Reuben J. Rose
David R. Hodgson



Manual of EQUINE PRACTICE

SECOND EDITION

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SECOND EDITION

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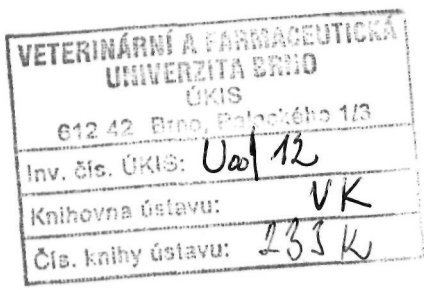
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Dedicated to our parents
Ben and Kit Rose
and
John and Judy Hodgson

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PREFACE

The first edition of *Manual of Equine Practice* had its genesis in a small handbook written in 1983 by Reuben Rose and published by the University of Sydney Postgraduate Foundation in Veterinary Science as the *Vade Mecum on Horses.* This book, which sold more than 3000 copies, provided the impetus for the *Manual of Equine Practice* and showed the need for a simple and informative book focused on the needs of students and practitioners.

The first edition of *Manual of Equine Practice* has been well received by veterinarians and students around the world. We have had extensive positive feedback about the book and its value to the veterinary student and large animal practitioner. Our aim in the first edition was to provide easily readable and accessible material for the veterinary student and equine practitioner. We became aware in our interactions with readers of the first edition that there were improvements that could be made and expansion of some of the material. We have retained the format of the first edition but have invited a variety of veterinarians from around the world with expertise in specific areas to update chapters and include new material. We believe that this has strengthened the text, which still retains its focus on diagnosis as a prelude to treatment. Within each body system chapter, we have subdivided the material into important clinical areas, and individual conditions are listed alphabetically in a standard format of: history and presenting signs, clinical findings and diagnosis, differential diagnosis, and treatment. A modest number of key references have been included in each section, which should be available to most practitioners. We have retained the format that relates any drug treatment to a reference section in the back of the book, providing details of the forms of the drug available, the trade names, and dose rates.

We continue to seek feedback on the book and particularly welcome cases of red wine being airfreighted to Australia!

REUBEN J. ROSE
DAVID R. HODGSON

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NOTICE

Equine medicine practice is an ever-changing field. Standard safety precautions must be followed, but as new research and clinical experience grow, changes in treatment and drug therapy become necessary or appropriate. The authors and editors of this work have carefully checked the generic and trade drug names and verified drug dosages to ensure that dosage information is precise and in accordance with standards accepted at the time of publication. Readers are advised, however, to check the product information currently provided by the manufacturer of each drug to be administered to be certain that changes have not been made in the recommended dose or in the contraindications for administration. This is of particular importance in regard to new or infrequently used drugs. Recommended dosages for animals are sometimes based on adjustments in the dosages that would be suitable for humans. Some of the drugs mentioned here have been given experimentally by the authors. Others have been used in dosages greater than those recommended by the manufacturer. In these kinds of cases, the authors have reported on their own considerable experience. It is the responsibility of those administering a drug, relying on their professional skill and experience, to determine the dosages, the best treatment for the patient, and whether the benefits of giving a drug justify the attendant risk. The publisher and editors cannot be responsible for misuse or misapplication of the material in this work.

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CHAPTER 1

Physical Examination

Reuben J. Rose and David R. Hodgson

A detailed physical examination is the cornerstone of equine practice. A difficulty that most equine practitioners face is the pressure of time, with the result that priority is given to treatment rather than to diagnosis. When a wrong diagnosis is made, it is often because key aspects of the physical examination are neglected. Sometimes this is something obvious, such as failure to auscultate the right side of the chest so that a heart murmur is missed. Other cases may be presented with one problem, for example, a foreleg lameness, but the most significant problem may be low-grade pulmonary disease that remains undetected because only the leg was examined and a general physical examination was not done. It is unrealistic to expect that a full physical examination will be undertaken on every horse seen in a busy equine practice because there are not enough hours in the day. However, it is essential to obtain an accurate history from the owner or trainer and to perform a basic examination on every horse seen. In most circumstances, this involves only an extra 2 to 3 minutes, but it can be of great value in saving later embarrassment or litigation.

One of the temptations when examining a horse for a particular problem is to stop the examination when a problem is discovered that matches the history. However, the importance of conducting a logical and sequential standardized examination cannot be overemphasized. First, a database of normal findings for the various body systems will be established, and second, familiarity and expertise develop in the expeditious performance of the examination. This helps to avoid the possibility of overlooking an important problem.

KEY POINTS

Important steps in establishing an initial diagnosis are

- *Determine the presenting complaint and establish the history relating to the particular problem.*
- *Undertake a complete physical examination in an attempt to define or localize the problem(s).*
- *Compile a specific problem list.*
- *Establish a series of differential diagnoses based on the problems identified.*
- *Undertake diagnostic tests, such as ultrasound, radiology, hematology, or blood biochemistry, that will assist in establishing a definitive diagnosis.*

It is essential to establish the most likely differential diagnoses before performing diagnostic tests because the tests should support the clinical findings. Too frequently, clinicians will perform a complete blood count and blood biochemical profile rather than a detailed clinical examination in the hope that something will show up. Not only is this poor veterinary medicine, but it also can divert attention from the real problem to a laboratory finding that may be of little clinical relevance.

Once these basic diagnostic steps are taken, therapy can commence, except in the case of emergencies that demand immediate treatment, for example, severe hemorrhage, shock, and colic. However, even in acute problems, a brief clinical examination that includes vital signs (heart rate, respiratory rate, rectal temperature, pulse quality, mucous membrane color, and capillary refill time) should be performed quickly before therapy.

SIGNALMENT

Diagnosis may be assisted by considering the age, breed, sex, and purpose for which the horse is used. Some specific details of signalment are in-

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eluded in Chapter 2, which outlines various presenting problems. Considering breed, sex, age, and use allows certain diseases to be placed at the top of the list of differential diagnoses. One of the problems in colleges of veterinary medicine is that a rather exotic range of cases may be presented that may not necessarily reflect the types of cases found in practice. For example, a student on equine rotation in some North American veterinary schools could easily get the impression that most horses presenting with colic require surgery. The reality is, of course, that in equine practice, many cases of colic will recover with minimal therapy.

KEY POINT

It should be remembered that "common diseases occur commonly."

One should always look for a simple explanation of a sign before something more complicated is diagnosed. For example, the horse at pasture with an acute hindleg lameness probably has a subsolar abscess rather than a hip luxation. Some details relating to signalment are discussed with examples related to specific diseases.

Age

MUSCULOSKELETAL DISEASE

Certain conditions, for example, multifocal septic arthritis, osteochondritis dissecans, and angular limb deformities, are more common in young horses. In contrast, degenerative joint disease is more common in older horses and is also affected by degree of use.

RESPIRATORY DISEASE

Age is important in determining the extent and severity of various respiratory disorders. Infectious respiratory disease tends to be more severe in the young and in the aged. This may be related to reduced immunocompetence. In contrast, chronic obstructive pulmonary disease (COPD) is found most commonly in middle-aged horses with repeated exposure to specific allergens. Horses with COPD usually have had prolonged exposure to a stable environment with poor ventilation.

CARDIOVASCULAR DISEASE

With cardiac disease, although congestive heart failure is most commonly found in older horses, congenital heart disease may not be manifest until athletic endeavors are required. Horses usually are presented because of exercise intolerance or dyspnea.

GASTROINTESTINAL DISEASE

Age may contribute to the incidence and type of gastrointestinal abnormalities. Gastric ulceration, pyloric stenosis, and ascarid impaction must be considered when a foal is presented with clinical signs of colic. Volvulus and intussusception of the small intestine occur more commonly in horses less than 3 years of age. Conversely, pedunculated lipomas are more common in older horses, as is gastric neoplasia.

NEUROLOGIC DISEASE

Neonatal maladjustment syndrome and trauma are the most common causes of neurologic disturbances in foals, whereas in yearlings and 2 year olds, cervical stenotic myelopathy is a common cause of spinal cord ataxia. The most common neurologic disease in older horses in the United States is protozoal myeloencephalitis.

SKIN DISEASE

Ringworm is a common cause of pruritus and focal alopecia in young horses, whereas *Culicoides* hypersensitivity is more common in older horses.

Breed

MUSCULOSKELETAL DISEASE

Breed predilections exist for some conditions, causing lameness. "Bucked shins" is a common problem of young Thoroughbred and Quarter horses in training. Chip fractures of the dorsal aspect of the carpal bones are more frequently diagnosed in Thoroughbreds and Quarter horses than in Standardbreds. Standardbreds tend to have a greater incidence of hindleg lameness and fractures of the pedal bone than Thoroughbreds or Quarter horses. Quarter horses appear to have a very high incidence of navicular disease, whereas the incidence is lower in Arabians. In ponies, upward fixation of the patella and laminitis are more common than in other breeds. Hyperkalemic periodic paralysis is a problem exclusively of Quarter horse and Quarter horse cross-breeds.

RESPIRATORY DISEASE

Although few breed-specific associations have been found for respiratory disease, combined im-

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munodeficiency is primarily found in Arabian foals. Many of these foals are presented with chronic respiratory infections that are unresponsive to therapy. In the Thoroughbred, laryngeal hemiplegia is more common than in other breeds and is generally first noticed between 2 and 3 years of age. Laryngeal hemiplegia is also very common in draft horses.

CARDIOVASCULAR DISEASE

Atrial fibrillation appears to be found more commonly in Standardbred trotters and pacers than in other performance horses. Congenital cardiovascular disease is more common in Arabian horses than in other breeds.

GASTROINTESTINAL DISEASE

Atresia ani may be more common in Appaloosa horses. Entrapment of the epiploic foramen is more common in Thoroughbred horses than in other breeds.

NEUROLOGIC DISEASE

Breed can be important in neurologic disease. Thoroughbreds have a higher incidence than other breeds of cervical stenotic myelopathy, whereas Arabians have an assortment of congenital problems, including cervical malformations and cerebellar abiotrophy.

SKIN DISEASE

Pemphigus foliaceus is found more commonly in Appaloosa horses than in other breeds. A condition, known as "curly coat," is an inherited condition in Percheron horses. A depigmenting condition known as "pinky syndrome" or Arabian fading syndrome occurs in Arabian horses.

Sex

Sex of the horse is less important than age, breed, and use in predisposing to or determining disease states. However, it is obvious that some conditions, for example, inguinal hernias, will occur only in colts or stallions. In contrast, in mares the hormonal changes associated with pregnancy and lactation may play a role in the onset of some diseases. Osteochondritis dissecans, cervical stenotic myelopathy, and idiopathic laryngeal hemiplegia are more common in males than in females, whereas rhabdomyolysis or "tying up" is more commonly ascribed to fillies and mares than stallions or geldings.

Use

Use of the horse is of major importance in determining the likelihood of various types of abnormalities. This will clearly interact with the horse's breed.

MUSCULOSKELETAL DISEASE

Fractures of the phalanges, sesamoids, and carpal bones, together with tendon strains, are more common in athletic horses, whereas subsolar abscesses and long bone fractures appear to be relatively more common in horses in groups at pasture. Back problems and neck injuries are more common in jumping and hunting horses. Navicular disease is more common in horses used for cutting, roping, and barrel racing.

RESPIRATORY DISEASE

Respiratory disease is more common in horses stabled in crowded, stressful, and/or poorly ventilated environments. In horses that are transported long distances, pneumonia and pleuritis, secondary to disturbances in respiratory defense mechanisms, are more common. Some functional upper airway disorders (e.g., laryngeal hemiplegia) will be of little or no significance in dressage horses, whereas such afflictions will severely limit horses performing at high exercise intensities.

CARDIOVASCULAR DISEASE

Minor cardiovascular problems and some major ones (e.g., severe valvular incompetence and atrial fibrillation) only may result in clinical manifestations in horses required to perform competitive athletic activities.

GASTROINTESTINAL DISEASE

Various types of colic appear to be more common in horses kept at pasture than in horses in stable environments. For example, sand colic and "grass sickness" are more common in pastured horses.

NEUROLOGIC DISEASE

Traumatic neurologic disease is common in showjumping, eventing, and steeplechasing horses and in all young horses when in early stages of training.

SKIN DISEASE

Ringworm is more common in horses kept in stables, particularly when there is a predominance of young horses under these circumstances.

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KEY POINT

Management, husbandry, and geography are important in determining the types and incidence of various diseases.

For example, pneumonia and septicemia in foals are more common on stud farms where there is overcrowding and poor management. *Rhodococcus equi* infections usually have a higher incidence in specific geographic areas that have hot dry summers. This may also be related to soil type, because infections are more common in foals kept on sandy soils. Other examples include sand colic, which tends to occur most commonly in drier sandy regions, and equine protozoal myeloencephalitis, with greatest incidence in the eastern regions of North America.

HISTORY

Before any examination, a thorough and accurate history should be obtained. History taking is a skill that has to be developed and is an art more than a science. The ability to ask the right question can sometimes be important in indicating the most likely differential diagnoses. In obtaining the history, leading questions should be avoided. These are questions where the owner is led to provide the answer that the veterinarian wants rather than the one that reflects the history of the horse's problem. One of the most common mistakes is to rush into making a diagnosis from the initial history. Misleading histories are quite common because many owners make their own diagnosis of their horse's problem before its presentation to the veterinarian. This often results in a selective history that reflects the owner's or trainer's bias.

History taking has two major components: past and present. These are equally important in obtaining the correct diagnosis and in determining the approach to therapy.

KEY POINT

The current history begins with questions relating to the presenting signs and should seek the owner's or trainer's assessment of the problem as static, worsening, or improving.

The detail and extent of the history collected will depend on the duration of the problem and need for any immediate therapy. For example, many details of history have little relevance to the immediate treatment of a horse that has severe colic and shows signs of shock.

KEY POINT

The past history should begin with questions about the horse's condition immediately before the onset of the problem, followed by selective details.

The details sought will vary from months to years before presentation, depending on the nature of the problem. For example, in a horse presented for colic, it is of great significance that the problem is recurrent over several years rather than a single, isolated event. Chronic problems have a more guarded prognosis than isolated events related to sudden changes in feed or exercise state. Changes in appetite and demeanor together with alterations in body condition are important features of a variety of diseases.

Some specific questions that provide important information relating to the problem being investigated are provided in Chapter 2.

KEY POINT

Despite careful and detailed history collection, the clinician should view the information with some skepticism.

It is common for conflicting details to be given when a second veterinarian questions the same client. In particular, there is great potential for conflict between veterinarians to arise because the client may be critical of the initial or referring veterinarian and present a very biased version of the first examination and treatment. If the case has been seen by another veterinarian, it is incumbent upon the consulting veterinarian to call the initial veterinarian to verify aspects of the treatment of the case and to ensure no misunderstanding or professional conflict arises.

KEY POINT

When obtaining the history, it is important to avoid any scientific jargon, because many owners will be reluctant to tell you that they do not understand. This also applies when discussing a diagnosis or deciding which option should be used in therapy.

It may be useful to confirm an important detail later in the examination by asking a previous question in a slightly different manner.

PHYSICAL EXAMINATION

This section discusses the broad approach to a physical examination. Specific information is pro-

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vided in each of the chapters on body systems, which include diagnostic aids. In the initial examination, the main consideration is to determine or confirm from the history provided which body system(s) is involved.

A number of approaches may be used when the initial examination is undertaken. Although many problem-oriented record systems work through a system approach to examination, it is probably more common in practice to start the examination at the front of the horse and work to the rear. The time spent on a specific region will be dictated by the history and presenting complaint. However, many problems involve multiple body systems. For example, in a Thoroughbred horse presented for exercise intolerance, it is quite common to find that the horse has a low-grade lameness, abnormal respiratory sounds on auscultation of the chest, evidence of atrophy of the left dorsal cricoarytenoid muscle, and a cardiac dysrhythmia. Therefore, it is important, even with the most obvious problem, to perform as thorough a physical examination as the circumstances allow.

General Overview

The initial part of the examination should involve an overview of the horse. This brief evaluation should be conducted at a distance of 2 to 3 m (6-10 feet), and the horse should be viewed from the front, left and right sides, and rear. Of particular importance is the demeanor, which can indicate if the horse is showing signs of systemic disease or is in pain. Note should be made of whether the horse is alert or shows signs of depression.

KEY POINT

During this brief examination, the mental state of the horse, together with any asymmetries, swellings, or other irregularities, should be noted before commencing a more detailed examination.

Evidence of scars, rub marks, or localized hair loss may be more evident at this stage of the examination. After taking note of obvious abnormalities, the more detailed examination should begin.

Examination of the Head and Neck

- *The nostrils should be checked* for evidence of any nasal discharge, asymmetry, and differences in airflow between the two sides. Additionally, the air from the nostrils should be smelled for any abnormal odors, which could indicate infection of the nasal conchae, sinuses, guttural pouches, or the lower respiratory tract.
- *The incisor teeth are inspected* for evidence of malocclusion (Fig. 1-1), after which digital pressure is applied to the mucous membrane above the corner incisor teeth to determine the capillary refill time (Fig. 1-2).
- *Normal capillary refill time is 1 to 2 seconds,* and this is increased in horses with shock due to a decrease in peripheral perfusion. The normal mucous membrane color is pale pink.
- *The teeth can be examined* by inserting a thumb in the interdental space and pressing on the hard palate to encourage the horse to open its mouth. For a more detailed examination, a one-hand technique (Figs. 1-3 and 1-4) can be used, with the back of the hand inserted via the interdental space and used to push the tongue between the teeth on the opposite side of the mouth. This means that the horse will have to bite its tongue before it crushes your fingers. Alternatively, a mouth gag can be used, as shown in Figures 1-5 to 1-7. Of the various gags available, we prefer the Swale's gag (see Figs. 1-6 and 1-7) because it is easy to apply and is well tolerated by most horses.

