, *527*
fractures, 341, *342*, *514*
radiographic anatomy, 523–524, *523–524*
Navicular bursa, 314, 337
Navicular bursoscopy, 337
Near East equine encephalitis (NEEE), 220
Neck, examination, 214
Neck, radiography, 550–551
anatomy, 550
projections, 550
radiopathology, 550–551
technique, 550
Necrotic tissue, 480
Needle electromyography (EMG), 215
Neomycin, 45, 54, *240*
Neonatal foal, 574–578
babesiosis, 208
encephalopathy, 577
equine herpesvirus 1 (EHV1), 402–403
evaluation, 574–576
examination, 574–575
fluid therapy, 565
history, 574
investigations, 575–576
isoerythrolysis (NI), 208, 449–450, 577
maladjustment syndrome (NMS), 434–435
management, 576–578
nursing care, 577
stabilization/management, 576–578
vital parameters, 574
Neoplasia (neoplastic disease), *118*, 243
abdominal *see* Abdominal neoplasia
back, 394
brain, 221
conjunctival, 246–247
corneal, 252
external ear, 2
eyelids, 245–246, *245*
gastric, 58
gutteral pouch, 83
nodular papular skin, 281
oesophageal, 6
omentum, *74*
optic nerve, 259
oral, 5
ovarian, 287–288, *287*
penis/prepuce, 174
peripheral nervous system, 233
pharyngeal/laryngeal, 103
reproductive tract, female, 178, *178*
sinu-nasal, 90
spinal cord, 228
testes, 302
ulcerative/exudative nodular skin, 277–278
urogenital tract, 75
uterine, 290
uveal tract, 255
vesicular, 166
vitreal, 257
vulva, 292
Neorickettsia risticii, 203, 413
Neostigmine methyl sulphate, 27
Nephrolithiasis, 164
Nephrotoxic drugs, 166
renal failure, 447
Nerium oleander, 465–466
Nerve blocks, 236, *237*, 311
Nerve pedicle grafting, 97
Neurological disease, 433–438
equine herpesvirus 1 (EHV1), 402–403
examination, 433
imaging, 434
poor performance, *459*
Neurology, 211–234
diagnostic approach, 212–216
differential diagnosis, 234
hindlimb incoordination, 392
see also Brain; Peripheral nervous system; Spinal cord
Neuropathy
postoperative, 497
see also Recurrent laryngeal neuropathy (RLN)
Neutropenia, 197, 450
Neutrophilia, 197
Newsprint test, 189, *189*
Nictitans flap, 242
Nictitating membranes
inflammatory diseases, 245
prominence, 245
Nightshades, 466
Nocardia spp, 125, 263, 276, 278
Nodules, 262
nodular parasitic infections, 275–276
nodular skin diseases, 275–281
Non-oxygen-carrying fluids, *563*
Non-steroidal anti-inflammatory drugs (NSAIDs)
anaesthesia, 494
back conditions, 395
bone contusion, 340
cervical laceration, 291
colic, 25
corneal stromal abscess, 251
diarrhoea, 443
disseminated intravascular coagulation (DIC), 205
iridocyclitis, 255
pericardial disease, 147
pododermatitis, 335
renal disease, 447
seizures, 435
septic arthritis, 323
squamous cell carcinoma (SCC), 278
thrombosis, 397
toxicity, 64
ulcerative keratitis, 250
vascular disease, 333
wound healing, 479
Nose *see* External nares
Nostrils, occluding, 112–113
Nuchal fractures, 105
Nucleic acids, detection, 400
Nutrient foramina, 511, *514*
Nutritional disorders
myodegeneration, 457
secondary hyperparathyroidism (NSH), 192, 327, 448
Nutritional support, 572–574
foals, 442, 574, 577
key points, 572
postoperative, 44
requirements, 572–574

O

Oak (*Quercus* spp), 466
Obel grading system, laminitis, 332
Obesity, management, 187–188
Obstetrics, 297
Obstructive disorders
dyspnoea, clinical signs, 93
nasolacrimal system, 247
upper airway diseases, *459*
Occipitoatlantoaxial malformation (OAAM), 225, 437

Occlusion, acquired, 247
Ocular conditions
  discharge, 260
  opacity, 260
  therapeutics, 239–243, *240*
Ocular surface, 237
Oculomotor nerve (III), 212, *213*
Odontogenic tumours, 15
Oedema, corneal, 248, 254, *254*
Oenanthe crocata, 465
Oesophageal conditions
  neoplasia, 6
  obstruction ('choke'), *3*, 6–8
  peristalsis, deglutination, 2
  rupture, 6
  stenosis, 441
  strictures/stenosis, 6
Oestrogen, 293
Oestrous cycle
  manipulation, 304–305
  stages, 283–285
Oestrus, 283–284, *284*
  excessive signs of, 307
  persistent, 304
  split, 304
Ointments, 241
Oleander, 465–466
Olecranon fracture, *506*
Olfactory nerve (I), 212, *213*
Omentum, neoplasia, *74*
Omeprazole, 443
Omphalitis, 446–447
Onchocercia spp, 265–266
Onchocercia cervicalis, 251–252, 422
Onchocercia gutturosa, 265
Onchocercia reticulata, 265
Onchocerciasis, 265–266
One-stage prothrombin time (OSPT), 198
Oocyte transfer (OT), 307
Oophorectomy, 179, *179*
Open pneumothorax, 129
Ophthalmology, 235–260
  conjunctiva/limbus/episclera/sclera, 246–247
  cornea, 247–252
  differential diagnosis, 259–260
  equipment, 236–239
  examination, 235–239, *237*
  eyelids, 244–246
  fundus, 257–259
  glaucoma, 255
  globe/orbit, 243–244
  imaging, 239
  investigations, 238–239
  lens, 256–257
  nasolacrimal system, 247
  ocular therapeutics, 239–243, *240*
    surgery, 242–243
    systemic medication, 242, *252*
    topical medication, 239–242, *240*, *242*
  optic nerve, 259
  stains, 239
  uveal tract, 252–255
  vitreous, 257
Opioids, 490, 494, *495*, 496
Optic disc shape, 257–258
Optic nerve disorders, 259
  neoplasia, 259
  optic atrophy, 259
  optic nerve syndrome, 217
  optic neuritis, 259
  proliferative, 259
  trauma, 259
Optic nerve (II), 212, *213*
Oral cavity, 8–10
  anatomy, 8, *9*
  dental anatomy, 8–9
  examination, 4, 16, *16–17*
  extraction, 18
  neoplasia, 5
  teeth, eruption, 10
  triadan system, 9–10, *11*
  see also Dental disorders
Oral corticosteroids, 120
Oral electrolyte solutions, 442
Oral iodides, 121, 128
Orbit, 243–244
Orbital fat, prolapse, 244
Orchitis
  bacterial, 302
  traumatic, 302
Organ dysfunction, 427–428
Organophosphorus poisoning, 230
Orthopaedic ultrasonography, 552, *552*
Osmotic fragility test, 198
Osmotic laxatives, colic, 26–27
Osseous cyst-like lesions, *508*, 510
  distal phalanx, 340–341
  fetlock joint, 353
  subchondral bone, 322
Ossifying myopathy, 387
Osteitis, 327
  infectious, radiography, 510
  'pedal', 340
Osteoarthritis (OA), 320–321, *321*
  back, 394
  carpometacarpal bone, 372
  carpus, 370, 536–537
  elbow, 538
  facet joints, 551
  fetlock joint, 353–354, *503*, 531
  foot, 526
  hip, 387
  hock joints, 379–380
  infectious, *500*, 510
  pelvis, 546
  proximal interphalangeal (PIP) joint, 348
  radiography, *500*, 501, *503–506*, 509–510
  shoulder, 539
  stifle, 387, 545
  talocalcaneal, 384
Osteochondral fragments, 532
Osteochondritis dissecans (OCD), 321–322, 353, 372
  hock, 380
  radiography, *505*, *507*
  shoulder, 376–377, *377*, 539
  stifle, 384
  tarsus, *505*, 543
Osteochondroma, 328
  distal radius, 374
Osteochondrosis, 321–322, *322*
  radiography, 501, *507*, 510, 532
Osteodystrophia fibrosa, 192, *192*, 327
Osteomyelitis, 327, 394, *500*, 532
Osteopaenia, 328
Osteophytes, 371–372, 501, *503–505*
Osteophytosis, *321*
Osteoporosis, 328
Otitis externa, 78
Otitis interna, 79–80
Otitis media, 79
Otoscope head, 236
Ovarian neoplasm, 287–288, *287*
Ovariectomy (oophorectomy), 179, *179*
Ovaries, 293
  enlarged, 287–288
  small, 288–289
Overlong feet, 339
Overo lethal white syndrome, 441
Ovulation, 292
  double, 304
Oximes, 230
Oxygen, maximising, 568
Oxygen-carrying fluids, 562–563, *563*
Oxytetracycline, 413–414, 447
Oxytocin, 296–297
Oxytropus spp (locoweed), 217, 465
Oxyuriasis, 266
Oxyuris equi (pinworm), 266, 421