KEY POINT

Abnormalities of tooth wear, dental abnormalities, and sharp edges on the labial side of the upper cheek teeth and the lingual side of the lower cheek teeth should be noted. Sharp edges on the teeth can contribute to a range of problems because of lacerations to the mucous membranes of the mouth when the bit is applied.

At this stage, the age of the horse also can be determined.

- *After examination of the nasal contours for swelling, percussion of the maxillary and frontal*



Figure 1-1. Incisor teeth inspected for malocclusion.

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Figure 1-2. Digital pressure applied to the gums above the corner incisor teeth to determine capillary refill time.

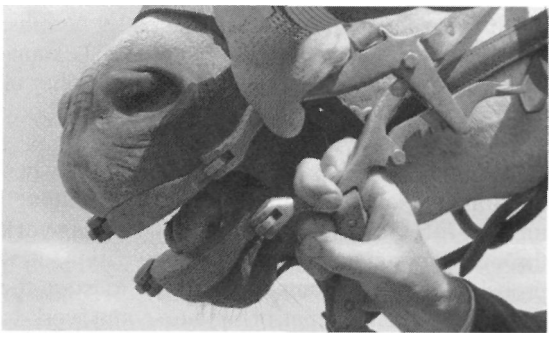


Figure 1-5. Application of a Hausmann mouth gag showing opening of the gag before examination of the mouth and teeth.



Figure 1-3. Introduction of the left hand into the left interdental space for a one-handed tooth examination.



Figure 1-6. Insertion of a Swale's mouth gag via the left interdental space to allow examination of the right dental arcades.



Figure 1-4. Position of the left hand in the oral cavity, forcing the tongue to the right occlusal surfaces of the cheek teeth so that palpation of the left cheek teeth can be done.

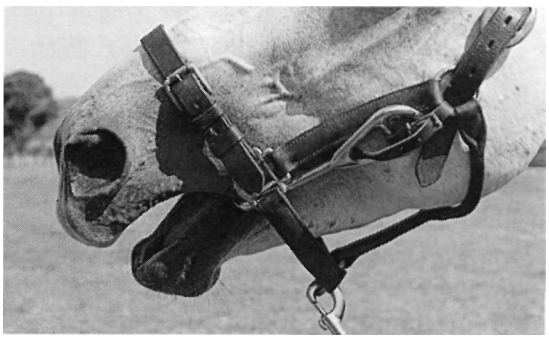


Figure 1-7. Swale's mouth gag in place.

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sinuses should be performed (Figs. 1-8 and 1—9). This ensures no evidence of pain or a dull sound, which could indicate sinusitis or the presence of fluid within the sinus cavities. The sound obtained is more distinctive if a thumb is placed in the interdental space so that the mouth is slightly open during percussion.

- *The eyes should be examined* for signs of corneal scarring, conjunctivitis, global inflammation, or cataracts. A menace response should be elicited (Fig. 1-10), and a direct pupillary light response (Fig. 1-11) and a consensual response should be determined. The third eyelid also should be examined by applying digital pressure to the eyeball via the upper eyelid (Fig. 1-12).
- *The pulse should be felt in the facial artery* as it turns around the angle of the mandible (Fig. 1-13A). Other sites for determining the pulse rate and quality are shown in Figure 1-13B to *D*. The important things to note are the pulse character, amplitude, and regularity.
- *Palpation for lymph node enlargement* should be performed between the rami of the mandibles and in the region of Viborg's triangle to determine if enlargement of the mandibular and pharyngeal lymph nodes is present.
- *The larynx also should be palpated* at this stage to determine if atrophy of the dorsal cricoaryte-

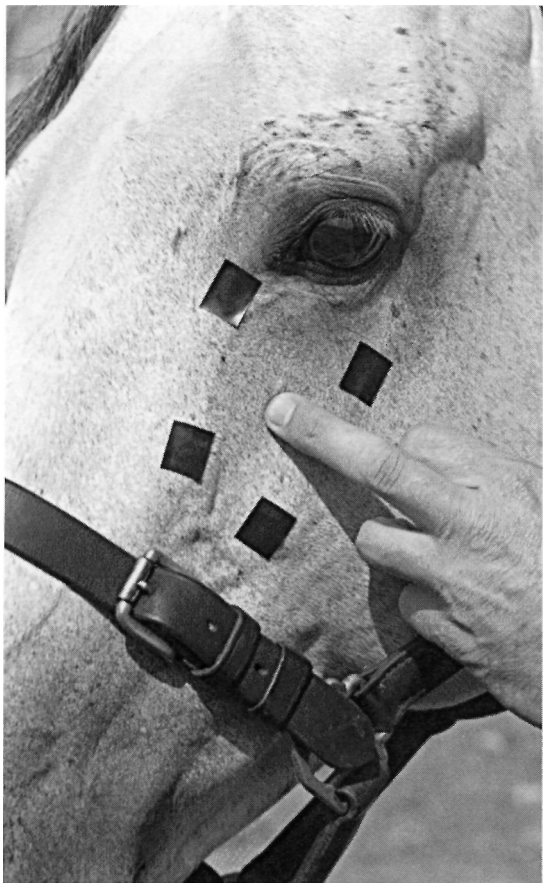


Figure 1-8. Percussion over the maxillary sinus. The black tape shows the boundaries of the sinus.

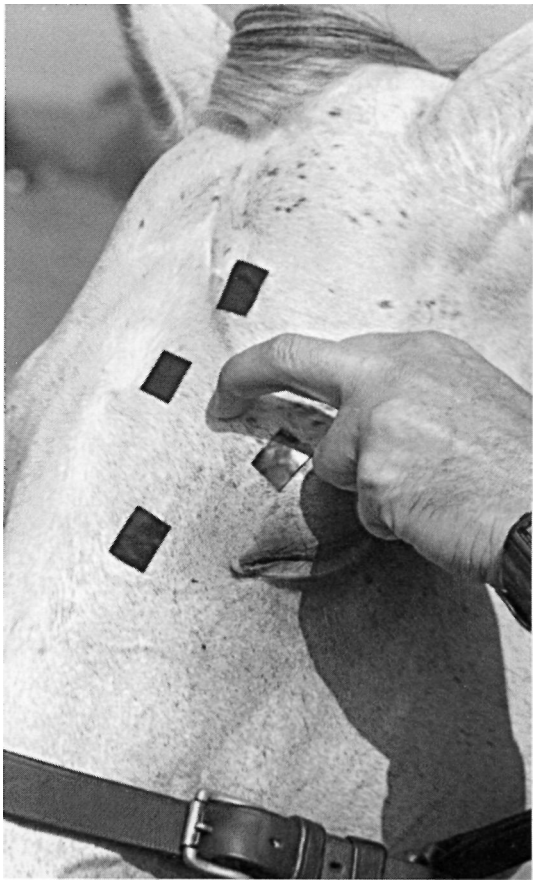


Figure 1-9. Percussion over the frontal sinus. The black tape shows the boundaries of the sinus.



Figure 1-10. Evaluation of the menace response.

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Figure 1-11. Evaluation of a direct pupillary light response.

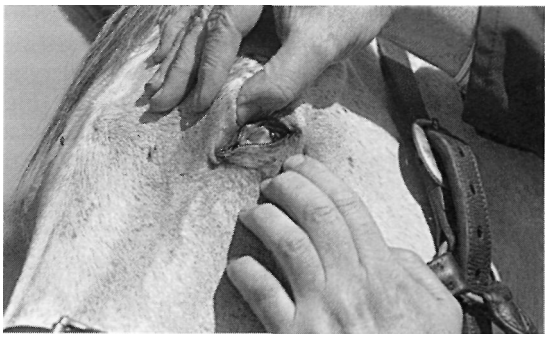


Figure 1-12. Protrusion of the third eyelid (nictitating membrane) by pressure applied to the eyeball via the upper eyelid.



Figure 1-13. *A.* Palpation of the pulse in the left facial artery over the left horizontal ramus of the mandible. *B.* Palpation of the pulse in the transverse facial artery just caudal to the lateral canthus of the right eye. C Palpation of the pulse in the median artery of the left foreleg. (Note that it may be difficult to detect a pulse in this location because the artery is quite deep.) D. Palpation of the pulse in the digital (palmar) artery of the right foreleg.

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noid muscle can be felt (Fig. 1-14). If present, as in horses with idiopathic left laryngeal hemiplegia, the muscular process of the left arytenoid cartilage will be more prominent than on the right side. Any scarring or thickening of skin that could suggest a previous laryngoplasty or laryngotomy should be noted.

KEY POINT

/f there is any indication of more prominence of the left muscular process of the arytenoid cartilage, a laryngeal adductor ("slap test") should be performed while palpating the muscular process on left and right sides.

Slapping the left thorax gently with the open hand should result in the right muscular process adducting, and this can be felt as a flicking or movement of the process. Similarly, slapping of the right thorax should result in the muscular process of the left arytenoid cartilage "flicking." In horses with idiopathic left laryngeal hemiplegia, the flicking of the left muscular process does not occur or is reduced in response to the slap test. The slap test response also may be lost in horses with cervical spinal cord disease. It should be noted that a response to the slap test may not be noted if the horse is excited or has been sedated.

- *Palpation of the lateral processes of the cervical vertebrae* is done (Fig. 1-15), and the range of lateral movement and degree of neck flexion are noted, together with any indications of pain.
- *The trachea also should be palpated* (Fig. 1-16) in the cervical region, and any narrowing should be noted. Both jugular veins should be checked for patency so that thrombophlebitis can be diagnosed. This problem is common in racehorses that receive regular intravenous injections. Evidence of an increased jugular pulse also should be noted because this could indicate congestive heart failure.



Figure 1-14. Palpation for prominence of the left muscular process of the arytenoid cartilage indicating atrophy of the left dorsal cricoarytenoid muscle. The left index finger is shown ventral to the tendon of the sternocephalicus muscle. The muscular process is palpated on the dorsum of the larynx. This position is also used when evaluating the "slap test" for laryngeal adductor function.



Figure 1-15. Palpation of the transverse processes of the cervical vertebrae shown on the left side of the neck.

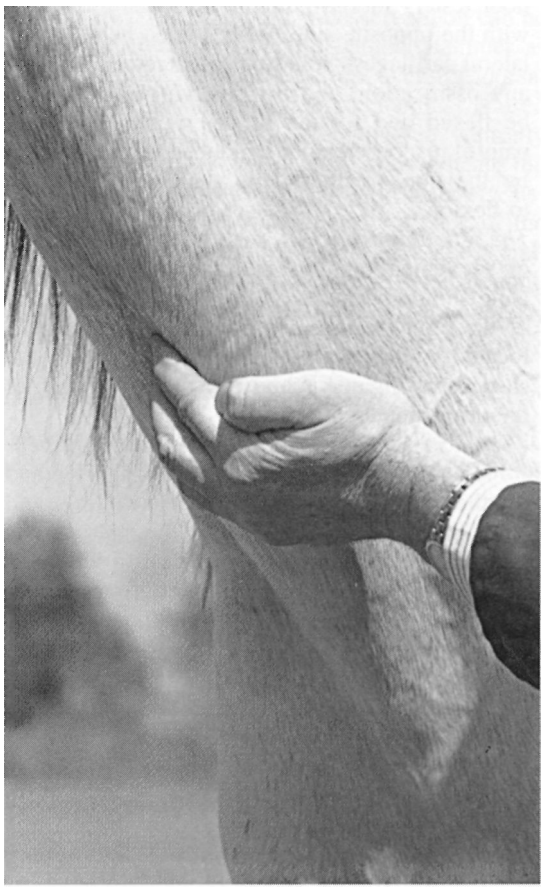


Figure 1-16. Palpation of the cervical trachea for signs of stenosis.

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EXAMINATION OF THE FORELEGS

- *The forelegs should be inspected* for any swellings, particularly from the carpus distally, because most forelimb lamenesses are found in the lower part of the leg. Note particularly the proximal carpal and midcarpal joints, the second and third metacarpal bones, the flexor tendons, the interosseous (suspensory) ligament, and the fetlock joint, including the palmar pouch. It is important to compare any response with that in the opposite foreleg.

KEY POINT

Figures showing detailed examination of the forelimb are shown in Chapter 4, Figures 4-1 to 4-14.

- *Examination of the foreleg should commence at the foot,* and after checking for signs of increased heat around the hoof wall and coronary band, hoof testers should be applied around the foot and across the frog to determine if pain can be elicited (see Chapter 4). Any response to hoof testers should be rechecked and compared with the opposite side. With the leg held up, the lateral cartilages should be palpated to check for any ossification, and the pastern region should be flexed and rotated to determine whether a painful response can be produced. This is shown by the horse withdrawing its leg in response to flexion.
- *The fetlock joint is flexed* to examine the range of movement and to ascertain signs of pain. Note should be made of any dorsal enlargement of the joint or distension of the palmar pouch, located in the distal metacarpus between the suspensory ligament and the palmar aspect of the third metacarpal bone.
- *The flexor tendons and interosseous (suspensory) ligament are palpated,* with weight on and off the leg, for evidence of heat, swelling, and pain. Note that firm pressure on the interosseous ligament near its point of bifurcation will produce a painful reaction in many normal horses. The area of the inferior check ligament, just distal to the carpus on the palmar aspect of the proximal third metacarpal bone, also should be palpated. However, it should be remembered that inferior check ligament desmitis can exist without demonstrable pain.
- *The second and fourth metacarpal bones are palpated* to determine if any swellings ("splints") are present proximally or for swelling and callus and pain associated with a fracture of the distal part of one of the small metacarpal bones, particularly the second metacarpal bone.
- *The dorsal aspect of the third metacarpal bone is palpated* to determine if pain resulting from bucked shins is present, and the area should be examined for any swelling and to determine if it is hotter than normal.
- *The carpus should be examined closely* because carpal injuries are common in athletic horses. Any distension of the joint capsule over the dorsal aspect of the proximal carpal (antebrachiocarpal) and middle carpal (intercarpal) joints should be noted and the joints carefully palpated and flexion tests performed. In some acute injuries, pain can be elicited by firm digital pressure being applied over the affected carpal bone. The carpus then should be flexed as much as possible. In many chronic carpal injuries, pain will not be evoked until the last few degrees of carpal flexion. A normal horse will not show any response to extreme carpal flexion, and therefore an adverse response should be treated with suspicion. The area above the carpus is difficult to examine, particularly with regard to localizing signs. Flexion, extension, and abduction of the upper forelimb can be carried out to check for signs of pain and the musculature palpated.

EXAMINATION OF THE CHEST

- *When examining the chest,* the most important part of the initial assessment is to *note the character and frequency of the respirations.* This is best done at a distance so that the degree of respiratory effort can be noted. In the absence of lower respiratory disease, a horse at rest will have a slow respiratory rate (8-16 breaths/min) with limited evidence of chest-wall movement. Any prolongation of inspiration or expiration should be observed. In addition, if the abdominal component of expiration is pronounced, a lower airway problem can be suspected. After palpation with the open hand over the apex beat, the heart should be auscultated using a stethoscope over this area and then progressively moved one to two intercostal spaces dorsally and cranially with the bell firmly placed well under the triceps muscles at a level just ventral to an imaginary line through the point of the shoulder (Fig. 1-17). This allows auscultation of sounds associated with the left atrioventricular valve and aortic and pulmonic valves. Auscultation should also be made over the right chest for abnormalities associated with right atrioventricular valve problems and other right outflow tract problems. Auscultation over the four valve areas should be undertaken for at least 1 minute, noting disturbances of rhythm or the presence and timing of murmurs. The de-

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Figure 1-17. Auscultation of the heart, showing position of bell of the stethoscope under the triceps musculature.

tailed auscultation of the heart is described in Chapter 6.

KEY POINT

The normal heart rate at rest ranges from 28 to 36 beats/min in the adult horse and 70 to 100 beats/min in foals during the first few days of life. Excitement or apprehension may result in mild increases in the resting heart rate.

- *Auscultation of both the left and right lung fields is carried out* (Fig. 1-18), but in most normal horses breathing quietly at rest, few sounds will be detected. The exception to this is auscultation over the hilar region and the trachea. If there is any suspicion of abnormal sounds (gurgles or wheezes), a plastic bag can be placed over the horse's nose to stimulate respiration due to the combination of hypoxia and rebreathing of carbon dioxide (see Chapter 5). This technique will increase both the frequency and depth of respiration and thus permit abnormal lung sounds to be heard more easily. Care should be exercised in using the rebreathing bag technique in horses with pleuritis due to the induction of apparent severe pain.