P

Packed cell volume (PCV), 35
Packing artefacts, *514*
Pain
  colic, 25
  gait characteristics, 311
  ocular, 259–260
Palatal defects
  congenital/acquired, 5, 103
  cysts, 5, 103
Palate
  diseases of, 548–549
  elevation, deglutination, 2
Palmar digital nerve block (PDNB), 313, *313*
Palmar fractures, 350
Palpebrae, inflammatory diseases, 245
Palpebral biopsies, 238–239
Palpebral reflex, 237
Pancreatic disease, 54
Pancytopenia, 210
Panophthalmitis, 244
Papillary light reflex (PLR), 237
Papilloma, squamous, 303
Papillomatoses ('warts'), 279
Papular dermatitis, equine, 275
Papules, 262, 279–281
Paracentesis abdominis, 34–35
Parafilaria multipapillosa, 275, 422
Parafilariasis, 275
Paralaryngeal (Whitehouse) approach, 80
Paralytic ileus, *23*
Paramarginal subchondral bone lysis, 372
Paramesonephric cysts, 289
Paranasal sinuses, 431–432
Paranoplocephala mamillana, 420
Paraphimosis, 173–174
Parascaris equorum, 124, 416, 442

Parasite-associated disease, 131, 416–417, 422
  cerebral thromboembolism, 221
  control strategies, 417–418, *417–418*
  cytology/histopathology, 417
  examination, 416
  haematological biochemistry, 416
  history, 416
  keratitis, 251–252
  nodular infections, 275–276
  otitis, 2
  post-mortem, 417
Parathyroid hormone (PTH), 192, 462
Parenteral nutrition, 190, 573
  administration, 573–574
  circumstances necessitating, 573
  types, 573
Paresis, 214, 437–438
Parotid melanomas, 104
Partial arytenoidectomy, 97
Partial obstruction, recurrent colic, 36
Partial parenteral nutrition (PPN), 573
Parturition, 296–297
  endocrinology, 296
  induction, 296–297
  signs, 296
  stages, 296
Parvovirus, 442
Pastern joint disorders, 347–359
  dermatitis (greasy heal), 282
  radiography, 316
  subluxation, 348
  ultrasonography, 317, *318*
  wound, *472*
Pasteurella spp, 125, 146
Patch, 262
Patella
  fractures, 386
  luxation, 386
  upward fixation, 385–386
Patent ductus arteriosus (PDA), 137–138, *137*, 428
Patent foramen ovale, 429
Patent urachus, 165, 446
Patient restraint, 516, *517*
Patterson's Curse (Echium plantagineum), 51–52
Pedal bone, 523, *523*
Pedal osteitis, 340, 525
Pedicle grafts, defined, 481
Pediculosis (lice), 264
Pelodera strongyloides, 266
Pelvis
  flexure, *28*
  fractures, 395–397, *396*, 546–547
Pelvis, radiography, 545–547
  anatomy, 546
  projections, 545
  radiopathology, 546–547
  technique, 546
Pemphigus foliaceus, 270–271, *272*
Penicillin, 45, 279, 323, 442
Penicillin G, *242*, *252*
Penis disorders, 173, 302–303
Penrose drains, *477*
Pentazocine, 26
Pentoxifylline, 333
Performance problems, 117, 119, 458
  causes, *459*
  diagnostic evaluation, 458
  neurological signs, 234
Pergolide, 188
Pergolide mesylate, 184–185
Pericardial disease, 147, 429
Pericardial friction rub, 138
Pericardiocentesis, 147
Periglandular fibrosis, 290
Perinatal asphyxia, 427–428
Perineal disorders
  fistulae, 176–178, *176–177*
  laceration, 176–178, *176–177*, 298
  neoplasms, 178
Perineural analgesia, 312–314
Periocular swelling, 260
Periodontal disease, 12–13, *12*
Periosteal new bone, *500–502*, 501, 532
Peripapillary chorioretinopathy, 259
Peripheral nerve blocks
  lower limb, 312, *313*
  upper limb, 314
Peripheral nervous system, 212, 229–233, 312
  damage, 231–233
  disease, 234
  generalised conditions, 229–231
  neoplasia, 233
  neuropathies, 49–50, 233
  trauma, 437–438
Peripheral perfusion, *33*, 35
Peripheral vestibular disease, 79–80
Peritoneal fluid cytology/biochemistry, 59
Peritonitis, 71–73
  causes, *72*
  colic, 47
Perivascular injections, 496
Permanent tracheostomy, 97
Peroneal nerve
  block, 314
  damage, 231
Peroneus tertius, rupture, 382
Persistent hymen, 292
Persistent oestrus, 304
Persistent pupillary membranes, 253
Persistent 'winter coat', 282
Petechia, 210
Pethidine, 26
Petroleum products, 467
$PGF_{2\alpha}$, 303
Phaeochromocytoma, 185–186
Phaeohyphomycosis, 276
Phalangeal periostitis, 347–348
Phalanges, stabilization, 324–325, *325*
Pharynx, 432
Pharyngeal conditions
  collapse, 102
  compression, 5
  constriction, deglutination, 2
  cysts, 5, 103
  diseases, 548–549
  lymphoid hyperplasia (PLH), 103
  neoplasia, 103
  paralysis, *3*, 5, 103
Phenobarbital, 435
Phenobarbitone, 435
Phenothiazine, 467, 490
Phenothiazine-derivative tranquilizers, 173
Phenylbutazone, 25, *252*, 343, 494
Phenylephrine HCl, *240*
Phenytoin, 435
Phimosis, 174
Phosphorus metabolism, 463
Photoelectric effect, 500
Photoperiod, artificial control, 304
Photosensitization, 50, 274, 466–467
Phthisis bulbi, 243
Phycomycosis, 276–277
Physeal fusion, *509*
Physitis, 327, 537
  septic, 577–578
Picornaviruses, equine, 404
Pigmentation disorders
  congenital, 281–282
  retinopathies, 258
Pilocarpine, 255
Pinch grafts, 486
Pinworm, 266, 421
Pirbuterol, *120*
Piroplasmosis *see* Babesiosis, equine
Pituitary pars intermedia dysfunction (PPID) (equine Cushing's disease (ECD)), 167, 182–185, *183*, 188, 282
Placenta, 293
Placental insufficiency, 295
Plant poisons, 217–218, 228, 462, 465–467
Plantar metacarpal pain, 360–361
Plantar process fractures, 350
Plaques, 262
Plasma, 568
  alternatives, 572
Plasma cell myeloma, 209
Plasma transfusions, 571–572
  administration/monitoring, 572
  indications, 571
  plasma/serum products, 571–572
Plaster of Paris, 13
Platelets, 197–198
Pleural space disorders, 550, 581
  effusion, 116, *116–117*
  metastases, 124–125
  transudates, 130
  tumours, primary, 124
Pleuritis, 116, *116*, *118*
Pleuropneumonia, *118*, 125–126
Pneumocystis pneumonia, 128
Pneumonia
  acute interstitial, 128
  chronic interstitial, 127, *127*
  fungal, 128, *128*
  interstitial, 433
  pleuropneumonia, *118*, 125–126
  Pneumocystis, 128
  Rhodococcus equi, 126–127, *126–127*
Pneumothorax, 117, 129–130, *130*
Pneumovagina ('wind-sucking'), 6–7, 104, 291
Pododermatitis, 334–335, *335*
Poikilocyte, 197
Point of maximal intensity (PMI), *135*, 138
Poisons
  feed-associated, 468
  organophosphorus, 230
  plant, 217–218, 228, 462, 465–467
  see also Toxic conditions

Polychromasia, 197
Polycystic kidney disease, 166
Polycythaemia, 206–207
Polydypsia, 183
Polymixin B, 45, *240*
Polyneuritis equi (cauda equina neuritis), 232–233, *232*
Polyps, sinu-nasal, 90
Polysaccharide storage myopathy (PSSM), 456–457
Polysynovitis, 126
Polyuria, 183
  /polydypsia (PU/PD), 167–168, *167*
Polyvalent antitoxin, 438
Ponazuril, 227
Portacaval shunts, 445
Portosystemic shunts, 445
Postanaesthetic myoneuropathy, 453–454, *453–454*
Posterior segment, examination, 238
Postmaturity
  clinical characteristics, 424
  defined, 424
Postoperative colic, 46
Postoperative diarrhoea, 47
Postoperative ileus (POI), 45–46
  categories, 46
Postpartum period, 297–298
Posture, examination, 213–214
Potassium, *252*
  derangements, 565
  metabolism, 463
Potassium chloride, 333
Potassium EDTA, *240*
Potomac horse fever, 413
Poultry mites, 265
Povidone iodine (PI), 476
Praziquantel, 418
Prednisolone
  atopic dermatitis, 267
  hypoadrenocorticism, 185
  insect hypersensitivity, 267
  ocular disease, *252*
  pemphigus foliaceus, 271
  purpura haemorrhagica, 282
  recurrent airway obstruction (RAO), 120
  strongyles, 419
Prednisolone acetate, *240*
Pregnancy, 292–296
  complications, 294–295
  diagnosis, 293
  elective termination, 296
  endocrinology, 293–294
  losses, *305*
  reproductive tract, changes, 293
Prehension, deglutination, 2
Premature complexes, cardiac arrhythmias, *136*
Prematurity, 424–425, *424–425*
  clinical characteristics, 424
  defined, 424
Prepubic tendon, rupture, 70, 295
Prepuce disorders, 173, 302–303
Prerenal azotaemia, 161
Presuturing, 478
Priapism, 174
Primary closure (first intention healing), 474–478
  anaesthesia, 476
  criteria, 474–475, *475*
  debridement, lavage, 475–476
  defined, 474
  delayed (third intention healing), 478–479
  drains, 476–478
  options, 476
  skin tension, 478
  surgical debridement, 475
  wound preparation, 475
Primary conditions
  corneal oedema, 248
  hyperparathyroidism, 192
  lung tumours, 124
  pleural tumours, 124
  renal disease, 447
Probstymaria vivipara, 422
Procainamide, 155
Procaine penicillin, *252*
Progesterone, 293–294
Progressive ethmoidal haematoma (PEH), 88–89, *89*
Prohormone pro-opiomelanocortin (POMC), 183
Proliferative enteropathy, 413–414
Proliferative phase, wound healing, 474
Prosthetic laryngoplasty, 97
Protein losing enteropathy, 58
Protein-bound calcium, 462
Prothrombin time (PT), 198
'Proud flesh', 277
Provocative tests, lameness, 311
Proximal annular ligament, 351
Proximal enteritis, 30–32
Proximal interphalangeal joint (PIP), 314, 348
Proximal jejunitis, 410–411
Proximal jejunum, *28*
Proximal metacarpal syndrome, 537
Proximal palmar metacarpus, lameness, 368
Proximal palmar pain, 360–361
Proximal phalanx, fractures, 348–350
Proximal radius, stabilization, 325
Proximal sesamoid bone, fractures, 354–355, *355*, 531
Proximal suspensory desmitis, 365–366
Proximate Linear Cutter, *41*
Prunus spp, 466
Pruritus (itch), 263–268
Pseudallescheria boydii, 276
Pseudohermaphroditism, male, 301
Pseudohyperparathyroidism, 192
Pseudomonas spp, 114, 248–250, 482
  wound healing, 474, 480
Pseudomonas aerogenes, 300
Pseudomonas mallei, 415
Pseudopregnancy, 304
Pseudotumour, conjunctival, 247
Psoroptes spp, 265
Psyllium hydrophilic mucilloid, colic, 26
Pteridium aquilinum, 466
Pulley sutures, 478
Pulmonary conditions
  acute obstructive disease, 121–122
  artery ruptures, 158
  congestion, 129
  differential diagnosis, 106
  exercise-induced haemorrhage (EIPH), 123–124, *123*
  hypersensitivity, 117–118
  hypertension, 429
  mass, 117
  neoplasia, 124–125
  oedema, 128–129, *129*
  pulmonic insufficiency (PI), 146
  summer pasture-associated obstructive disease (SPAOPD), *118*, 122
Pulse character, 33
Punch grafting, 485–486, *486*
Purpura haemorrhagica, *199–200*, 205–206, *205*, 282
Puss, foot, 525, *525*
Pustule, 262
Pyelonephritis, 162, 165–166
  antimicrobial therapy, 163
  chronic, 163
Pyometra, 290
Pyrantel, 418
Pyrethroids, 264
Pyrimethamine (Daraprim), 227
Pyrrolizidine, 51–52, *52*
Pyrrolizidine alkaloids, 465
  toxicosis, 445
Pythiosis, 276–277, 416
Pythium spp, 276
Pythium insidiosum, 276, 416, 473