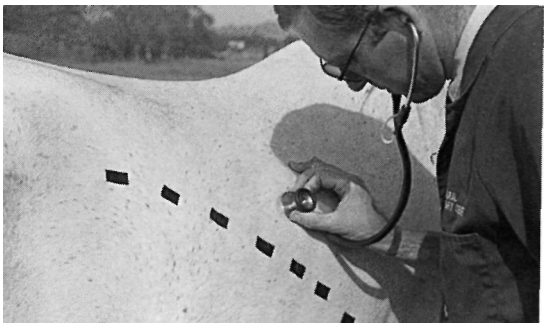


Figure 1-18. Auscultation of the lung fields showing outline of the diaphragm with black tape.

- *Percussion of the chest wall should be undertaken* if there is any indication of a respiratory abnormality. This is achieved using a plexor and pleximeter. Alternatively, a dessert spoon can be used as a pleximeter and a patellar hammer as a plexor. The spoon is placed over the dorsal aspect of one of the ribs and the hammer is used to tap on the spoon as it is moved ventrally (see Chapter 5). A dull sound may indicate the presence of fluid in the chest. Alternatively, using the thumb and third finger, the back of the fingernail can be flicked against the chest wall (Fig. 1-19). The area of the thorax over the lung fields should be percussed from dorsal to ventral and from cranial to caudal.

EXAMINATION OF THE ABDOMEN

Detailed examination of the abdomen is difficult, and if an abdominal problem is suspected, various

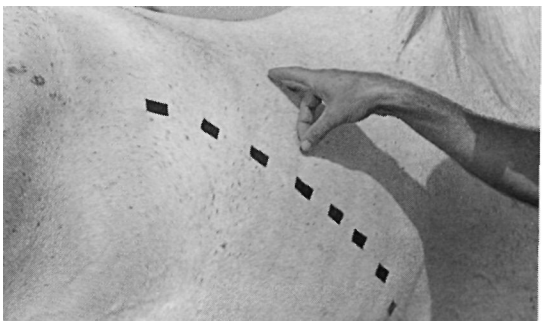


Figure 1-19. Digital percussion technique over the lung fields to determine areas of dullness. The middle finger is flicked against the chest wall.

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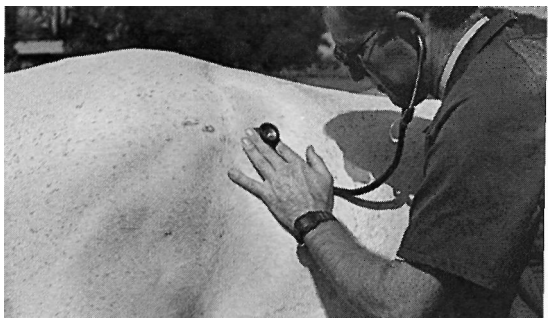


Figure 1-20. Auscultation of the abdomen showing examination of the right paralumbar fossa.

specialized procedures may be necessary. However, at the initial examination, note should be taken of the abdominal outline to determine abdominal distension, which may be found in certain types of colic where there is tympany of the large bowel.

- *Auscultation of the abdomen should be performed* on the left and right sides, over both paralumbar fossae and midflank and lower flank regions (Figs. 1-20 and 1-21). Over the right paralumbar fossa, ileocecal valve sounds (these sound like a toilet flushing) may be heard approximately every 30 to 60 seconds. It is important to determine whether the gut sounds are normal, increased, or decreased. Such a determination can only be made after examining a large number of normal horses to form an effective reference base.
- *If abdominal distension is present,* percussion while listening with a stethoscope placed over the area of distension can be useful to determine if there is a gas-filled viscus.
- *The rectal temperature should be taken* at this stage. Normal values are in the range 98 to 102°F (36.5-39°C) for adult horses. Rectal temperature can be increased because of transport, stress, or excitement, and foals tend to have rectal temperatures at the high end of the reference range.

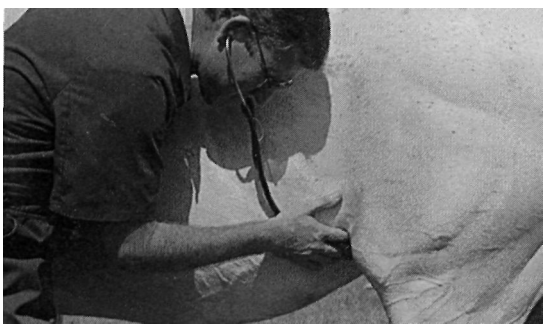


Figure 1-21. Auscultation of the abdomen showing examination over the left lower flank region.

EXAMINATION OF THE BACK

Structural or anatomic abnormalities of the back, including scoliosis (abnormal lateral deviation of the verebral column), lordosis (abnormal ventral deviation), and kyphosis (abnormal dorsal deviation), can be best appreciated by standing behind (but not too close) to the horse that is weight bearing evenly on the hindlegs. It is sometimes useful to have something to stand on so the horse's back can be viewed from above, particularly for a tall horse.

- *The dorsal spinous processes should be palpated* over the thoracic and lumbar regions, and firm pressure should be applied over the tuber sacral. In horses with hindleg weakness or sacroiliac pain, mild pressure at this site will result in the horse crouching away from the examiner.
- *Stroking, for example with the blunt end of a ballpoint pen,* over the thoracolumbar area results in the horse dipping its back, whereas doing so at the caudal sacral region will cause the horse to arch its back. Some horses are hypersensitive to this examination, and the reaction found should be assessed and differentiated from a response to a pathologic process involving soft tissue or vertebrae.

EXAMINATION OF GENITALIA

- *Unless the history indicates* the possibility of a urogenital problem, *a detailed examination of the genitalia is not necessary.*
- *In both stallions and geldings, the preputial area should be examined* for any signs of discharge, which could indicate an infection, squamous cell carcinoma, or habronemiasis. In a colt or stallion, the testicles should be palpated to make sure that the horse is not a cryptorchid and to determine whether any other palpable abnormalities exist.
- *In fillies and mares, the perineal conformation should be noted* to determine the likelihood of pneumovagina or previous perineal injury causing a clinical problem. The presence of current or previous Caslick's operations also should be noted. If there are any signs of discharge from the vulva or "scalding" around the hindlegs, a more detailed reproductive or urinary examination is required, together with catheterization of the bladder and a rectal examination.

EXAMINATION OF THE HINDLEGS

The distal part of the hindlegs is examined in a similar manner as for the forelegs.

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- *The hock is the most common site of chronic hindleg lameness* and therefore should be inspected carefully for swelling. However, in many cases of osteoarthritis involving the lower hock joints, no swelling is apparent.
- *The stifle should be examined* by palpating between the three patellar ligaments. The femoropatellar pouch can be palpated between the medial and middle patellar ligaments and the middle and lateral patellar ligaments. In gonitis, it is common to find distension of the femoropatellar pouches.
- *Hindleg symmetry* should be checked because in some chronic hindleg lamenesses, atrophy of muscle groups, particularly the gluteal muscles on the affected side, may be evident.

EXAMINATION OF THE GAIT

After completing the general examination, the history will indicate whether the gait should be assessed in detail. This is most likely to be necessary for a musculoskeletal or neuromuscular problem. The basic examination should include walking and trotting the horse on a hard surface to detect any signs of lameness or ataxia. It also may be useful to lunge the horse if a musculoskeletal problem is suspected because some lamenesses are more obvious when the animal is trotted in a tight circle. Examining the horse under saddle or at the track in harness (for Standardbred horses) may be necessary.

Rectal Examination

A rectal examination is not usually considered to be part of the normal examination, but it should be undertaken where there is a history of a gastrointestinal problem, an unusual hindleg lameness, an internal reproductive disorder, weight loss, or fever of unknown origin. It is important that the horse is adequately restrained and, if necessary, tranquilized. Restraint is best and most safely achieved in stocks, although the use of tail restraint with or without a sideline is also quite effective.

Details of how to perform a rectal examination are presented in Chapter 7. For general examination, we have found the following sequence to be useful. After introduction of the arm, the pelvis is examined for any swelling or crepitus. If a pelvic problem is suspected, different regions of the pelvis can be palpated while an assistant gently rocks the horse by pushing against the tuber coxa on either the left or right side. After examining the pelvis, the bladder and reproductive organs (in the mare) should be examined. Next, the left lateral abdominal wall is palpated. With further anterior exploration, the caudal border of the spleen is felt and dorsally the caudal border of the left kidney is evident. Although variable, the pelvic flexure of the large colon may be palpable on the left side of the ventral caudal abdomen. On the right side of the abdomen, the small colon can be determined by the presence of fecal balls and its mobility. Depending on the degree of distension of the cecum, different portions can be palpated. If it is severely distended with gas or impacted with food, the base can be felt toward the dorsal abdominal wall. However, in most conditions, the only reliable finding is the ventral taenia. Palpation of the mesenteric root, cranial mesenteric artery, and lymph nodes is difficult in most large horses, particularly if there is abdominal pain present. In stallions, it is always wise to check the integrity of the internal inguinal rings, which can be felt on the lateral part of the ventral abdominal wall just cranial to the femoral canal. In some conditions, enlargement of the inguinal lymph nodes can be detected.

KEY POINT

After completing a rectal examination, it is very important to inspect the glove for blood, which could indicate mucosal damage or a rectal tear. If blood is found, immediate steps to determine the cause and severity should be taken (see Chapter 7 for detailed discussion).

Prepurchase Examination

The prepurchase examination, also referred to as the "soundness examination" or "vet check," has great potential for conflict. Many vendors sell horses with problems that they hope will not be detected before money changes hands. The veterinarian may unwittingly be caught in the middle of a dispute, which may result in litigation. There are some important points about prepurchase examinations that can help in preventing these problems:

- The examination should only be performed on behalf of the purchaser, not the seller.
- Veterinarians should resist any attempt by a vendor to get them to examine a horse that is for sale because there is inevitably a conflict of interest.
- Written statements from both the seller and purchaser may be helpful and prevent misunderstandings in the future about the reasons for the examination. The American Association of Equine Practitioners has a useful format for history collection and examination (Table 1-1).
- It should be clarified with the client exactly what is included in the examination. In many

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TABLE 1-1. Prepurchase Examination Form, American Association of Equine Practitioners

Name of horse Breed Tattoo Sex Color Age Markings

Seller's statement before examination:

Seller's name Address Phone number

How long have you been acquainted with this animal?

How long have you had this animal under your personal care?

Do you have knowledge of present or past Diseases Lameness

Treatments Vices (stable or being ridden) Disabilities

Medications Eccentricities

Do you have knowledge of past performances of this animal for the proposed use?

Do you have a personal estimate of the suitability of this animal for this purpose?

Unique Exceptional Adequate No opinion

Signature of Seller Date

Address

Buyer's statement of the purchase of this horse:

Buyer's name Address Phone number

To what use do you intend to put this horse (degree of work, hours to be used)?

What is the age, size, ability, and experience of the intended rider?

How long have you been acquainted with this animal?

How long have you tried this animal?

How many of the proposed uses have you tried?

Of what relative importance are the following to you?

Appearance of the horse including (and any) blemishes

Performance Temperament

How do you rate the suitability of this horse for the intended purpose?

Unique Exceptional Adequate

What type of care (stabling) is anticipated for this horse?

Intensive (continual care and supervision)

Average (stabled daily for feeding, etc.)

Casual (on pasture most of the time)

Signature of Buyer Date

Address

PHYSICAL EXAMINATION

Place Date Time

Weather

GENERAL HEALTH AND APPEARANCE

Approximate height Approximate weight

Cert. of height (pony) Temperature (rectal)

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Complete the following with N = normal, AB = abnormal, NE = not examined.

A. Bilateral symmetry
1. Head and neck
2. Body
3. Legs
4. Feet

B. Eyes
1. Symmetry
2. Reflexes
3. Lids
4. Mucous membranes
5. Cornea
6. Ophthalmoscopic examination

C. Mouth
1. Lips
2. Tongue
3. Teeth
4.. Gums
5. Mucous membrane
6. Odor
7. Bite

D Nasal and paranasal
1 Symmetry
2. Airflow
3. Odor
4. Mucous membranes
5. Percussion
6. Exudate

E. Pharynx, larynx, trachea
1. Palpation
2. Cough induction (reflex)
3. Auscultation at rest
After exercise
After recovery

F. Cardiovascular
1. Palpation (heart and pulse)
2. Auscultation at rest
After exercise
After recovery
3. Pulse rate and quality

G. Pulmonary
1. Percussion
2. Auscultation at rest
After exercise
After recovery
3. Respiratory (rate rest)

H. Digestive
1. Percussion
2. Auscultation
3. Inspection of feces

I. Genital-urinary
1. External
2. Inspection and palpation
a. Breeding soundness, mares
Barren Maiden
Foaling
(1) Rectal examination
(2) Speculum examination
(3) Culture
b. Breeding soundness, stallions
Rectal examination
(2) Test breeding
(3) Culture
(4) Semen examination
(5) Inspection and palpation

J. Integument
Note especially "used" marks (interference with the saddle, sores, firing or other treatment, dermatoses, etc.). Insignificant scars need not be enumerated.

K. Musculoskeletal
a. Vertebral column
(1) Symmetry
(2) Palpation
(3) Manipulation
b. Limbs
(1) Symmetry
(2) Palpation
(3) Manipulation
c. Gaits
(2) Freedom of movement on hard surface
(3) On soft surface
(4) On a straight way
(5) Turning both ways

L. Vices: Cribbing Weaving
Digging Savaging Other
1. Stable manners
2. Field manners

M. Nervous system
1. Inspection
2. Has horse been nerved
3. If so, where

Conditions other than normal found in the animal (list by title):

SPECIAL PROCEDURES

ECG
Endoscopy
Radiographs
Rectal examination
Nerve blocks
Laboratory studies
Other

Signature:
Address:
Date:

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cases, problems arise because the client has not understood the nature of the examination. A prepurchase examination is a detailed physical examination without the use of any diagnostic aids such as radiography, ultrasonography, endoscopy, and electrocardiography. The latter specialized techniques may be offered as additional aids to the routine examination. The term *complete physical examination* should be avoided. However, the possibility of use of additional diagnostic procedures should also be discussed and offered to the client.

KEY POINT

A prepurchase examination must result in a written report that clearly identifies the horse examined and lists the problems found. The terms sound *and* unsound *should be avoided.*

The report should contain a clear description and markings (preferably a diagram) of the horse together with the time, date, and place of examination. The American Association of Equine Practitioners advises that no opinion should be given as to the suitability of the horse, which is the sole responsibility of the buyer. However, the report should provide an opinion as to the functional significance of any abnormal findings listed.

It is easy to overlook a body system if a checklist is not used. The format set out in Table 1-1 is that suggested by the American Association of Equine Practitioners, and we have found it very helpful in ensuring a detailed examination.

Physical Restraint

With better drugs available for restraint of horses, physical restraint is less important than in the past. However, there are a range of techniques that are of considerable use in equine practice. It is important to be confident when approaching horses because they can detect nervousness and are often more difficult to handle if they sense this emotion in the handler. Most horses are used to being approached from the left or near side.

KEY POINT

The most common mistake seen in students and clinicians used to working with horses in stocks, is that they stand in front of horses (at pasture or in a stall or loose box) when performing tasks such as stomach tubing and teeth examination. This is a certain way to be struck by the front feet of a horse.



Figure 1-22. Restraint of a horse by grasping a fold of skin on the left side of the neck.

All examinations and procedures performed in the unrestrained horse should be done standing beside and close to the horse at the level of the shoulder. The simplest restraint procedures for temporary diversion of the horse to allow insertion of a needle or injection include grasping a fold of skin on the neck (Fig. 1-22) and twisting an ear (Fig. 1-23). If further restraint is required, the use of a twitch is useful (Figs. 1-24 and 1-25). However, it should be noted that many clients,

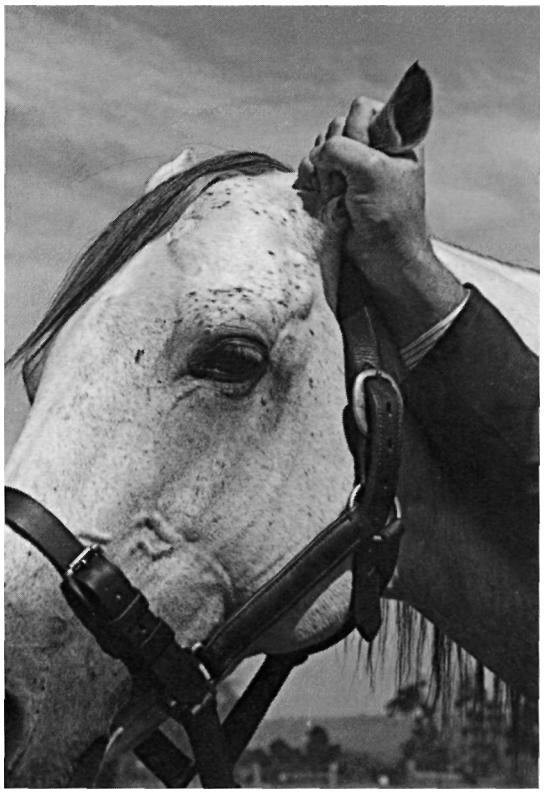


Figure 1-23. Twisting of an ear for restraint in a horse.

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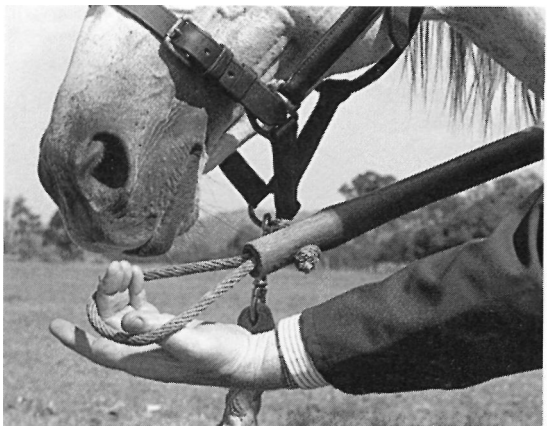


Figure 1-24. A rope twitch showing the position of the fingers in the twitch loop before grasping the upper lip.



Figure 1-26. Twitch applied to the upper lip showing half hitches around the twitch handle and position in which the handler should stand.

particularly those with pleasure horses, do not like to see their horses twitched, and therefore, the twitch should be used only where necessary. Once the twitch is applied, several half hitches should be made around the handle of the twitch using the lead rope, and the person holding the twitch should stand at the level of the shoulder (Fig. 1-26). In some cases, horses will try to strike when the twitch is applied or when a procedure such as stomach tubing is undertaken. In these horses, it may be helpful to use the horse's blanket, with the lining on the outer side, around the neck like a table napkin (Fig. 1-27). This not only quiets most horses but also prevents them from inflicting trauma to the examiner if a horse attempts to strike. The other useful restraint technique is the single sideline (Figs. 1-28 to 1-31). A single sideline will assist in restraint of difficult horses and may be useful for performing rectal examinations where stocks are not available and for performing standing castrations. Protection for the examining veterinarian also can be achieved if rectal examinations are performed around the door



Figure 1-25. Application of the twitch to the upper lip.

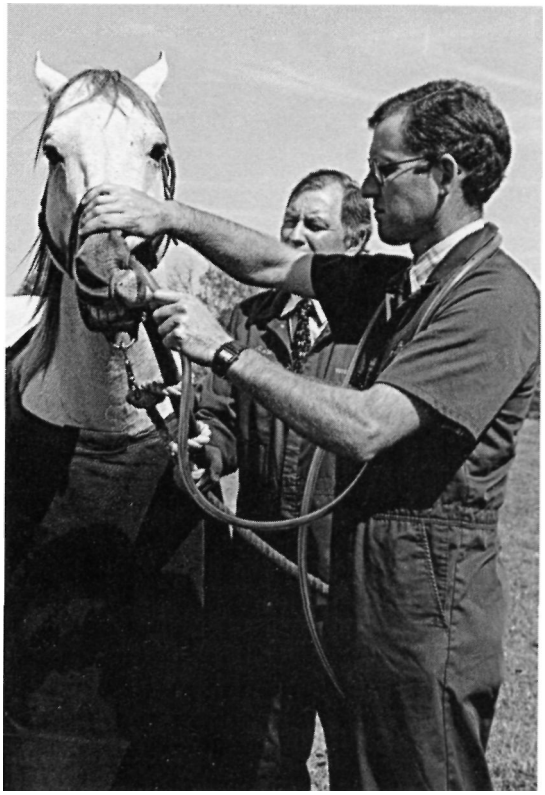


Figure 1-27. Demonstration of a horse's blanket being used to assist in preventing the horse from striking. The blanket is placed inside out and fastened around the horse's neck.

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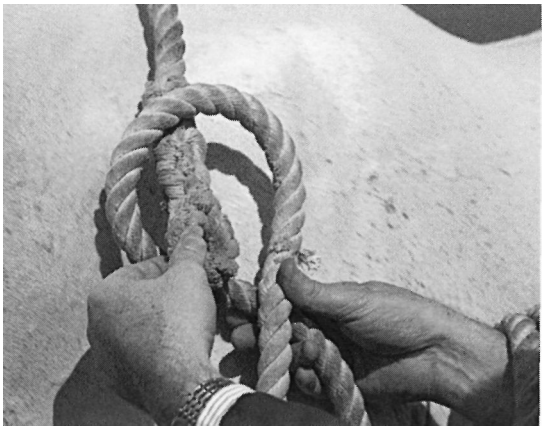


Figure 1-28. Beginning of a bowline knot for application of a single sideline. A loop is made in the rope through which the free end of the rope is passed.

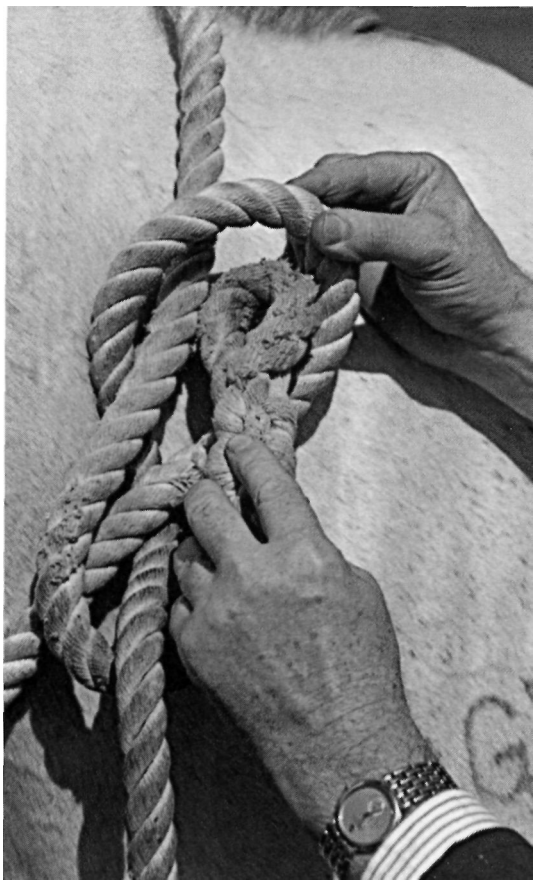


Figure 1-29. Completion of the bowline knot for a single sideline. The free end of the rope is passed through the loop, around the rope, and passed back into the loop.

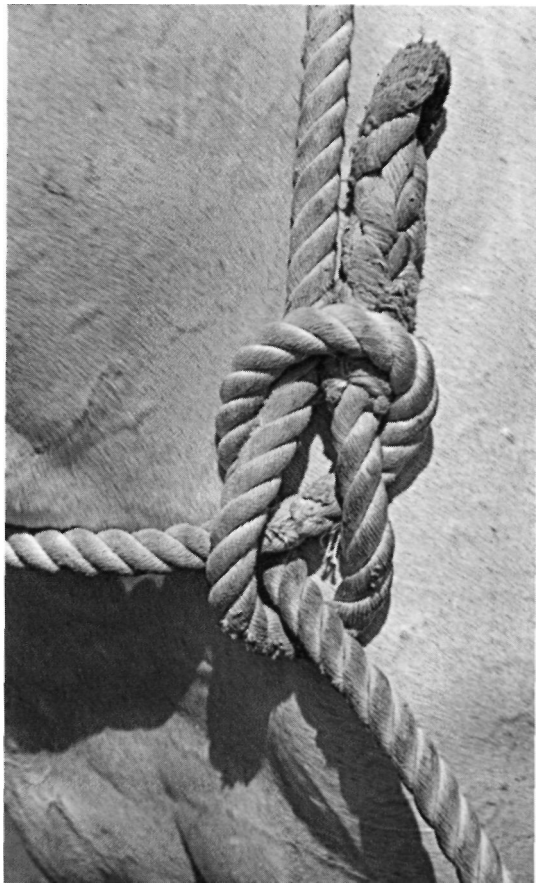


Figure 1-30. Bowline knot tied for a single sideline. This type of knot will not slip.

of a box stall or with the horse backed up to several bales of hay.

Other simple forms of restraint include covering the horse's eyes (Fig. 1-32), which may be useful in horses that are reactive to needles, and the use of a stallion chain placed over the dorsum

Text continued on page 23

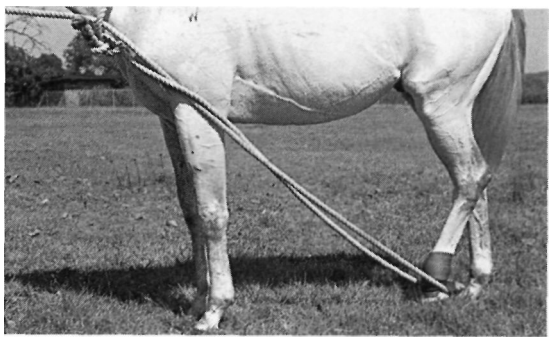


Figure 1-31. Single sideline applied for restraint.

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Figure 1-32. Covering the left eye to aid in restraint.

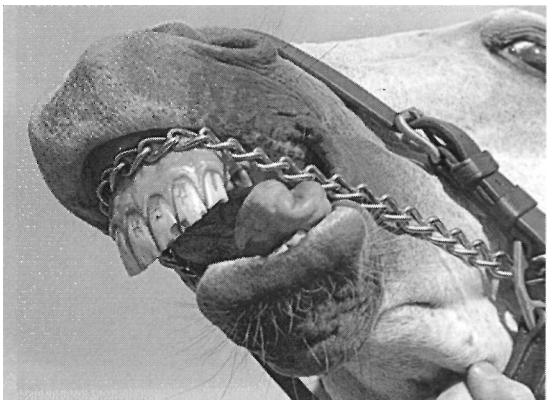


Figure 1-34. Use of a stallion chain applied under the top lip to aid in restraint.

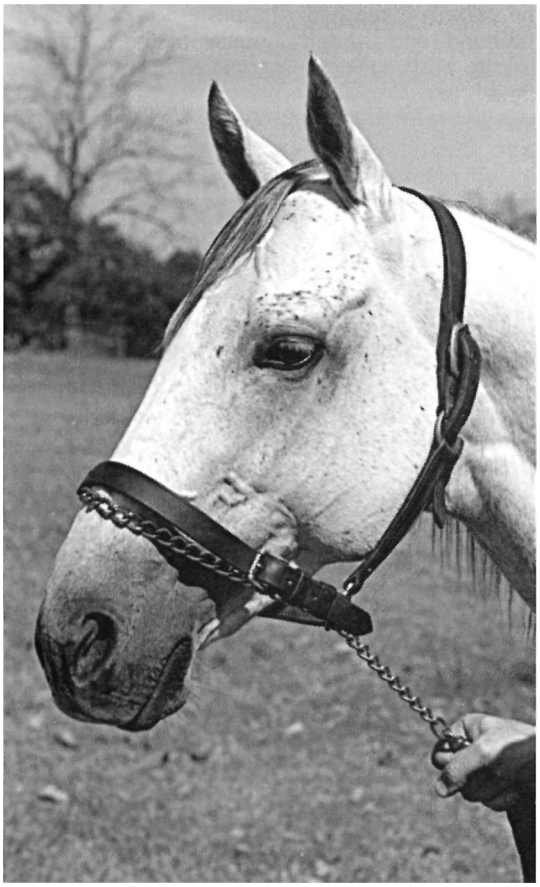


Figure 1-33. Use of a stallion chain applied over the dorsum of the nose to aid in restraint.

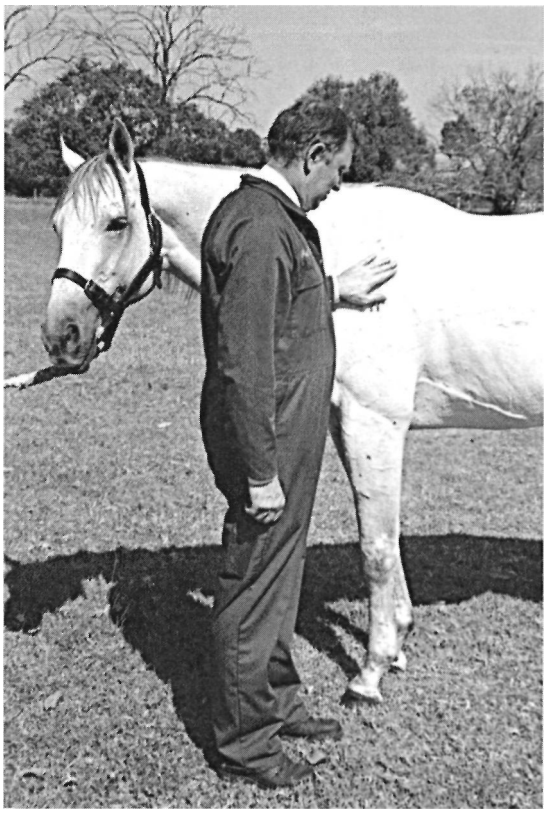


Figure 1-35. Commencing position for picking up the left foreleg with the examiner facing toward the rear of the horse.

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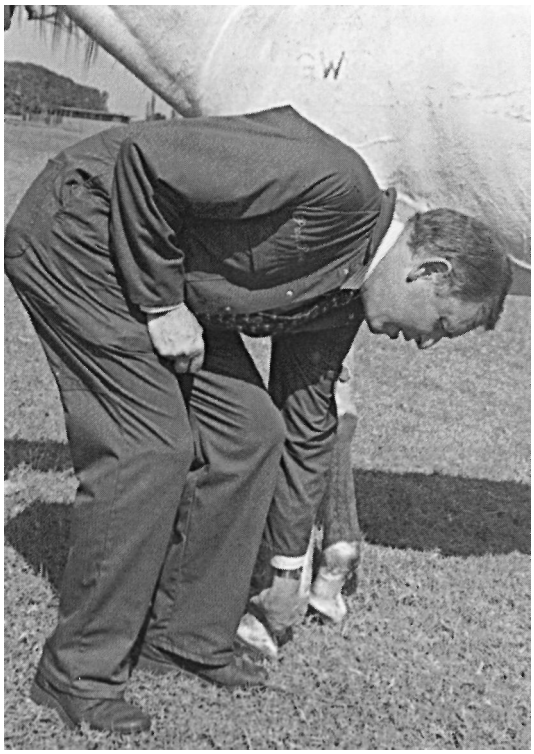


Figure 1-36. To pick up the left foreleg, the left hand is run distally down the leg until the area of the pastern is reached. The pastern is grasped with the hand, and the left shoulder of the examiner is used to push against the horse and shift weight off the left leg.

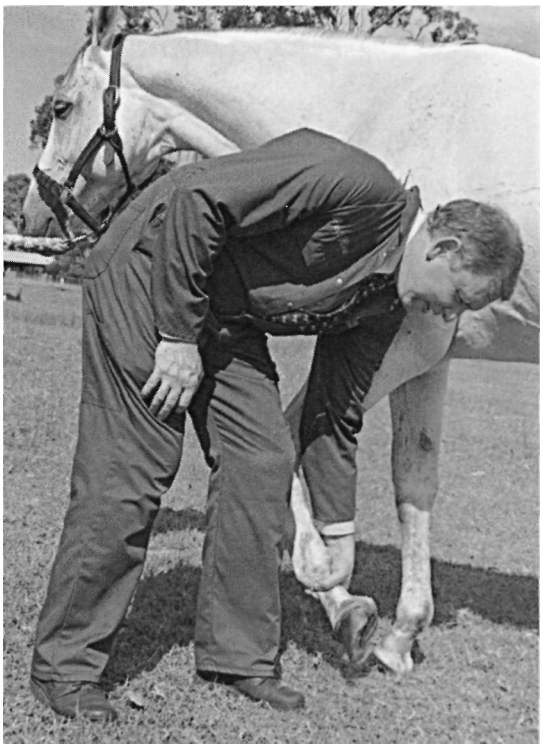


Figure 1-37. The left leg is picked up.

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Figure 1-38. To examine the horse's foot, it is held between the knees of the examiner. This is most easily done if the examiner's toes are pointed inward.

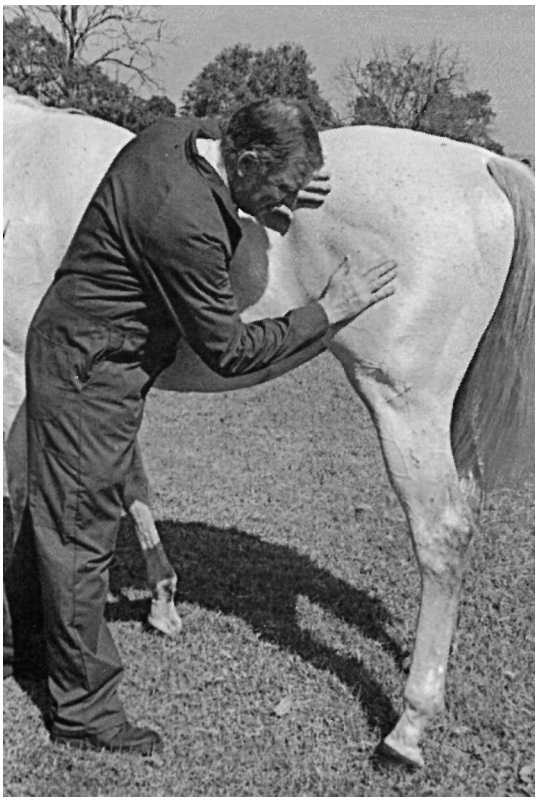


Figure 1-39. Commencing position for picking up the left hindleg.