Q

Quercus spp, 466
Quinapril, 149
Quinidine, 152–153
Quinidine gluconate, 155
Quittor, 338, *338*

R

Rabies, 220
Radial immunodiffusion (RID) techniques, 199
Radial nerve paralysis, 231
Radiography, 391, 499–552
  advantages/disadvantages, 516
  assessment, 504–509, *509*
  cassette labelling, 519
  exposure settings, 518–519, *518*
  focus-film distance, 519
  indications, 502
  interpretation, 502–509
  musculoskeletal problems, 509–516
  patient preparation, 516, *517*
  physics, 499–502
  projections, 516–518, *517*
  report form, *509*
  scatter reduction, 519
  viewing equipment, 504
  see also specific anatomical regions
Radiolucent lines, 515, *515*
Ragwort (*Senecio jacobea*), 51–52
Rales, 113

Ramipril, 149
Ranitidine, 443
Rattlebox (*Crotalaria* spp), 51–52, 217
Re-breathing bag, 112–113, *113*
Rectovaginal fistula, 298
Rectovestibular fistulae, 177, *177*
Rectovestibular lacerations, *176*, 177
  postoperative care, 177–178
Rectum, *28*
  examination, 34, 168
Recurrent airway obstruction (RAO), 117–121, *118*, *120*
  aetiology, 117–121
  clinical signs, 119
  diagnosis, 119–120
  epidemiology, 119
  pathogenesis, 118
  pathological changes, *119*
  treatment, 120–121, *120*
Recurrent laryngeal neuropathy (RLN), 95–97, *96*
  aetiopathogenesis, 95
  clinical signs, 95–96
  endoscopy, 96, *96–97*
  'grunt-to-the-stick' test, 96
  palpation, 96
  prevalence, 95
  prognosis, 97
  treatment, 97
Red cell distribution width (RDW), 196
Red maple leaf, toxicity, *199–200*, 466
Reflex testing, 434
Relative polycythaemia, *199–200*
Relaxin, 294
Relaxing incisions, 478
Remodeling phase, wound healing, 474
Renal function, 159–160
Renal disease
  biopsy, 160
  neoplasia, 166
  primary, 447
  renal failure, 161, 462
    acute (ARF), 162
    chronic (CRF), 162–163
  secondary hyperparathyroidism, 192
  tubular necrosis (RTN), 162
Repair (fibroblastic/proliferative) phase, wound healing, 474
Reproduction, 283–308
  differential diagnosis, 307–308
  dystocia, 297
  management, 303–307
  non-pregnant mare, 283–292
  postpartum period, 297–298
  stallion, 298–303
  see also Parturition; Pregnancy
Reproductive tract, female, 176
  changes, pregnancy, 293
  diseases, 287–292
  dystocia, 178
  neoplasia, 178, *178*
  ovariectomy (oophorectomy), 179, *179*
  perineal lacerations/fistulae, 176–178, *176–177*
Reproductive tract, male, 300
Repulsion, 19
Respiratory system
  endoscopy, 430
  examination, 429–430, 575
  locomotion and, 92
  radiography, 429–430, 549
  respiratory rate/pattern, 112, *112*, 429–430
  ultrasonography, 430
Respiratory system disorders, 429–433
  acidosis, 464–465
  alkalosis, 465
  differential diagnoses, 131–132
  equine herpesvirus 1 (EHV1), 402–403
  exercise, 93
  foals, 577
  respiratory distress syndrome (RDS), 430–431
  respiratory noise, differential diagnosis, 105
  see also Acute respiratory distress
Respiratory tract *see* Airway obstruction; Lower respiratory tract (LRT); Upper respiratory tract (URT)
Restraint, 236
Resuscitation, 427
Reticulated leucotrichia, 281
Retinal disorders
  atrophy, 258
  blood vessels, 257–258
  degenerative retinopathies, 258–259
  detachment, 258
  haemorrhage, 258
  pigmentation, 258
Retinal pigmented epithelium (RPE), 258
Rhinitis
  mycotic, 89–90
  viruses, 404
Rhinopneumonitis, 402–403, 432
Rhizopus spp, 128
Rhodococcus equi, 236, 278–279, 437, 441, 443
  pneumonia, 126–127, *126–127*
Rhododendron spp, 466
Rhododendrons, 466
Richter's hernia, 68
Ricinus communis, 465
Rickets, 328
  anaemia, 203
Riddleneck (*Amsinckia* spp), 51–52
'Ridglings' *see* Cryptorchidism
Rifampin, 126–127, 414
'Rigs' *see* Cryptorchidism
'Ringbone', 347–348
Ringworm (dermatophytosis), 269–270, 416
Rodenticides, 467
Romifidine, 26, 490–492
Ropivacaine, *495*, 496
Rose Bengal, 239
Rotavirus spp, 407–408, 441
Round ligament rupture, 546
R-V lacerations *see* Rectovestibular lacerations
Rye grass staggers, 228, 466