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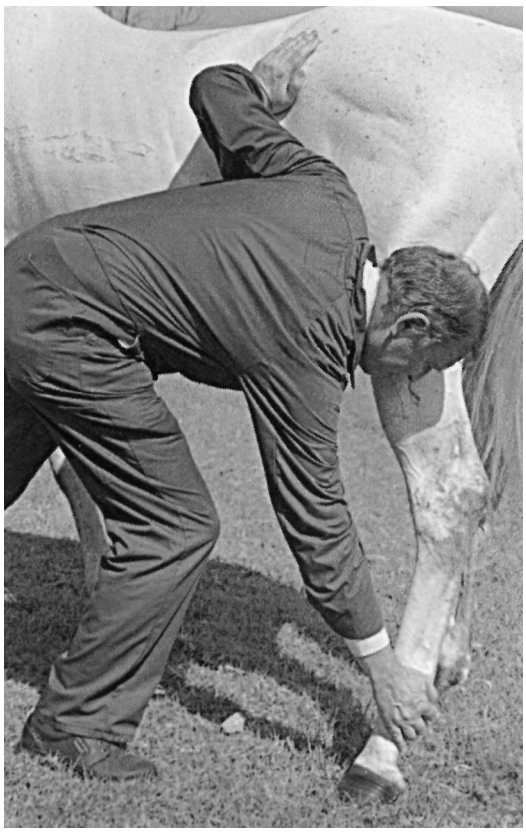


Figure 1-40. With the left hand placed on the tuber coxa, the right hand is run distally to the plantar aspect of the pastern.

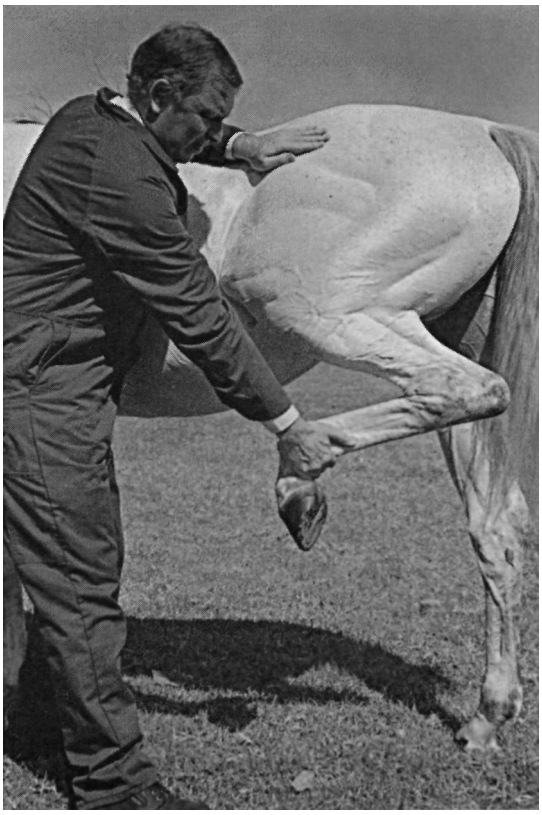


Figure 1-41. Using the left hand, weight is shifted onto the right hindleg of the horse and the left hindleg is pulled cranially.

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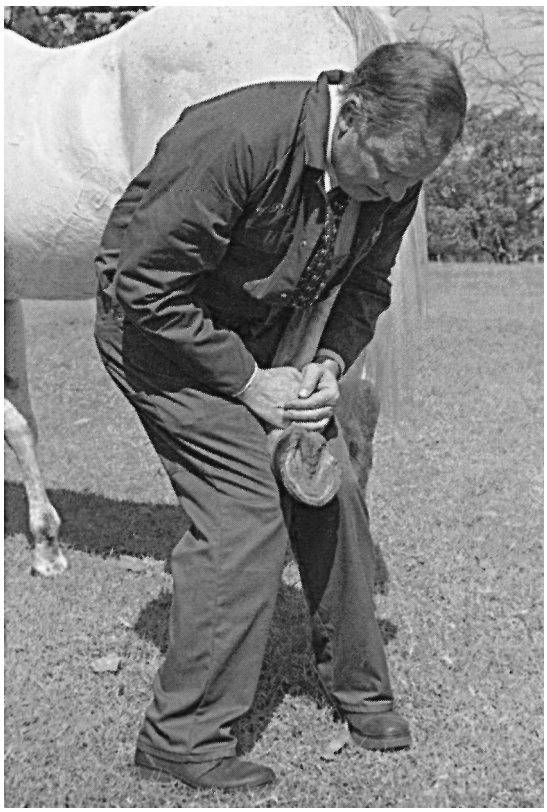


Figure 1-42. The examiner walks caudally, stepping between the hindlegs, so that the leg is moderately extended and the left hindfoot is rested on the knees.

of the nose (Fig. 1-33). With the latter technique, care must be taken to ensure that excessive tension is not placed on the lead but rather a short sharp jerk is given where needed. Because some horses object to a twitch, an alternative is the placement of a stallion chain under the top lip (Fig. 1-34). The chain is attached to the right side of the headstall and, after passing under the upper lip, is taken through the D ring on the headstall.

Lifting one of the forelegs or hindlegs may be useful to aid in restraint and must be undertaken to examine the limbs. The correct techniques for lifting the foreleg and hindleg are shown in Figures 1-35 to 1-42.

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CHAPTER 2

Protocols for Common Presenting Complaints

Reuben J. Rose and David R. Hodgson

Many problems encountered in equine practice have similar presentations despite different final diagnoses. A typical example is a horse with colic, where dysfunction in different organ systems can be expressed as abdominal pain.

KEY POINT

It is important when commencing an investigation of a problem to consider the signalment and history before a detailed physical examination. A list of differential diagnoses can then be made before further diagnostic tests or determination of the ultimate diagnosis.

This section lists some of the most common presenting complaints encountered in equine practice. Key diagnostic possibilities related to signalment (age, breed, use, and sex), important questions to consider when taking a history, and a range of differential diagnoses are presented. We designed this section to provide a framework that may be useful in dealing with a clinical problem. For each of the problems, this list of protocols is by no means complete or exhaustive but rather an attempt to consider the common issues in reaching a final diagnosis.

LIST OF PRESENTING PROBLEMS

Abdominal Pain, Colic (p. 25)
Alopecia (p. 27)
Anemia (p. 28)
Ataxia (p. 29)
Collapse (p. 30)
Coughing (p. 31)
Diarrhea (p. 32)
Dysphagia (p. 34)
Dyspnea (Resting) (p. 35)
Edema, Peripheral (p. 36)
Exercise Intolerance (p. 37)
Infertility, Mare (p. 38)
Infertility, Stallion (p. 39)
Lacrimation, Excessive and/or Photophobia (p. 40)
Lameness, Acute (p. 40)
Lameness, Chronic (p. 42)
Limb Swelling (p. 43)
Nasal Discharge (p. 44)
Pruritus (p. 45)
Respiratory Noise (p. 46)
Sudden Death (p. 47)
Urine Output Changes (p. 47)
Weight Loss or Failure to Thrive (p. 48)

ABDOMINAL PAIN, COLIC

SIGNALMENT

Age of the Horse

Parasites—young horses
Gastrointestinal ulcers—young horses
Intussusception—young horses
Large intestine impaction—older horses
Anterior enteritis—adult horses
Lipomas—older horses
Enteroliths—older horses
Epiploic foramen entrapment—young horses

Breed and Use

ATHLETIC/PERFORMANCE HORSES

Spasmodic colic

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Large intestine impaction
Anterior enteritis
Gastroduodenal ulcers
Small intestine volvulus and torsion
Pleuritis
Peritonitis
Colitis
Sand colic
Grain overload
Herniation of small intestine through the epiploic foramen
Entrapment of bowel over the nephrosplenic ligament

PLEASURE HORSES

Spasmodic colic
Impaction colic
Sand colic
Thromboembolic cranial mesenteric arteritis
Grain overload
Enteroliths
Cystic urolithiasis
Entrapment of bowel over the nephrosplenic ligament
Strangulating lipoma
Myopathies—e.g., "tying up"
Enteroliths
Abdominal tumors/abscessation
Cholelithiasis
Peritonitis
Pleuritis

FOALS

Retained meconium
Colitis
Gastroduodenal ulcers
Small intestine intussusception
Rupture of urinary bladder

Sex

Inguinal herniation of the small bowel—stallions
Large bowel torsion—postpartum mares
Uterine torsion—pregnant mares
Cystic urolithiasis—geldings and stallions
Retained meconium—male foals
Rupture of urinary bladder—male foals

HISTORY

How long has the pain been present?
Is this an acute onset or has the horse been slowly developing signs?
Is the horse straining?
Has the horse been rolling?
What is the horse's deworming history?
What medication has the horse received and at what dose?
What has the horse been eating and when did it last eat?
Has the diet of the horse changed recently?
Is the horse exposed to sand at pasture?
Have the teeth been checked recently?
Is the pain continuous or intermittent?
How long is it since feces were passed?
Are the feces normal in appearance and consistency?
Have there been any other recent episodes of abdominal pain?

PHYSICAL EXAMINATION

General Inspection

Attitude—signs of depressed mental status or normal
Behavior—pawing, attempting to roll, glancing at flanks, frequent urination, sweating
Abdominal distension

Detailed Inspection

Vital signs: heart rate (HR), respiratory rate (RR), temperature, pulse quality
Gut sounds—normal, increased, or decreased
Capillary refill time
Mucous membrane color
Temperature of extremities

DIAGNOSTIC PROCEDURES

Stomach Intubation

Presence/absence of excess fluid
Gastric reflux—spend 5 minutes to ensure that no reflux is present. Backfiush tube with water and try to siphon off fluid

Rectal Examination

Impaction of the large bowel
Displaced bowel
Distended small bowel loops
Tumors/abscesses
Check nephrosplenic space, inguinal canal (stallions)

Abdominocentesis

Fluid volume
Fluid color
Fluid turbidity
Presence of blood
Presence of ingesta

Clinical Pathology

Hematocrit and total serum/plasma protein
White cell count and differential
Venous or arterial acid-base status and/or plasma lactate determination
Abdominal fluid: cytology, total protein, bacteriology
Serum/plasma electrolyte values
Serum/plasma fibrinogen
Liver function: 7-glutamyl-transferase (GGT), L-iditol dehydrogenase (L-iDH), alkaline phosphatase (AP), bile acids

Ultrasound

Examination of the liver

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Examination of the kidneys
Examination of bowel
Examination of any abnormal structures detected on rectal examination
Examination of urachus, bladder, and abdominal contents in foals

DIFFERENTIAL DIAGNOSES

Spasmodic colic*
Large intestine impaction*
Anterior enteritis*
Large intestine displacement/torsion
Gastroduodenal ulcers
Grain overload
Sand colic (in regions with sandy soil type)
Small intestine volvulus and torsion
Small intestine intussusception
Peritonitis
Colitis
Herniation of small intestine through the epiploic foramen
Thromboembolic cranial mesenteric arteritis
Enteroliths
Fecoliths
Strangulating lipoma
Abdominal tumors
Abdominal abscesses
Cystic urolithiasis
Hepatitis
Cholelithiasis
Blunt trauma to abdomen
Pleuritis
Ruptured bladder (foals)
Rectal or reproductive tract perforation
Uterine torsion

ALOPECIA

SIGNALMENT

Age of the Horse

Seborrheic alopecia—older horses
Hormonal disturbances—older horses
Ringworm—young horses
Culicoides hypersensitivity—older horses

Breed and Use

Pemphigus foliaceus (Appaloosa)

Sex

No significant effect

HISTORY

How long has the problem been present?
Has the diet recently been changed?
Is pruritus associated with the alopecia?
Has the horse been given any treatment recently?
Has the alopecia been treated and with what?
What was the response to treatment?
Has the horse been washed with any shampoos or disinfectants?
Is the horse eating normally?
Is the horse losing weight?

PHYSICAL EXAMINATION

General Inspection

General body condition
Attitude—signs of depression
Note distribution of alopecia (where tack applied or where rubbing could have occurred)

Detailed Inspection

Vital signs: HR, RR, temperature, gut sounds, capillary refill time
Determine whether the lesions are localized or generalized
Note if any evidence of pruritus, erythema, exudate, crusting
Evidence of self-trauma
Examination of hair in a pruritic region and examination of skin with a hand lens

Clinical Pathology

Hemogram
Total plasma protein
Liver function tests: GGT, AP, SDH

Examination of the Skin

Skin scrapings
Impression smears
Intradermal skin testing
Acetate tape preparations
Bacterial/fungal cultures
Skin biopsies (histopathology, fluorescent antibody testing)

DIFFERENTIAL DIAGNOSES

Dermatophilosis*
Dermatophytosis*
Culicoides hypersensitivity
Cutaneous habronemiasis
Anhidrosis
Sarcoid
Pemphigus foliaceus/bullous pemphigoid
Seborrheic dermatosis
Equine linear keratosis
Cutaneous onchocerciasis
Ectoparasites (e.g., biting flies, lice, ticks, harvest mites or "chiggers," mosquitoes)
Oxyuriasis
Rubbing/self-trauma
Folliculitis/furunculosis
Urticaria due to inhaled allergens or drug administration

*Denotes most likely diagnosis.

*Denotes most likely diagnosis.

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Counter irritant
Burn
Contact dermatitis
Photosensitization, primary and secondary
Scratches ("grease heel")
Phycomycosis
Purpura
Neurologic diseases (e.g., rabies, polyneuritis equi, self-mutilation syndrome)

ANEMIA

SIGNALMENT
Age of the Horse
Neonatal isoerythrolysis—newborn foals
Equine infectious anemia—mature horses
Guttural pouch mycosis—mature horses
Piroplasmosis (babesiosis)—mature horses
Ehrlichiosis—mature horses
Breed and Use
ATHLETIC/PERFORMANCE HORSES
Anemia of chronic disease
Blood loss anemia
Gastrointestinal parasitism
Gastrointestinal ulcer disease
Nonsteroidal anti-inflammatory drug (NSAID) toxicity
Phlebotomy
Neoplasia
Vasculitis
Equine viral arteritis
PLEASURE HORSES
Anemia of chronic disease
Gastrointestinal parasitism
Nutritional anemia (starvation)
Blood loss anemia
Gastrointestinal ulcer disease
NSAID toxicity
Guttural pouch mycosis
Transfusion reactions
Equine infectious anemia
Neoplasia
Equine viral arteritis
Piroplasmosis
Leptospirosis
Chronic liver disease
Thrombocytopenia
Oxidant toxicosis (e.g., drugs, onions, red maple leaf)
Vasculitis (purpura hemorrhagica)
Disseminated intravascular coagulation
Coagulopathies
Dicoumarin poisoning (sweet clover)
Aplastic anemia
Autoimmune anemia
FOALS
Neonatal isoerythrolysis
Septicemia
Transfusion reactions
Aplastic anemia
Trauma
Hemophilia
Sex
No significant effect
HISTORY
Is there a history or evidence of hemorrhage or injury?
Is the horse receiving any medication?
Is the horse regularly dewormed?
Does the horse have evidence of intercurrent disease or malnutrition?
What are the constituents of the diet?
Has the horse been tested for piroplasmosis?
Is the horse housed in an area where viral or parasitic causes of anemia are common? If so, has the horse been infested with ticks?
What is the status of Coggins testing?
Is there evidence of exercise intolerance?
Are there other horses with similar signs?
PHYSICAL EXAMINATION
General Inspection
Note general condition of horse
Mental status of the horse
Evidence of edema
Detailed Examination
Vital signs: HR, RR, rectal temperature, mucous membrane color, capillary refill time, presence of icterus
Examination of mucous membranes for petechial or ecchymotic hemorrhage
Examine regional lymph nodes for enlargement
Examination of feces
Rectal examination
Examination of urine for hematuria
Examination for evidence of epistaxis
DIAGNOSTIC PROCEDURES
Clinical Pathology
Hemogram and fibrinogen
Thrombocyte count
Coagulation profile
Presence of Heinz bodies
Serum biochemistry: GGT, L-iDH, aspartate aminotransferase (AST), AP, creatinine, urea, electrolytes
Protein electrophoresis
Methemoglobin quantitation
Fecal occult blood determination
Blood smears for equine piroplasmosis and ehrlichiosis

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Abdominocentesis
Thoracocentesis

Bone Marrow Aspiration

Cytology
Myeloid:erythroid ratio

Serology

Coggins test
Coombs test
Equine viral arteritis titers and virus isolation
Equine piroplasmosis serology

Endoscopic Examination

Blood from the guttural pouch openings
Blood from the ethmoid region
Presence of blood in the trachea
Examination of the stomach

Ultrasound

Ultrasound of thorax and abdomen for masses or free fluid

DIFFERENTIAL DIAGNOSES

Anemia of chronic disease*
Gastrointestinal parasitism*
Blood loss anemia
Nutritional (starvation)
NSAID toxicity
Aplastic anemia
Autoimmune anemia
Equine infectious anemia
Gastrointestinal ulcer disease
Neoplasia
Coagulopathies (e.g., disseminated intravascular coagulation)
Piroplasmosis
Leptospirosis
Chronic liver disease
Thrombocytopenia
Oxidant toxicosis (e.g., drugs, onions, red maple leaf)
Vasculitis
Dicoumarin poisoning (sweet clover)
Guttural pouch mycosis
Neonatal isoerythrolysis
Septicemia
Transfusion reactions

ATAXIA

SIGNALMENT

Age of the Horse

Cerebellar abiotrophy—foals
Neonatal maladjustment syndrome—foals
Septicemia—foals
Cervical stenotic myelopathy—young horses
Viral encephalomyelitides—mature horses
Protozoal myeloencephalitis—mature horses
Atlantooccipital malformation—young horses (Arabians)
Equine degenerative myeloencephalopathy—young horses
Idiopathic vestibular syndrome—mature horses
Otitis media/interna—mature horses
Rabies—mature horses
Vertebral osteomyelitis—foals
Arabian idiopathic epilepsy—foals

Breed and Use

ATHLETIC/PERFORMANCE HORSES

Cervical stenotic myelopathy
Head/spinal cord trauma
Protozoal myeloencephalitis
Herpesvirus myeloencephalitis
Idiopathic vestibular syndrome
Viral encephalomyelitides
Equine degenerative myeloencephalopathy
Rabies
Otitis media/interna
Botulism

PLEASURE HORSES

Cervical stenotic myelopathy
Head/spinal cord trauma
Protozoal myeloencephalitis
Herpesvirus myeloencephalitis
Idiopathic vestibular syndrome
Viral encephalomyelitides
Equine degenerative myeloencephalopathy
Rabies
Neuritis of the cauda equina
Hepatoencephalopathy
Lead poisoning
Botulism
Organophosphate toxicosis
Otitis media/interna
Locoweed toxicosis
White snakeroot poisoning
Narcolepsy/epilepsy
Verminous myelitis
Ivermectin toxicosis
Leucoencephalomalacia
Hypocalcemia
Cerebellar abiotrophy (Arabians)
Atlantooccipital malformation (Arabians)

FOALS

Head/spinal cord trauma
Neonatal maladjustment syndrome
Septicemia
Idiopathic epilepsy (Arabians)
Vertebral osteomyelitis
Toxoinfectious botulism

*Denotes most likely diagnosis.

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Sex
Epilepsy (mares), increased incidence when in estrus

HISTORY
How long have the signs been present?
Are there other neurologic signs?
Is there a history of trauma?
Is there a history of intercurrent infectious disease?
Are other horses affected?
Has the horse's diet altered recently?
Are the signs progressive?
Were the signs sudden or insidious in onset?
Has the horse received any medication? *(Note:* Ivermectin administration)
Is the horse insured?

PHYSICAL EXAMINATION

General Inspection
Note mental status and behavior
Signs of trauma
Signs of asymmetry

Detailed Examination
Vital signs: HR, RR, temperature, capillary refill time, gut sounds
Neurologic examination
Palpation of the neck and back
Laryngeal adductor ("slap") test by palpation of the larynx

Diagnostic Procedures
Endoscopy of nasopharynx, larynx, and trachea
Laryngeal adductor ("slap") test
Radiography of head and neck
Myelography
Cerebrospinal fluid (CSF) collection

Clinical Pathology
Hemogram and fibrinogen
Liver enzymes
CSF: cytology, protein, color, Western blot for protozoal myeloencephalitis
Serology
Eastern equine encephalomyelitis (EEE), western equine encephalomyelitis (WEE), herpesvirus

DIFFERENTIAL DIAGNOSES
Head/spinal cord trauma*
Cervical stenotic myelopathy*
Protozoal myeloencephalitis*
Herpesvirus myeloencephalitis*
Viral encephalomyelitides
Equine degenerative myeloencephalopathy
Hepatoencephalopathy
Idiopathic vestibular syndrome
Lead poisoning
Botulism
Organophosphate toxicosis
Rabies
Otitis media/interna
Neuritis of the cauda equina
Locoweed toxicosis
White snakeroot poisoning
Narcolepsy/epilepsy
Neonatal maladjustment syndrome
Septicemia
Idiopathic epilepsy (Arabians)
Vertebral osteomyelitis
Toxoinfectious botulism
Verminous myelitis
Idiopathic polyneuropathy

*Denotes most likely diagnosis.

COLLAPSE

SIGNALMENT

Age of the Horse
Congenital cardiovascular disorders—foals
Acquired cardiovascular disease—mature horses
Hepatoencephalopathy—mature horses
Idiopathic convulsive syndrome—foals
Rabies—mature horses

Breed and Use

ATHLETIC/PERFORMANCE HORSES
Ruptured aorta or pulmonary artery
Trauma
Acquired cardiovascular disease
Seizure disorders
Hyperkalemic periodic paralysis (quarter horses)
Hypocalcemia
Inadvertent intracarotid injection of medications

PLEASURE HORSES
Cardiovascular disease
Seizure disorders (e.g., epilepsy, convulsions)
Narcolepsy
Hyperkalemic periodic paralysis (Quarter horses)
Trauma
Hypocalcemia
Hyperparathyroidism
Inadvertent intracarotid injection of medications

FOALS
Congenital cardiac disease
Narcolepsy
Idiopathic epilepsy (Arabians)
Neonatal maladjustment syndrome
Septicemia
Trauma

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Sex
- Mares are more commonly affected by epilepsy when in estrus
- Ruptured aorta more common in stallions

HISTORY
- How long have the signs been present?
- Is the collapse related to exercise or excitement?
- Is the collapse related to feeding?
- Are signs exacerbated by external stimuli (e.g., noise, light)?
- Is the collapse a regular or isolated event?
- Is there a recent history of trauma?
- Are there any warning signs before collapse?
- Does the horse show signs of distress before or after collapse?
- Are siblings or close relatives affected similarly?
- Are signs exacerbated by administration of phenothiazine tranquilizers?

PHYSICAL EXAMINATION

General Inspection
- Behavior of the horse
- Mental status of the horse
- Signs of trauma
- Head posture

Detailed Examination
- Vital signs: HR, RR, temperature, capillary refill time, evidence of jugular pulse or edema
- Presence of icterus
- Auscultation of the heart
- Neurologic examination

DIAGNOSTIC PROCEDURES

Clinical Pathology
- Hemogram, fibrinogen, electrolytes, and ionized calcium
- Liver enzymes

Electrocardiography
- Conduction disturbances
- Dysrhythmias

Ultrasound
- Chamber enlargement
- Valvular lesions
- Papillary muscle rupture

Hyperkalemic Periodic Paralysis Challenge Test
- Potassium chloride by stomach tube
- Genetic testing

DIFFERENTIAL DIAGNOSES

Adults
- Ruptured aorta or pulmonary artery (death)*
- Trauma*
- Acquired cardiovascular disease
- Seizure disorders
- Narcolepsy
- Inadvertent intracarotid injection of medications
- Heatstroke
- Hypocalcemia
- Hyperparathyroidism
- Idiopathic syncope

Foals
- Congenital cardiac disease*
- Narcolepsy*
- Idiopathic epilepsy (Arabians)*
- Neonatal maladjustment syndrome
- Septicemia
- Trauma

*Denotes most likely diagnosis.

COUGHING

SIGNALMENT

Age of the Horse
- *Rhodococcus equi* infections—foals
- Pneumonia—foals
- Viral respiratory disease—young horses
- Strangles infections—young horses
- Allergic respiratory disease (chronic obstructive pulmonary disease [COPD])—mature horses
- Upper respiratory problems—young horses

Breed and Use

ATHLETIC/PERFORMANCE HORSES
- Viral respiratory disease
- Bacterial pneumonia/pleuritis
- Upper respiratory tract problems
- Strangles
- Inflammatory (reactive) lower airway disease
- Exercise-induced pulmonary hemorrhage (EIPH) ("bleeders")
- Pharyngitis
- Tracheitis secondary to laryngoplasty surgery

PLEASURE HORSES
- Strangles
- Viral respiratory disease
- Bacterial bronchitis/pneumonia/pleuritis
- Allergic respiratory disease (COPD)
- Inflammatory (reactive) lower airway disease
- Lungworm

FOALS
- *Rhodococcus equi* infections
- Adenovirus (Arabians)
- Viral respiratory disease

*Denotes most likely diagnosis.

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Bacterial bronchitis/pneumonia
Parasites (roundworm infestations)

Sex

No significant effect

HISTORY

How long has the horse been coughing?
How would the cough be described?
Has the horse been dewormed recently?
Has there been a nasal discharge at any stage?
Is more than one horse in the stable affected?
Is the cough related to feeding?
Is the cough related to exercise?
Is the cough related to stabling?
Is the horse eating and drinking normally?
Has the horse lost weight?
Are there signs of systemic disease (e.g., fever)?
Has the horse been in contact with any donkeys?
Has the horse been vaccinated?

PHYSICAL EXAMINATION

General Inspection

Presence of nasal discharge
Lymph node enlargement
Odor of breath
Character of respiration
General body condition

Detailed Examination

Sinus percussion
Retropharyngeal lymph node palpation
Palpation of larynx (muscular process)
Auscultation of the chest, including rebreathing
Respiratory frequency

DIAGNOSTIC PROCEDURES

Transtracheal Aspiration

Cytologic examination
Bacteriologic examination

Bronchoalveolar Lavage

Cytologic examination

Endoscopic Examination

Rhinolaryngoscopy
Examination of the trachea to level of tracheal bifurcation

Radiography

Head, sinuses, and larynx, if upper airway problem
Chest

Ultrasound

Examination of thorax

Arterial Blood Gas Analysis

PaO_2 determination

Hematology

Total and differential white cell count
Plasma fibrinogen

DIFFERENTIAL DIAGNOSES

Viral respiratory tract infection*
Pharyngitis/tracheitis*
Allergic respiratory disease (COPD)*
Pneumonia/pleuritis, bacterial
Inflammatory (reactive) airway disease
EIPH
Inadvertent intratracheal administration of medication
Rhodococcus equi infection—foals
Streptococcal pneumonia—foals
Lungworm
Epiglottic entrapment
Previous laryngoplasty surgery
Parasitic infestation (e.g., roundworms in foals)
Strangles

DIARRHEA

SIGNALMENT

Age of the Horse

Gastrointestinal parasites—young horses
"Foal heat" diarrhea—foals 7 to 14 days old
Rotavirus diarrhea
Gastrointestinal ulcers—young horses
Intussusception—young horses
Salmonellosis—young horses
Clostridial diarrhea—young horses
Inflammatory bowel disease—older horses
Neoplasia—older horses
Nutritional—foals
Colitis—older horses
Equine monocytic ehrlichiosis ("Potomac fever")—older horses

Breed and Use

ATHLETIC/PERFORMANCE HORSES

Idiopathic diarrhea
Drug-induced diarrhea (e.g., NSAIDs, antibiotics)
Stress-induced diarrhea
Salmonellosis
Gastroduodenal ulcers
Colitis
Equine monocytic ehrlichiosis ("Potomac fever")
Intestinal clostridiosis
Chronic liver disease
Strongylosis
Toxemia
Peritonitis

PLEASURE HORSES

Idiopathic diarrhea

*Denotes most likely diagnosis.

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Drug-induced diarrhea (e.g., NSAIDs, antibiotics)
Stress-induced diarrhea
Salmonellosis
Gastroduodenal ulcers
Colitis
Equine monocytic ehrlichiosis ("Potomac fever")
Intestinal clostridiosis
Chronic liver disease
Strongylosis
Heavy-metal intoxication
"Blister beetle" intoxication
Fungal diarrhea
Peritonitis

FOALS

"Foal heat" diarrhea
Rotavirus
Gastroduodenal ulcers
Small intestine intussusception
Strongyloides westeri infestation
Parascaris equorum **(roundworm)** infestation
Bacterial septicemia/gastrointestinal tract **infection:** *Salmonella, Escherichia coli, Clostridium, R. equi*
Failure of passive transfer of immunity
Nutritional diarrhea

Sex

No significant effect

HISTORY

How long has the diarrhea been present?
Is the horse drinking and if so, how much?
Have there been dramatic changes in fecal consistency?
Is the horse regularly dewormed?
Are there other horses in contact that are affected with diarrhea?
Have there been any stressful events (e.g., transport) associated with diarrhea onset?
Has the horse been undergoing any drug treatment, particularly antibiotics or NSAIDs?
Has the horse had any change in its diet recently?

PHYSICAL EXAMINATION

General Inspection

Attitude—signs of depression
Frequency and volume of diarrhea
Consistency and composition of feces
Signs of pain

Detailed Inspection

Vital signs: HR, RR, temperature, pulse quality
Gut sounds
Capillary refill time
Mucous membrane color
Skin turgor
Temperature of extremities

DIAGNOSTIC PROCEDURES

Clinical Pathology

Hematocrit and total serum/plasma protein
Leucocyte count and differential leucocyte count
Venous or arterial acid-base status
Serum/plasma electrolytes, osmolality
Plasma/serum fibrinogen
Liver enzymes: GGT, AP, AST, total bile acids
Abdominal fluid analysis: total protein, leucocytes, cytology, bacteriology
Urinary cantharidin concentration

Fluid Balance

Weight of horse
Estimation of fluid loss: total plasma protein (TPP), packed cell volume (PCV), osmolality, skin turgor, capillary refill

Fecal Examination

Fecal volume estimation
Fecal occult blood
Fecal bacteriology: cultures over several days necessary for *Salmonella* (results improved if combined with culture of rectal mucosal biopsy)
Parasite ova
Fecal mycology
Examination for sand

Nutritional Examination

Heavy-metal measurement in feed samples
History of eating off sandy soil
Blister beetles

Rectal Examination

Abnormal masses
Distended bowel
Areas of localized pain

Abdominocentesis

Fluid color
Fluid turbidity
Presence of blood

Small Intestinal Absorption Tests

D-Glucose
D-Xylose

DIFFERENTIAL DIAGNOSES

Adult Horses

Idiopathic*
Cyathostoma infestations*
Stress-induced diarrhea*
Gastroduodenal ulcers
Salmonellosis
Drug-induced diarrhea (e.g., phenylbutazone, antibiotics)
Colitis X

*Denotes most likely diagnosis.

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Equine monocytic ehrlichiosis ("Potomac fever")
Intestinal clostridiosis
Chronic liver disease
Strongylosis
Toxemia
Heavy-metal intoxication
"Blister beetle" intoxication
Fungal diarrhea
Nutritional diarrhea
Postimpaction diarrhea
Peritonitis

Foals

Idiopathic*
Cyathostoma infestations*
"Foal heat" diarrhea*
Rotavirus
Small intestine intussusception
Strongyloides westeri infestation
Parascaris equorum (roundworm) infestation
Bacterial septicemia
Salmonellosis
Failure of passive transfer of immunity
Nutritional diarrhea
Drug-induced diarrhea (e.g., phenylbutazone, antibiotics)

DYSPHAGIA

SIGNALMENT

Age of the Horse

Cleft palate—foals
Guttural pouch tympany—foals
Botulism—older horses
Choke—older horses
Encephalomyelitis—older horses
Guttural pouch mycosis—older horses
Strangles—young horses
Rabies—older horses
Toxoinfectious botulism—foals (Shaker foals)
Septicemia—foals
Neonatal maladjustment syndrome—foals

Breed and Use

ATHLETIC/PERFORMANCE HORSES

Choke
Strangles
Pharyngeal paralysis
Postlaryngoplasty/laryngeal surgery
Teeth problems
Esophageal stricture
Head trauma
Guttural pouch disease
Equine protozoal myeloencephalitis
Viral encephalomyelitis (EEE and WEE)

PLEASURE HORSES

Choke
Strangles
Pharyngeal paralysis
Teeth problems
Esophageal stricture
Head trauma
Grass sickness
Botulism
Rabies
Lead poisoning
Guttural pouch disease
Hepatoencephalopathy
Equine protozoal myeloencephalitis
Viral encephalomyelitis (EEE and WEE)
Hyperkalemic periodic paralysis (Quarter horses)

FOALS

Cleft palate
Neonatal maladjustment syndrome
Guttural pouch tympany
Head trauma
Septicemia with central nervous system signs
Liver failure/hepatoencephalopathy
Toxoinfectious botulism (Shaker foals)
Haloxon toxicity

Sex

No significant effect

HISTORY

How long have the signs been present?
Is there a known history of trauma?
Are any other horses affected?
Is there a recent change in diet?
Is there any evidence of feed contamination?
Is the horse attempting to eat?
Are the signs progressive?
Does the horse have access to yellow star thistle or Russian knapweed at pasture?
Has the horse been vaccinated against viral encephalomyelitides?
Are there other signs of neurologic dysfunction?
Is there saliva, pus, or blood coming from the mouth or nose?
Has the horse received any medication or other treatment?

PHYSICAL EXAMINATION

General Inspection

Vital signs: HR, RR, temperature, capillary refill, gut sounds
Evidence of salivation
Demeanor
Skin turgor
Presence of feed, pus, or blood at the nose

*Denotes most likely diagnosis.