S

Sacral spine, ligaments, *390*
Sacroiliac joint disease, 394
Sagittal fractures, 349
St Johnswort, photosensitization, 467
Salinomycin, 457
Salivary gland disorders, 104
Salmeterol, *120*
Salmonella spp, 31, 408–410, 437, 439–441
Salmonella agona, salmonellosis, 409
Salmonella anatum, salmonellosis, 409
Salmonella enterica, 408–409
Salmonella enteritidis, 409–410
Salmonella infantis, 409
Salmonella krefeld, 409
Salmonella newport, 409
Salmonella saint-paul, 409
Salmonella typhimurium, 409–410
Salmonellosis, 408–410, *409*
Sand enteropathy, 62
Sarcocystis neurona, 422
Sarcoidosis, equine, 273
Sarcoids, 277–278
  external ear, 2
  sarcoid tissue, *473*
Sarcoptes, 265
Sarcoptes scabiei, 265
'Satellite lesions', 249, *249*
0-10 scale, lameness, 311
Scaling/crusting (skin disorder), 262, 268–274
Scapulohumeral dysplasia, 377
Scar, 262
Schirmer tear test (STT), 238
Sciatic nerve, damage, 231
Scintigraphy, 391, 554–556
  advantages/disadvantages, 556
  detection, 554
  indications, *513*, 554–556
  interpretation, 555–556, *555*
  principles, 554
  procedure, 555
  radiopharmaceutical doses, 554
Scirrhous cord, 170, *170*
Sclera, 246–247
Sclerosis, *500*, 501, *505–507*, 510
  third carpal bone, 536, *536*
Scrotum
  acquired abnormalities, 300–303
  congenital abnormalities, 301
  development, defective, 308
  enlarged, 308
  lacerations, 301
Seasonal anoestrus, 285
Seborrhoea, 273
Secondary bacterial folliculitis, *271*
Sedation
  adult horses, 490–491
  colic, 26
  drugs, 490
  foals, 491
Seedy toe, 330
Seizures
  foals, 435
  idiopathic, 216–217
Selective IgM deficiency, 450
Selenium, 148, 467
Semen
  altered, 303
  collection, 298–299, 305–306
  cooled, 306
  frozen, 306

poor quality, 308
treatment, 305–306
Senecio spp (groundsel), 51–52, 217
Senecio jacobea (ragwort), 51–52
Senile retinopathy, 258
Septic conditions
foals, 425–426
hip, 387
meningitis, 435
neonatal foal, 576–577
physitis, 577–578
renal disease, 447
tenosynovitis, digital flexor tendon sheath (DFTS), 357–358
Septic arthritis, 323, 376
distal interphalangeal (DIP) joint, 346
fetlock, 532
foals, 439, 577–578
Septicaemia, 445
Sequestrum, *501*, 510
formation, fetlock, *501*, 532
Serology, 400
Serum hepatitis, 52
Serum insulin concentrations, 186
Serum products, 571–572
Serum sickness, 52
Sesamoiditis, 354, 531, *531*
Setaria spp, 221
Severe combined immunodeficiency (SCID), *199–200*, 207, 449
Sevoflurane, 493
'Shaker foal syndrome', 229, 438
'Sheared heels', 339
Shearing forces, 482
Sheet grafts, 481–485
techniques, 484–485, *485*
Shivering, 233
'shaker foal syndrome', 229, 438
Shock, 565–568
classification, 426–427, 566–567
colic, 45
defined, 565–566
foals, 426–427
management, 567–568
monitoring, 568
pathophysiology, 566
rate, intravenous fluids, 563
stages, 567
Shoulder, 376–378
analgesia, 315
anatomy, 376
fractures, 539
Shoulder, radiography, 316, 539–540
anatomy, 539
radiopathology, 539–540
technique, *508*, 539
'Sidebone', 341
Silicon dressings, 481
Silver sulfadiazine, *240*
Simulium spp, 266
Sinoatrial (SA) block, 150–151
Sinu-nasal disorders, 84–85, 90–91
anatomy, 84
biopsy, 85
differential diagnosis, 106
empyema, primary/secondary, *86*, 87–88, *88*
endoscopy, 84–85
facial flap surgery, 86–87, *86*
history, 84
imaging, 85, *85*
medical management, 86
mycotic rhinitis/sinusitis, 89–90
neoplasia/polyps, 90
physical examination, 84
presenting signs, 84
treatment objectives, 86
trephination, 86
Sinus disorders
arrest, 150–151
arrhythmia, 150
bradycardia, 150
cysts, 89
tachycardia, 150
Sinusitis, *506*, 548
mycotic, 89–90
Skin
expanders, 478
undermining, 478
Skin diseases, 261–282
alopecia/scaling/crusting, 268–274
congenital/developmental, 281–282
depigmentation, 281
diagnostic approach, 261–263
differential diagnosis, 262
examination, 262
nodular, 275–281
pruritis (itch), 263–268
tests, 262–263
ulceration/crusting, 274–275
Skin grafting, 481–487
acceptance physiology, 482
acceptance/cosmesis, 485
allografts/xenografts, 487
classification, 481–482
donor site, 482, 487
failure, causes, 482
graft storage, 487
indications, 481
island grafting, 485–487
recipient site, 487
sheet grafts, 482–485
wound preparation, 482
Slab fractures, 371, 511
carpus, 511, *512*, 535
Slap test (laryngeal adductor response), 213, *213*, 225
Slit lamp biomicroscopy, 236
Small airway inflammatory disease (SAID), 122–123
Small airway obstruction, 118
Small intestine (colon), 42–43
examination, 38
gut lumen, occlusion, *36*
impaction colic, *28*
resection, 40–41, *40*
simple obstruction, 22–23, *23*
strangulating obstruction, *23*, 24–25
volvulus, 444
Smoke inhalation, 130–131
Snake bite, 218
Sodium, *252*
derangements, 564
metabolism, 463
vascular disease, 333
Sodium cromoglycate, 121
Sodium thiosulphate, 467
Soft palate
midline cleft, *4*
role, 92–93
see also Dorsal displacement of soft palate (DDSP)
Soft-tissue
back, 392
calcification, 515, 540, *540*
carpus, 371, 374–375
fetlock, *531*, 532–533, *533*
stifle, 545
X-rays, 501–502, *507–508*
Solanum spp, 466
Solar dermis, puncture, 336–337
Sole, penetrating wounds, 336–337
'Sore shins', 360
Sorghum grass ataxia, 228
Sorghum spp, 466
Spasmodic colic, 27–28
Spasticity, 214, 234
Sperm count, low, 307
Spermatic cord, torsion, 173
Spherocyte, 197
Spinal abscess, 227
Spinal cord, 222–229
ataxia, 222–225, *223*, *224*
compression, 550–551
diseases, neurological signs, 234
equine degenerative myeloencephalopathy (EDM), 228
equine motor neuron disease (EMND), 228–229
inflammatory conditions, 227
neoplasia, 228
toxic conditions, 228
trauma, 226–227, 437
vertebral malformations, 225–226
see also Back
Splint bones, fractures, 361–363
Splints, 361, *362*, 537, *538*
Split oestrus, 304
Split pastern, 349
Split-thickness grafts
defined, 481
sheet, 483–484, *483*
Spondylosis, 551
deformans, 393
Sporadic lymphangitis, 209
Sporothrix schenkii, 276, 416
Sporotrichosis, 276, 416
Sposmolytics, 26
Spotted leucotrichia, 281
Squamous cell carcinoma (SCC), 74, 245, *245*, 278
Squamous papilloma, 303
Stabilization, emergency, 324–325
Staphylectomy, 98–99
Staphylococcus spp, 125, 248–249, 276, 278–279
bacterial pyoderma, 270, 279
Staphylococcus aureus, 209, 270
Staphylococcus hyicus, bacterial pyoderma, 270
Staphylococcus intermedius, bacterial pyoderma, 270
Stapling techniques, automated mechanical, 41

Stationary night blindness, congenital, 258
Steatitis (yellow fat disease), 450
Stenosis
  acquired, 247
  oesophageal, 441
  tracheal, *118*
  vertebral canal, 551
Stent bandage, 478
Sterile nodular papular skin diseases, 279–281
Sterile ulcerative/exudative nodular skin diseases, 277
Sterno-thyro-hyoideus myectomy, 99
Steroids, 395
Steroid let down syndrome, 185
Stifle, 384–387
  anatomy, 384
  fractures, 545
Stifle, radiography, 316, 543–545
  anatomy, 544
  projections, 544
  radiopathology, 544–545
  technique, *507*, *515*, 544
Stomach
  diseases, 56–58
  impaction colic, *28*
  neoplasia, *74*
Stomoxys calcitrans, 266
Strabismus, 243–244
Strangles, 411–413, *411*
  abscessation, 5
  'bastard (false)', 278, 411
  gutteral pouch, 81
Strangulated hernia, 67
Straw dust, 117–118
'Street Nail' procedure, 337
Streptococcus spp
  abscesess/cellulitis, 278
  bacterial pyoderma, 279
  breeding soundness examination (BSE), 299–300
  endocarditis, 146
  immune-mediated haemolytic anaemia, 201
  keratomalacia, 250
  pleuropneumonia, 125
  ulcerative keratitis, 248–249
  ulcerative lymphangitis, 279
  vertebral osteomyelitis, 437
  wound healing, 475
Streptococcus equi, 191, 220, 236, 278, 443
  strangles, 81, 411–413
Streptococcus zooepidemicus, 443
Stride, phases of, 310–311
Stringhalt, 233
Strongyles, 418–420, *419*
Strongyloides westeri, 266, 421, 442
Strongylus edentatus, 418
Strongylus equinus, 418
Strongylus vulgaris, 158, 221, 397, 418
Strychnine, 467
Stumbling, 234
Styrofoam™ pad, *334*
Subcarpal block, 314
Subchondral bone
  cysts, *508*, 510
  densification, 372
  injury, 352
  lysis, paramarginal, 372
Subchondral cystic (cyst-like) lesions (SCLs), 322
  bone, *508*, 510
  carpus, 372, *375*, 537
  elbow, 375–376, *375*, 538
  shoulder, 377
  stifle, 384–385, *385*, *508*, 545
Subconjunctival injection, 242, *242*
Subconjunctival micro-osmotic pumps, 242
Subcutaneous emphysema, 91
Subcutaneous fluids, 563
Subepidermal bullous diseases, 274
Sub-epiglottal cysts, 5–6, *6*, 100
Subluxations
  lens, 257
  radiography, 510, *511*
  tarsal, *511*, 543
Sub-palpebral lavage, 241, *241*
Subsolar abscess, *335*
Sub-total arytenoidectomy, 97
Sucralfate, 443
Sudan grass, 466
Summer pasture-associated obstructive pulmonary disease (SPAOPD), *118*, 122
'Summer sores', 174–175, *175*, 275, 473
Superficial digital flexor tendon
  displacement, 381
  spontaneous rupture, 364
  tendinitis, 363–364, *363*
Suppurative meningitis, 435
Supracondylar lysis, *532*
Supraglenoid tuberosity (tuber scapulae), 377
Suprascapular neuropathy ('sweeney'), 378
Suprascapular paralysis, 231
Supraventricular arrhythmias, 429
Surgical arthrodesis, 379
Surgical debridement, 475
Surra, 422
Suspensory apparatus, breakdown, 355–356
Suspensory ligament (SL)
  desensitization, 313–314
  desmitis, 365–367, *366*
Sutures
  mattress tension, 478
  patterns, tension, 478
  wound closure, 476
Swainsonia spp (darling pea), 217, 465
'Swamp cancer', 276–277
Sway response, *215*
'Sweeney', 378
Sweet pea, 466
Swelling
  back, 391
  fetlock, 368
  nodular skin diseases, 278–279
  scrotum, 300, *300–301*
  soft-tissue, 371
  tarsal sheath, *380*
Synchronous diaphragmatic flutter, 455–456
Synchysis scintillans, 257
Synovial cyst, 226
Synovial distensions, tarsus, 543
Synovial fluid analysis, 315–316
Synovial pressure test, 470, *471*
Synovitis
  idiopathic, 319
  traumatic, 319–320
Synthetic colloids, 572
Systemic disorders
  amyloidosis, 281
  corticosteroids, 120
  multiple body systems, 425–428
  mycoses, 277
  neurological signs, 234
  poor performance, *459*
Systolic heart sounds, 135
  clicks, 138
  ejection murmur, 137