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Detailed Examination
- Palpation of the throat and esophagus
- Cranial nerve examination
- General neurologic examination
- Chest auscultation for evidence of aspiration pneumonia

DIAGNOSTIC PROCEDURES

Nasogastric Intubation
- Determination of any obstruction sites

Radiography of the Head and Neck
- Plain radiographs
- Contrast radiography (e.g., barium)

Clinical Pathology
- PCV and plasma or serum total protein
- Fibrinogen
- Electrolytes and osmolality
- Plasma urea nitrogen and creatinine
- Liver enzymes/biochemistry: GGT, AP, total bile acids
- Tissue or body fluid lead (consult laboratory for appropriate test)

Endoscopic Examination
- Examination of pharynx and larynx
- Examination of soft palate
- Examination of guttural pouch openings
- Examination of nasopharynx during attempted swallowing
- Examination of the esophagus

CSF Examination
- Color
- Cytology
- Protein
- Western blot test for protozoal encephalomyelitis

Serology
- Titers for EEE and WEE

Fluorescent Antibody Detection for Rabies
- Skin, brain (consult laboratory)

DIFFERENTIAL DIAGNOSES
- Choke*
- Teeth problems*
- Strangles
- Pharyngeal paralysis
- Hepatoencephalopathy
- Esophageal stricture
- Postlaryngoplasty/laryngeal surgery
- Head trauma
- Grass sickness
- Botulism
- Guttural pouch disease
- Nigropallidal encephalomalacia (e.g., yellow star thistle, Russian knapweed poisoning)
- Equine protozoal myeloencephalitis
- Viral encephalomyelitis (EEE and WEE)
- Rabies
- Lead poisoning

*Denotes most likely diagnosis.

DYSPNEA (RESTING)

SIGNALMENT

Age of the Horse
- Viral respiratory disease—young horses
- Bacterial respiratory disease—young horses
- Nasal and thoracic tumors—older horses
- Allergic respiratory disease (COPD)—older horses

Breed and Use

ATHLETIC/PERFORMANCE HORSES
- Viral respiratory disease
- Strangles
- Bacterial pneumonia/pleuritis/bronchitis
- Allergic respiratory disease (COPD)
- Nasal tumors

PLEASURE HORSES
- Viral respiratory disease
- Strangles
- Bacterial pneumonia/pleuritis/bronchitis
- Chronic allergic bronchitis (COPD)
- Nasal tumors
- Progressive hematoma of the ethmoid
- Thoracic tumors

FOALS
- Bacterial pneumonia
- Guttural pouch tympany
- Congenital malformations of the upper airway

Sex
- No significant effect

HISTORY
- Is there a history of nasal discharge?
- Does the horse have a cough?
- If the dyspnea is apparent at rest, is it related to the horse being housed?
- Is the dyspnea seasonal?
- Is the dyspnea worsened by feeding?

PHYSICAL EXAMINATION

General Inspection
- Examination of the nares for discharge (uni/bilateral)
- Evidence of systemic disease (e.g., weight loss)
- Examination of the head and upper respiratory tract for swellings

Detailed Examination
- Percussion of the sinuses
- Look in the nose
- Oral examination to determine presence of tooth problems
- Auscultation of the thorax
- Smell the horse's breath
- Palpation over the guttural pouches
- Palpation of the trachea
- Examination of the lymph nodes

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DIAGNOSTIC PROCEDURES
- Rhinolaryngoscopy
- Examination of nasal conchae (turbinates)
- Guttural pouch openings
- Examination of guttural pouches
- Ethmoid region
- Pharynx
- Larynx and trachea

Radiography
- Sinuses
- Guttural pouches
- Nasal conchae
- Ethmoid
- Tooth roots
- Chest

Ultrasound
- Chest

Bacteriology
- Transtracheal aspirate
- Guttural pouch aspirate
- Bronchoalveolar lavage

Hematology and Biochemistry
- White cell count and differential
- Fibrinogen
- Red cell count
- Protein electrophoresis
- General biochemical profile

DIFFERENTIAL DIAGNOSES
- Viral or bacterial lower respiratory tract disease*
- Chronic allergic bronchitis (COPD)*
- Pleural effusion*
- Interstitial pneumonia
- Tooth-root tumors
- Nasal tumors
- Endotoxemia
- Partial upper airway obstructions
- Guttural pouch tympany
- Progressive hematoma of the ethmoid
- Heart failure
- Liver failure
- Anaphylaxis
- Bronchospasm

EDEMA, PERIPHERAL

SIGNALMENT

Age of the Horse
- Septicemia—young horses
- Congestive heart failure—older horses
- Vasculitis—older horses
- Pleuropneumonia—older horses
- *Rhodococcus equi* infection—young horses

Breed and Use
- No significant effect

Sex
- No significant effect

HISTORY
- When was the swelling/edema first noticed?
- Is the edema becoming more evident?
- Is the horse demonstrating evidence of pain associated with the edema?
- Are there other systemic signs associated with the edema (e.g., petechial hemorrhages)?
- Is the edema associated with the recent administration of any medications?
- Is the horse showing signs of depression?
- Is the edema initiated or exacerbated when the horse is confined?
- Is the appetite normal?
- Is the horse losing weight?
- Has there been a recent change in the diet?
- Does the horse have normal feces?
- Does the horse drink and urinate normal volumes?
- Has the horse received any treatment for the edema and if so what effect has it produced?

PHYSICAL EXAMINATION

General Inspection
- Note the distribution of the edema
- Is there evidence of systemic disease (e.g., weight loss, diarrhea, or nasal discharge)?
- Determine whether the edema is pitting
- General body condition
- Attitude—signs of depression or bright

Detailed Inspection
- Vital signs: HR, RR, temperature, capillary refill time, presence or absence of icterus
- Check if the swelling is hot, painful, or pitting on palpation
- Examine the horse for lameness
- Check mucous membranes for petechiae
- Detailed auscultation of the chest
- Rectal examination

DIAGNOSTIC PROCEDURES

Clinical Pathology
- Complete blood count (CBC) and differential white blood cell count, fibrinogen, electrolytes
- Total plasma protein and protein electrophoresis
- Fecal egg count
- Liver enzymes, bilirubin, serum bile acid concentration
- Creatinine and urea nitrogen
- Urinalysis

*Denotes most likely diagnosis.

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Fractional electrolyte excretions
Coombs test
Coggins test
Equine viral arteritis testing
Equine ehrlichiosis testing
Abdominocentesis (if indicated)
Thoracocentesis (if indicated)
Acid-base status

Bacteriology

Fecal culture/identification of *Salmonella* spp. and *Clostridia* spp.
Culture of thoracic or abdominal fluid (if indicated)
Blood culture (septicemic foals)
Transtracheal aspirate

Ultrasound and Radiology

Echocardiography
Thoracic examination for presence of fluid or pulmonary lesions
Ultrasonography of the abdomen
Radiography of the chest

Gastrointestinal Studies

Oral D-glucose absorption test
Oral D-xylose absorption test
Liver biopsy

DIFFERENTIAL DIAGNOSES

Lymphatic obstructions*
Trauma*
Cellulitis*
Parasitism
Drug reaction (eruption)
Primary hypersensitivity (e.g., urticaria)
Secondary hypersensitivity (e.g., neoplasia)
Erythema multiforme
Vasculitis
Protein-losing enteropathy
Liver failure
Phenylbutazone toxicity
Heart failure
Glomerulonephritis
Pleuropneumonia
Confinement
Equine ehrlichiosis
Chronic abscessation
Rhodococcus equi infection (foals)
Angioedema

EXERCISE INTOLERANCE

SIGNALMENT

Age of the Horse

Congenital cardiovascular anomalies—foals
Neonatal isoerythrolysis—foals
Vitamin E/selenium deficiency—foals
COPD—mature horses

Breed and Use

ATHLETIC/PERFORMANCE HORSES

Lameness
Infectious respiratory disease
Subclinical respiratory disease
EIPH
Laryngeal hemiplegia and upper airway disorders
Recurrent rhabdomyolysis
Cardiovascular disease
Paroxysmal atrial fibrillation
Anemia
Neurologic disease

PLEASURE HORSES

Lameness
Infectious respiratory disease
COPD
Cardiovascular disease
Recurrent rhabdomyolysis
Hyperkalemic periodic paralysis (Quarter horses)
Anemia
Neurologic disease
Parasitic infections
Malnutrition
Liver or renal disease

FOALS

Vitamin E/selenium deficiency
Congenital heart disease
Infectious respiratory disease
Septicemia

Sex

No significant effect

HISTORY

Is there evidence of intercurrent disease?
Does the horse make a respiratory noise?
Is the horse distressed after exercise?
Does the horse have a cough?
Is there a history of EIPH?
Is there a nasal discharge?
Has any lameness been noted?
If a racehorse, is there a decrease in performance on previous race starts or has the horse never performed up to expectation?
Is there a history of trauma?
If the horse is racehorse, is the decrease in performance abrupt during a race?

PHYSICAL EXAMINATION

General Inspection

Note general condition of horse
Mental status of the horse
Presence of jugular pulse
Presence of cough
Respiratory stridor

*Denotes most likely diagnosis.

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Detailed Examination
- Vital signs: HR, RR, temperature
- Auscultation of thorax, including rebreathing
- Percussion of the thorax
- Evidence of dysrhythmia
- Lameness examination
- Neurologic examination
- Palpation of regional lymph nodes
- Palpation of muscular process of arytenoid cartilage
- Laryngeal adductor ("slap") test

DIAGNOSTIC PROCEDURES

Clinical Pathology
- CBC and fibrinogen
- Plasma or serum biochemistry: AST, GGT, creatine phosphokinase, electrolytes, urea nitrogen, creatinine
- Glutathione peroxidase activity, serum vitamin E concentration

Endoscopy
- Examination of upper airway at rest and during exercise
- Examination of trachea, presence of mucopus and blood

Bronchoalveolar Lavage and/or Transtracheal Aspiration
- Cytology
- Bacteriology

Electrocardiography
- Atrial fibrillation
- Major conduction disturbances
- Presence of ventricular premature contractions

Arterial Blood Gas Analysis
- Evidence of hypoxemia and/or hypercapnia during exercise

Radiography
- As indicated by lameness and/or neurologic examinations

Serology
- Respiratory viruses
- Coggins test
- Western blot test for protozoal myeloencephalitis

Treadmill Exercise Testing
- Telemetry electrocardiography
- Blood lactate measurements
- Oxygen uptake measurements
- Blood volume determination
- Arterial blood gas measurements during exercise

DIFFERENTIAL DIAGNOSES
- Lameness (clinical and subclinical) and back disorders*
- Infectious respiratory disease (clinical and subclinical)*
- Noninfectious lower respiratory tract disease*
- Inadequate fitness*
- Laryngeal hemiplegia and other upper airway disorders
- Recurrent rhabdomyolysis
- Acquired cardiovascular disease (e.g., paroxysmal atrial fibrillation)
- Infectious disease with systemic manifestations
- EIPH
- Anemia
- Neurologic disease (e.g., cervical stenotic myelopathy, protozoal myeloencephalitis)
- Parasitic infections
- Malnutrition
- Vitamin E/selenium deficiency
- Congenital heart disease
- Liver disease
- Renal disease

*Denotes most likely diagnosis.

INFERTILITY, MARE

SIGNALMENT

Age of the Horse
- Ovarian atrophy—older mares
- Conception rate decreases linearly with age of the mare

Breed and Use
- No significant effect

HISTORY
- Has the mare had a foal previously?
- Is the mare cycling regularly?
- How long has the mare been out of training and has she received anabolic steroids?
- How long does the mare remain in season?
- Has there been any discharge noted from the vulva?
- Has the behavior of the mare changed?
- How many times was the mare bred?
- Has the mare been bred artificially or naturally?
- When was the last time the mare was bred?
- Has the mare conceived but the pregnancy has not continued?
- Are other mares pregnant that have been bred to the same stallion?

PHYSICAL EXAMINATION

General Inspection
- General body condition
- Attitude—signs of depression or bright

*Denotes most likely diagnosis.

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Evidence of systemic disease
Evidence of chronic musculoskeletal problems

Detailed Inspection
Examination of perineal conformation (note vulval angle)
Examination for evidence of discharge from the reproductive tract
Vital signs: HR, RR, temperature, mucous membranes, capillary refill time
Examination of the udder

DIAGNOSTIC PROCEDURES

Speculum Examination
Examination of the vagina for scars, lacerations
Examination of the cervix

Rectal Examination
Palpation of any abnormal abdominal masses
Palpation of the uterus
Palpation of the ovaries
Palpation of the cervix

Clinical Pathology
Cervical or uterine bacteriologic swab
Endometrial biopsy: histologic examination
Endometrial or cervical cytology

Ultrasound Examination
Ultrasound examination of uterus
Ultrasound examination of the ovaries

Endoscopic Examination
Examination of the uterus

Endocrinologic Examination
Plasma progesterone concentration

DIFFERENTIAL DIAGNOSES
Endometritis*
Urine pooling*
Pneumovagina*
Cervical abnormalities (e.g., adhesions, cervicitis)*
Uterine hypoplasia
Endometrial cysts
Transitional estrus, particularly if early in breeding season
Ovarian problems (e.g., follicular atresia, ovulation failure, tumor, persistent corpus luteum)
Fallopian tube abnormalities
Poor mare management (e.g., breeding at wrong time, poor estrus detection, etc.)
Perineal lacerations or rectovaginal fistula
Subfertile or infertile stallion
Chromosomal abnormalities
General physical problem (e.g., malnutrition, chronic laminitis, severe degenerative joint disease, etc.)

*Denotes most likely diagnosis.

INFERTILITY, STALLION

SIGNALMENT

Age of the Horse
Testicular degeneration—older stallions

Breed and Use
No significant effect

HISTORY
Has the stallion had systemic disease in the past few months?
Has the stallion been in training before the breeding season?
Has the stallion received anabolic steroids?
Is there any discharge from the penis or prepuce?
Is the stallion reluctant to mate?
Are mares failing to conceive, or is there early embryonic loss?
What percentage of mares have become pregnant in the current and previous breeding seasons?
Has the stallion had any injuries?
Has the stallion had a breeding soundness examination and what were the results?

PHYSICAL EXAMINATION

General Inspection
General body condition
Attitude—signs of depression or bright
Evidence of systemic disease
Evidence of chronic musculoskeletal problems

Detailed Inspection
Examination of penis and prepuce
Examination of testicles for size, resilience, signs of pain
Examination for evidence of discharge from prepuce
Vital signs: HR, RR, temperature, mucous membranes, capillary refill time

DIAGNOSTIC PROCEDURES

Rectal Examination
Examination of accessory sex glands
Examination of inguinal rings

Semen Examination
Motility
Abnormalities of sperm
Total sperm count
Urea nitrogen concentrations
Presence of erythrocytes

Bacteriology
Semen culture, including microaerophilic techniques
Cytologic examination

Histopathology
Testicular biopsy (only if indicated)

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DIFFERENTIAL DIAGNOSES
- Systemic disease*
- Trauma*
- Testicular degeneration*
- Venereal infections
- Infectious orchitis
- Inappropriate stallion management
- Balanoposthitis
- Urethritis
- Phimosis
- Paraphimosis
- Tumors of the penis, prepuce, or testicle
- Urospermia
- Hemospermia
- Habronemiasis
- Cryptorchidism
- Torsion of the spermatic cord
- Varicocele
- Epididymitis
- Seminal vesiculitis
- Lack of libido

LACRIMATION, EXCESSIVE AND/OR PHOTOPHOBIA

SIGNALMENT

Age of the Horse
- Entropion—young foals
- Recurrent uveitis—older horses
- Ocular neoplasia—older horses
- Habronemiasis—older horses
- Nasolacrimal duct obstruction—older horses
- Atresia of the lacrimal puncta—foals

Breed and Use
- No significant effect

Sex
- No significant effect

HISTORY
- Is there a history of trauma?
- How long has the problem been present?
- Is the horse reluctant to go out in the daylight?
- Does the eye remain closed at all times or only in the light
- Does the horse show any systemic signs (e.g., depression, inappetence)?
- Is the discharge from the eye clear or purulent?
- Is the cornea cloudy?
- Does the eye appear inflamed?

PHYSICAL EXAMINATION

General Inspection
- Note whether one or both eyes affected
- Attitude and mental status of the horse

Detailed Inspection
- Vital signs: HR, RR, temperature, capillary refill time
- Globe size: increased or decreased
- Presence of swelling or other abnormalities of the eyelids
- Inspection of nasal opening of nasolacrimal duct
- Examination of the conjunctiva and sclera
- Pupillary light reflexes
- Menace response
- Examination of the third eyelid
- Examination of corneal surface using direct light source

DIAGNOSTIC PROCEDURES

Nerve Blocks
- Auriculopalpebral block to remove motor function of upper eyelid
- Supraorbital block to desensitize the upper eyelid
- Topical anesthesia using proparacaine 0.5% to desensitize conjunctiva and cornea

Fluorescein Test
- Fluorescein strip to demonstrate corneal imperfections

Bacteriology and Cytology
- Scraping and culture for establishing cause of corneal ulcer

Ophthalmoscopy
- Examination of the anterior chamber and lens
- Examination of the retina

DIFFERENTIAL DIAGNOSES
- Corneal ulcers*
- Trauma*
- Conjunctivitis*
- Recurrent uveitis (e.g., periodic ophthalmia, immune-mediated uveitis)*
- Blepharitis
- Retrobulbar abscess
- Squamous cell carcinoma of the third eyelid
- Orbital cellulitis
- Entropion
- Habronemiasis
- Ectopic cilia
- Distichiasis
- Sarcoid
- Absence of distal nasolacrimal duct punctum

LAMENESS, ACUTE

SIGNALMENT

Age of the Horse
- Metabolic bone disease/developmental orthopedic disease—young horses

*Denotes most likely diagnosis.