T

Tabanid spp, 266
Tachypnoea, idiopathic, 431
Tail, examination, 214
Talocalcaneal osteoarthritis, 384
Tapetal colour, 257
Tapeworms, 420
Tarsal bone, collapse, 382–383, 543
Tarsocrural joint, analgesia, 315
Tarsometatarsal joint, 315
Tarsorrhaphy, 242–243
Tarsus
  fractures, *512*, 543
  radiographic anatomy, 542–543
  slab fracture, 511, *512*
Tarsus, radiography, 540–543
  anatomy, *540–542*, 542–543
  projections, 540–541, *540–542*
  radiopathology, 543
  technique, 534, 541–542
Taxus spp, 466
Taylorella equigenitalis, 285, 300
Teasing programmes, 305
Teeth *see* Dental disorders
Temperamental difficulties, 392
Temporal teratoma, 78–79
Temporary transphyseal bridging, 374
Temporohyoid osteoarthropathy (THO), 79, 222
Temporo-mandibular joint (TMJ) disorders, 4–5
Tendinitis
  deep digital flexor tendon (DDFT), 344–345, *344*, 364–365, *365*
  superficial digital flexor tendon, 363–364, *363*
Tendons
  lacerations, 365
  ruptures, 439
Tenosynovitis
  digital flexor tendon sheath (DFTS), 357–358
  tarsal sheath, 543
Tension
  pneumothorax, 129
  suture patterns, 478
Testes
  diseases, 168, 301–302
  enlarged, 308
  small, 302, 308
  testicular feminization, 301
  testicular size/consistency, 299

Tetanus ('lockjaw'), 229–230
anti-toxin, *240*
Tetralogy of Fallot, 428
Theileria equi, 201, 421–422
Theiler's disease, 52
Thelazia lacrymalis, 422
Theophylline, 121
Theraflex™ pad, *334*
Thermography, 392
Thermoregulatory problems, 458
pathophysiology, 458
Thiamine, 435
Thiopental, 492
Third carpal bone, sclerosis, 536, *536*
Thoracocentesis, 117, *117*, 198, 430
Thoracolumbar spine, ligaments, *390*
Thoracotomy, 126
Thorax
auscultation, 429
penetrating wounds, 470, *471*
percussion, *113*
Thorax, radiography, 116, *116*, 502, 549–550
evaluation, 549
projections, 549
radiopathology, 549–550
technique, 549
Thoroughpin, 380–381, *380*
Thread worm, 266
Throat conditions, 104–105
radiography, 93
Thrombocytopaenia, 210
immune-mediated (IMTP), *199*, *199–200*, 204, *204*, 450
Thrush, 336
Thyroid
adenoma, 191–192
neoplasia, 104
Thyroid-stimulating hormone (TSH) (thyrotropin), 182, 190
Thyrotropin-releasing hormone (TRH), 182, 190
stimulation test, 184
Tibial nerve
block, 314
injury, 231
Ticarcillin, *242*
'Tie forward' (laryngeal advancement), 99
Tiletamine-zolazepam (TZ), 492
Tiludronate, 340, 343, 353–354, 395
Timolol, 255
Tissue
adhesives, 476
swabbing/scraping, 238
Tobramycin, *240*, *242*
Toe-dragging, 234
Togaviral equine encephalitides, 219–220
Togavirus spp, 219–220
Topometry, 239
Total arytenoidectomy, 97
Total iron binding capacity (TIBC), 198
Total parenteral nutrition (TPN), 573
Total protein (TP), 35
Toxic conditions
brain, 217–219
hepatobiliary diseases, 445
metritis, 298
neurological signs, 234
plants causing, 217–218, 228, 462, 465–467
treatment, 465
Trachea, 432
aspiration, 114–115, *115*
collapse, congenital, 103
stenosis, *118*
Tracheobronchial foreign body, *118*
Tracheostomy, permanent, 97
Tracheotomy intubation, 97, 103–104
Tramadol, 494, *495*
Trans tracheal aspirate, 430
Transient hypogammaglobulinaemia, 208, 450
Transport
emergency, 325
surgical centre, 37
Transudates, 117
Transverse colon, *28*
Trauma
arthritis, 319–320, 370
brachial plexus, 437–438
brain, 217
brain injury, 217
conjunctiva, 246
distal interphalangeal (DIP) joint, 346
external nares, 83
eyelids, 244
facial/sinus, 91
fetlock, *507*, 532
gutteral pouch, 83
iris, 253
keratitis, 248, *249*
optic nerve, 259
orchitis, 302
peripheral nerve, 437–438
scrotum, 300–301
shoulder luxation, 378
spinal cord, 226–227, 437
synovitis, 319–320
vertebral, 437
Tremors, 234
Trephination, sinu-nasal disorders, 86
Triadan system, 9–10, *11*
Triamcinolone, 267, 271
Triamcinolone acetate, *242*
Triamcinolone acetonide, 120, *120*
Trichogram, 263, 271–272, *272–273*
Trichomonas spp, 422
Trichophyton equinum, 269, 416
Trichophyton mentagrophytes, 269
Trichostrongylus axei, 422
Tricuspid atresia, 428
Tricuspid regurgitation (TR), 145–146
Trifolium spp, 467
Trigeminal nerve (V), 212, *213*
Triglyceride (TG), 189
Trilostane, 185
Trimethoprim sulphur, 270
Trimethoprim-sulphonamide, *252*
Triodontophorus spp, 418
Trochlear nerve (IV), 212, *213*
Trombicula spp, 264
Trombiculidiasis, 264
Tropicamide, *240*
Truncus arteriosus, 429
Trunk, examination, 214
Trypanosoma brucei, 421–422
Trypanosoma brucei brucei, 421
Trypanosoma brucei gambiense, 421
Trypanosoma brucei rhodesiense, 421
Trypanosoma congolense, 421
Trypanosoma equiperdum, 421–422
Trypanosoma evansi, 421–422
Trypanosoma godfreyi, 421
Trypanosoma simiae, 421
Trypanosoma vivax, 421
Trypanosominasis, 421–422
Trypanozoon spp, 421
Tuber scapulae (supraglenoid tuberosity), 377
Tuberculosis, 415
Tubular genital tract, neoplasms, 178, *178*
Tumours
alimentary system, 15
calcinosis, tumoral, 328, 387, 545
granulosa cell, 287, *287*
jaw, 15
lung, 124
mediastinal, 125
odontogenic, 15
peripheral nerve sheath, 233
pleural, 124
testis, 302
Tunnel grafting, 486–487
Turn-out syndrome, 185
'Tushes', 8
Twinning, 294
Tyzzer's disease, 217, 415, 444–445

U

Udder development, premature, 295
Ulceration, 262, 274–275
gastric, 56–57, *57*, 577
gastroduodenal, *36*, 443
ulcerative keratitis, 248–250, *249*
ulcerative lymphangitis, 279
ulcerative nodular skin diseases, 275–278
Ulnar fractures, 375
Ulnar nerve block, 314
Ultrasonography, 391–392, 552–554
advantages/disadvantages, 554
common artefacts, 553–554
indications, 552–554
parameters, 553
physics, 552
procedure, 553–554
terminology, 553
tissue characteristics, 553
Umbilical structures, examination, 575
Umbilical cord abnormalities, 295
remnant infections, 446–447
Umbilical hernias, 67–69
closed reduction, 68
examination, 68
open reduction, 68–69, *69*
Ungular cartilage necrosis, 338, *338*
Unilateral blindness, 260
Unilateral purulent nasal discharge, 131

Upper alimentary system, 1–20
dysphagia *see* Dysphagia
function *see* Deglutination
oesophageal obstruction, *3*, 7–8
oral trauma, 7
tumours, 15
see also Oral cavity
Upper limb, peripheral nerve blocks, 314
Upper motor neuron (UMN) damage, 222
Upper respiratory tract (URT)
acute respiratory distress, 578
disease, 431–432
emergency treatment, 579, *580*
examination, 578
investigations, 582
obstruction disorders, 95
ultrasonography, 94
Urachus, patent/persistent, 165, 446
Ureterolithiasis, 164
Urethral calculi, 164
Urethral rents, 165–166, *166*, *175*
Urethritis, 165–166
Urinalysis, 198
Urinary system, 159–179
acute renal failure (ARF), 162
castration, 169–170
chronic renal failure (CRF), 162–163
congenital monorchidism, 170
cryptorchidism, 170–171
cutaneous habronemiasis ('summer sore'), 174–175, *175*, 275, 473
cystitis, 163
evaluation, 159–161, *160*
haematuria, 165–166
haemospermia, 175–176, *175*
inguinal herniation/rupture, 171–173, *172*
paraphimosis, 173–174
patent/persistent urachus, 165
penis/prepuce, disorders, 173–174
phimosis, 174
polyuria/polydypsia (PU/PD), 167–168, *167*
prerenal azotaemia, 161
priapism, 174
spermatic cord, torsion, 173
testis, diseases of, 168
urolithiasis, 164, *164*
uroperitoneum, 164–165
Urogenital system diseases, 446–447
congenital defects, 446
examination, 446, 575
urogenital tract neoplasia, 75
Urolithiasis, 164–166, *164*
Uroperitoneum, 164–165
Urovagina, 291–292
Urticaria, 279–280, *280*
Uterus, 289–290, 293
Uterine disorders
abscess, 290
neoplasia, 290
prolapse, 298
rupture, 298
torsion, 295
uterine body pregnancy, 295
uterine tubes, 289
Uveal tract, 252–255
Uveitis *see* Iridocyclitis

V

Vagina, 291–292
artificial, 305, *305*
examination, 286, *286*
Vagus nerve (X), 213, *213*
Valve diastema, 12–13
Valvular regurgitation, *137*
Varicocoele, 300, *301*
Varicose veins, vaginal, 286, *286*
Vascular channels, 511, *514*
Vascular disorders, 156–158
anomalies, 166
corneal vascularization, 254, *254*
foals, 435
peripheral, 333
ruptures, 158
Vascular theory, navicular disease, 342
Vasodilators, 149
Vasopressin, 182, 583–584
Venereal diseases
code of practice, 300
pathogens, 299–300, *299*
Venezuelan equine encephalitis (VEE), 219
Ventilation, 582
Ventral hernias, 69
Ventral midline dermatitis, equine, 266
Ventral ruptures, 295
Ventricular arrhythmias, 153–155, *154*, 429
Ventricular knocks, 138
Ventricular rhythms, *136*
Ventricular septal defect (VSD), *137*, 142–143, 428
Ventriculo-cordectomy, 97
Ventro-axial luxation of corniculate process of arytenoid cartilage (VALCPA), 102–103
Verminous encephalitis, 221
Verminous nephritis, 165–166
Vernal transition, 285, 304
Vertebral disorders
canal, stenosis, 551
column deformities, 392
osteomyelitis, 437
spinal cord malformations, 225–226
trauma, 437
Vertical mattress tension sutures, 478
Very-low density lipoprotein (VLDL), 189
Vesicle, 262
Vesicular glands, 302
Vesicular disorders
neoplasia, 166
stomatitis (VSV), 275, 406
Vestibular disorders, 231–232
peripheral, 79–80
vestibular syndrome, 217
Vestibulocochlear nerve (VIII), 213, *213*
Vetches, photosensitization, 467
Viborg's Triangle, 80
Vicia spp, 467
Villonodular synovitis, 351
Vinegar (acetic acid), 54
Viral infections, 131, 274–275, 408
abortion, 294
antigens, detection, 400
arteritis *see* Equine viral arteritis (EVA)
diagnosis, 400
encephalitides, 219–220
encephalitis, 435
foals, 432
keratitis, 251
Virus isolation, 400
Visual dysfunction, 436
Vital parameters, neonatal foal, 574
Vitamin D deficiency, 328
Vitamin E, 148
Vitamin K deficiency, 206
Vitiligo, 281
Vitreous, 257
Volvulus
large intestine, 444
small intestine, 444
Vulva, 292
bleeding, *286*, 295, 308
discharge, 308

W

Walking sutures, 478
Warfarin, 467
'Warts', 279
Water balance, 159–160
Water dropwort, 465
Water hemlock, 465
West Nile virus (WNV), 406–407
Western equine encephalitis (WEE), 219
Wheals ('hives'), 262
Wheezes (rhonchi), 113
White snakeroot, 466
Whitehouse (paralaryngeal) approach, 80
modified, 80–81
Whole blood, 568
collection, 569–571, *570*
transfusions, 569
'Wind-sucking', 6–7, 104, 291
'Wobbler' syndrome, 321–322
Wolf teeth, extractions, 18
Wolff's law, 501
Wounds, 469–487
assessment, 470–473, *471*
classification, 469–470
closed, 469–470
complications, colic, 46
contraction, 479–480
defined, 469
expansion, 479
open, 469–470
radiography, 515–516, *515–516*
see also Skin grafting; Wound healing
Wound healing, 473–474
first intention *see* Primary closure
second intention (delayed), 183, 479–481
third intention, 478–479
debridement phase, 474
impairing factors, 470–473, *471–473*
inflammatory phase, 473–474
management methods, 474
maturation (remodeling) phase, 474

repair (fibroblastic/proliferative) phase, 474
see also Skin grafting
'Wry nose', 83

X

Xenografts, 487
defined, 481
X-rays
bone, changes, 501
detection, 500
image parameters, 500–501
interaction, matter, 500
production, 499–500
soft tissues, 501–502, *507–508*
uses, 501
see also Radiography
X-Y sex reversal, 301
Xylazine, 26, 303
anaesthesia, 492
epidural, *495*, 496
sedation, 490–491

Y

Yellow fat disease (steatitis), 450
Yellow star thistle poisoning, 218
Yew, 466

Z

Zinc, 467
Zygomycosis, 276–277