*Denotes most likely diagnosis.

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Degenerative conditions—mature horses
Septic conditions—foal
"Bucked" shins—2-year-old racehorses

*Breed **and Use***

ATHLETIC/PERFORMANCE HORSES

"Bucked" shins
Chip fractures, fetlock, carpus
Bowed tendons (tendon strain)
Suspensory desmitis
Nail prick
Tying up
Sesamoid fractures
Degenerative joint disease
Septic arthritis
Long-bone fractures, proximal phalanx, condylar fractures—third metacarpal bone (McIII)
Fissure fracture—McIII, proximal phalanx
Laminitis
Rupture of the suspensory apparatus
Fractures of second metacarpal bone (McII)
Curb

PLEASURE HORSES

Foot abscess
Nail prick
Long-bone fractures
Direct trauma
Laminitis
Tying up
Foals
Septic arthritis, polyarticular
Trauma, fractures of long bones

Sex

No significant effect

History

Was the lameness related to a specific exercise episode?
Is the lameness related to an episode of trauma?
Did the lameness worsen in the period immediately after exercise?
Did the lameness worsen in the days after the episode?
Did the lameness improve with rest?
Did the lameness improve with "bute"?
Was the horse shod recently?
Does the horse rest its leg and if so what position does it adopt?
Has the horse been injected in a joint in the affected leg recently?

PHYSICAL EXAMINATION

General Inspection

Presence of swelling: joints, tendons, dorsal metacarpus
Symmetry of the limbs
Weight bearing of limbs
Examination of gait at the walk and trot (hard even surface)
Lunging of horse, both clockwise and counterclockwise

Detailed Examination

Digital pulse, prominence
Temperature (subjective), foot to carpus/tarsus (comparison with normal leg)
Pain on palpation or flexion
Hoof testers
Hoof hammer
Flexion test response

DIAGNOSTIC PROCEDURES

Nerve Blocks

If a fracture is suspected, nerve blocks should be avoided
Palmar digital
Palmar (abaxial)
It is essential to exclude the foot before proceeding to other possible sites.
Palmar metacarpal (low four point)

Intraarticular Local Analgesia

Distal interphalangeal (coffin) joint
Fetlock joint
Intercarpal (midcarpal)
Radiocarpal (proximal carpal)
Elbow
Shoulder
Hock, tarsometatarsal and tarsocrural joints
Stifle
Hip

Radiography

Additional oblique views of the foot, fetlock, carpus, and tarsus
Use of skyline views of the carpal bones
Fractures of the third carpal bone can sometimes be missed without inclusion of a skyline view
Air arthrograms can be considered to delineate soft-tissue swellings in the dorsal fetlock

Ultrasound

Use to better define tendon and ligament injuries for prognosis

Joint Fluid Analysis

Total and differential leucocyte count, total protein, cytology (including Gram stain) and culture, if a septic arthritis is suspected

Nuclear Scintigraphy

Specific or whole leg scans to determine "hot spots"

DIFFERENTIAL DIAGNOSES

Subsolar (foot) abscess*
Acute joint sprains*

*Denotes most likely diagnosis.

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Osteochondral (chip) fractures in fetlock or carpus*
Nail prick*
"Bucked" shins
Tendon strains and suspensory desmitis
Tying up (rhabdomyolysis)
Muscle injuries
Fractures of a long bone (particularly distal phalanx, proximal phalanx, and condylar fractures of the third metacarpal bone)
Laminitis
Fissure fracture, proximal phalanx, McIII
Direct trauma
Septic arthritis
Infection of tendon sheath
Infection of navicular bursa
Fractures of sesamoid bones

KEY POINT

If a fissure fracture is suspected, although no radiographic changes are found, It is advisable to immobilize the leg with a cast and check the radiographic appearance after 7 days in case the fracture displaces.

LAMENESS, CHRONIC

SIGNALMENT
Age of the Horse
Metabolic bone disease—young horses
Degenerative conditions—mature horses
Congenital deformities—young horses
Navicular disease—mature horses
Breed and Use
ATHLETIC/PERFORMANCE HORSES
Degenerative joint disease
Pedal osteitis
Navicular disease
Bone spavin
Osteochondrosis dissecans (OCD)
Bowed (strained) tendons and suspensory desmitis
Nonunion of distal phalangeal (pedal bone) fractures
Sacroiliac pain
Back injuries
PLEASURE HORSES
Navicular disease
Degenerative joint disease
Fibrotic myopathy
Chronic laminitis
Pedal osteitis

*Denotes most likely diagnosis

FOALS
Developmental orthopedic disease
Sex
Chronic lameness is more likely to be presented in geldings
HISTORY
What is duration of the lameness?
Has the lameness gradually been worsening?
Does the lameness improve with exercise?
Is the lameness more apparent immediately after exercise?
Is the lameness more apparent after the horse has"cooled down"?
Does the lameness improve with rest?
Is there an improvement with "bute"?
Has the horse been shod recently?
Does the horse rest the affected leg?
If the leg is rested, how does the horse hold the affected leg? (pointing the toe may indicate navicular disease)
Have there been any temperament changes noted? (sometimes related to back pain)
PHYSICAL EXAMINATION
General Inspection
Presence of swelling: joints, tendons
Atrophy of muscles, particularly gluteal and muscles of the shoulder
Hoof wear
Hoof cracks
Old scars
Detailed Examination
Temperature (subjective), foot to carpus/tarsus (comparison with normal leg)
Pain on palpation or flexion, particularly distal joints
Hoof testers (middle third of frog)
Flexion test response+
DIAGNOSTIC PROCEDURES
Nerve Blocks
Palmar digital
Palmar (abaxial)
It is essential to exclude the foot before proceeding to other possible sites
Palmar metacarpal (low four point)
Subcarpal (check ligament)
Intraarticular Local Analgesia
Distal interphalangeal (coffin) joint
Fetlock joint
Intercarpal (midcarpal)
Radiocarpal (proximal carpal)
Elbow
Shoulder

+Flexion tests of the fetlock and carpus are essential in aiding localization of pain. Spavin test in the hindleg will aid localization of pain to the hock and stifle.

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Hock, tarsometatarsal and tarsocrural joints
Stifle
Hip
Radiography
Additional oblique views in the foot, fetlock, carpus, tarsus
Use of skyline views of the carpal bones
Fractures of the third carpal bone can sometimes be missed without inclusion of a skyline view
Air arthrograms can be considered to delineate soft-tissue swellings in the dorsal fetlock
Ultrasound
Use to better define tendon and ligament injuries for prognosis
DIFFERENTIAL DIAGNOSES
Chronic degenerative joint disease*
Navicular disease*
Pedal osteitis*
Bone spavin*
Articular ringbone
Tendon and ligament strain/sprain
Back injuries/sacroiliac pain
"Wobblers"
OCD and bone cysts
Chronic proliferative synovitis
Nonunion fractures of pedal bone

KEY POINT
In some chronic lamenesses, several conditions may be contributing to the problem.

LIMB SWELLING

SIGNALMENT
Age of the Horse
Septic arthritis—foals
Osteomyelitis—foals
Epiphysitis—yearlings
"Bucked" shins—2-year-old racehorses
Tendon/ligament injuries—young horses
Joint disease—young horses
*Breed **and** Use*
ATHLETIC/PERFORMANCE HORSES
"Bucked" shins
Trauma
Joint disease, fetlock and carpus
Tendon/ligament injuries
Fractures, chip fractures involving fetlock and carpus
Soft-tissue injuries/trauma
"Splints"
Monoarticular septic arthritis
OCD
Abscess
Palmar annular ligament constriction
Hematoma
Bog spavin
Splint bone (McII) fractures
Interference, knee knocking
Hobble chafing
Curb
PLEASURE HORSES
Trauma/soft-tissue injuries
Hygroma
Splints
Fractures
Joint disease
Ringbone
Localized osteitis/osteomyelitis
Abscess
Periosteal new bone reaction
Muscle injuries
Hematoma
FOALS
Septic arthritis (joint ill)
Osteitis/osteomyelitis
Trauma/soft-tissue injuries
Tumoral calcinosis
Abscess
Sex
No significant effect
HISTORY
Is the horse lame?
How long has the swelling been present?
Has the swelling changed in size?
Is the horse showing signs of systemic illness?
Is there heat and pain associated with the swelling?
Is the swelling related to a joint?
Is the swelling related to a tendon or tendon sheath?
Is the swelling hard or soft?
Is there a draining sinus related to the swelling?
PHYSICAL EXAMINATION
General Inspection
Symmetry of the swelling
Location of the swelling
Examination at walk and trot to detect lameness
Relationship of swelling to a joint
Detailed Inspection
Palpation of the swelling
Aspiration, if indicated
If related to a joint, determination of pain on flexion

*Denotes most likely diagnosis.

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DIAGNOSTIC PROCEDURES

Radiography

- Oblique views are important
- Use low kVp and slight underexposure to highlight soft tissues
- Contrast radiography, if indicated

Ultrasound

- Essential in tendon and ligament injuries
- Useful in other swellings to determine soft tissue changes

Joint Fluid Analysis

- Only indicated if there is suspicion of septic arthritis

Nerve Blocks

- To confirm relationship between swelling and lameness
- Intraarticular anesthesia is important if swelling is due to joint effusion

DIFFERENTIAL DIAGNOSES

- Localized trauma*
- Degenerative joint disease/joint effusion*
- "Splints"*
- Tendon/ligament injuries*
- Abscess *(Corynebacterium* spp., *Clostridium* abscesses)
- Hematoma
- "Bucked" shins
- Bog spavin
- Splint bone fractures
- OCD
- Septic arthritis
- Tenosynovitis
- Ringbone
- Bone spavin

NASAL DISCHARGE

SIGNALMENT

Age of the Horse

- Viral respiratory disease—young horses
- Bacterial respiratory disease—young horses
- Cleft palate—young horses
- Nasal tumors—older horses
- Guttural pouch infections—all ages
- Progressive hematoma of the ethmoid—older horses

Breed and Use

ATHLETIC/PERFORMANCE HORSES

- Viral respiratory disease
- Strangles
- Bacterial pneumonia/pleuritis/bronchitis
- Pharyngitis
- EIPH
- Guttural pouch infection
- Sinusitis
- After laryngoplasty surgery
- Tooth-root infections
- Nasal conchal (turbinate) necrosis

PLEASURE HORSES

- Viral respiratory disease
- Strangles
- Bacterial pneumonia/pleuritis/bronchitis
- Guttural pouch infection
- Sinusitis
- Nasal conchal (turbinate) necrosis
- Nasal tumors
- Tooth-root infections
- Progressive hematoma of the ethmoid

FOALS

- Cleft palate
- Congenital pharyngeal abnormalities
- Bacterial pneumonia

Sex

- No significant effect

HISTORY

- Is the discharge unilateral or bilateral?
- Is the discharge clear, mucoid, purulent, or bloody?
- Is the discharge related to feeding or head posture?
- How long has the discharge been present?
- Is the quantity of the discharge increasing?
- Is there an associated cough?
- Are other animals in the barn/pasture affected?
- Is there evidence of systemic disease (e.g., depression, inappetence, fever, etc.)?
- Has the horse been vaccinated?
- Is the discharge malodorous?
- Has there been any change in facial contours?

PHYSICAL EXAMINATION

General Inspection

- Examination of the nares for discharge (uni/bilateral)
- Evidence of systemic disease (e.g., weight loss)
- Examination of the head and upper respiratory tract for swellings

Detailed Examination

- Percussion of the sinuses
- Look in the nose
- Oral examination to determine presence of tooth problems
- Auscultation of the thorax
- Smell the horse's breath
- Palpation over the guttural pouches

DIAGNOSTIC PROCEDURES

Rhinolaryngoscopy

- Examination of nasal conchae (turbinates)
- Guttural pouch openings
- Examination of guttural pouches
- Ethmoid region

*Denotes most likely diagnosis.

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Pharynx
Larynx and trachea

Radiography
Sinuses
Guttural pouches
Nasal conchae
Ethmoid
Tooth roots
Chest

Bacteriology
Sinus centesis
Transtracheal aspirate
Guttural pouch aspirate
Bronchoalveolar lavage
Anaerobic culture

Hematology and Biochemistry
White cell count and differential
Fibrinogen
Red cell count
Protein electrophoresis

Thoracocentesis
Cytologic examination
Bacteriologic examination
Glucose measurements

DIFFERENTIAL DIAGNOSES
Viral or bacterial respiratory disease*
Pharyngitis*
Pneumonia*
Sinusitis*
Strangles
EIPH
Dysphagia
Guttural pouch infections
Progressive hematoma of the ethmoid
Tooth-root infections
Nasal tumors
Nasal conchal (turbinate) necrosis
Heart failure
Cleft palate
Congenital pharyngeal abnormalities

PRURITUS

SIGNALMENT

Age of the Horse
Culicoides hypersensitivity—older horses
Dermatophytosis—younger horses
Urticaria—older horses

Breed and Use
No significant effect

Sex
No significant effect

HISTORY
Does the problem occur in a certain season?
Is more than one horse affected?
How long has the problem been present?
How severe is the pruritus?
Does the condition adversely affect the horse's behavior?
Has the horse been treated?
What was the response to treatment?
How long since the horse has been treated?
Is the horse eating normally?
Is the horse losing weight?

PHYSICAL EXAMINATION

General Inspection
General body condition
Attitude—signs of depression or bright

Detailed Inspection
Vital signs: HR, RR, temperature, gut sounds, capillary refill time
Determine whether the lesions are localized or generalized
Is there alopecia associated with the pruritus?
Is there evidence of self-trauma?
Clipping of hair in a pruritic region and examination of skin with a hand lens

Clinical Pathology
CBC, differential white cell count
Total plasma protein

Examination of the Skin
Skin scrapings
Impression smears
Intradermal skin testing
Acetate tape preparations
Bacterial/fungal cultures
Skin biopsies (histopathology, fluorescent antibody testing)

DIFFERENTIAL DIAGNOSES
Culicoides hypersensitivity (Queensland itch)*
Dermatophytosis*
Ectoparasites (e.g., biting flies, lice, ticks, harvest mites or "chiggers," mosquitoes)*
Cutaneous onchocerciasis
Pinworm infestation, *Oxyuris equi*
Folliculitis/furunculosis
Urticaria due to inhaled allergens, drug administration, or feedstuffs
Contact dermatitis
Eosinophilic dermatitis
Photosensitization, primary and secondary
Dermatophilosis
Seborrheic dermatosis
Pemphigus foliaceus
Scratches ("grease heel")
Phycomycosis
Habronemiasis

*Denotes most likely diagnosis.

*Denotes most likely diagnosis.

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Neurologic diseases (e.g., rabies, polyneuritis equi, self-mutilation syndrome)

RESPIRATORY NOISE

SIGNALMENT

Age of the Horse

Congenital airway disorder—newborn foal
Laryngeal hemiplegia—young racehorse
Epiglottic problems—young horse
Soft palate dislocation—young performance horse
Strangles—young horse
Nasal tumor—older horse
Guttural pouch tympany—foal

Breed and Use

ATHLETIC/PERFORMANCE HORSES

Laryngeal hemiplegia
Epiglottic entrapment
Subepiglottic cysts
Soft palate dislocation
Pharyngitis
Rostral displacement of the palatopharyngeal arch
Atheroma of the false nostril
Tooth-root abnormalities impinging on ventral nasal meatus
Nasal tumors

PLEASURE HORSES

Tooth-root abnormalities
Nasal tumors
Retropharyngeal abscess
Tracheal stenosis
Laryngeal hemiplegia

FOALS

Guttural pouch tympany
Retropharyngeal abscess
Bilateral laryngeal paralysis

Sex

Males are more likely to be affected by laryngeal hemiplegia than females

HISTORY

Can you describe the type of noise (e.g., roaring, whistling, gurgling)?
Is the noise apparent when the horse is at rest?
Is there evidence of dyspnea?
If the noise occurs during exercise, is it present at all speeds?
Is the noise getting worse with time?
Has the horse had a nasal discharge?
Has the horse been coughing?
Is the noise related to inspiration or expiration? (during cantering or galloping, as the leading leg hits the ground, the horse expires, whereas inspiration occurs when the forelegs are protracted)
Is there any noticeable swelling associated with the nose or face?
Has the horse been vaccinated?

PHYSICAL EXAMINATION

General Inspection

Presence of any nasal/facial swelling
Inspection of the external nares
Smell the breath

Detailed Examination

Percussion of the paranasal sinuses
Palpation of muscular process for prominence (atrophy of dorsal cricoarytenoid muscle)
Auscultation of upper airway and thorax
Palpation for soft-tissue mass in pharyngeal region
Postexercise arytenoid depression maneuver (pushing on muscular process to worsen the respiratory noise)
"Slap test" to assess adductor function

DIAGNOSTIC PROCEDURES

Rhinolaryngoscopy

Appearance of upper airway
Symmetry of the larynx
Evidence of inflammation
Normal appearance of openings to guttural pouches and no discharge evident from these when swallowing is induced
"Slap test" of adductor function
Constriction of nostrils to induce abduction of the larynx
Appearance immediately after exercise
Endoscopic appearance of trachea—presence of mucopus
Appearance of upper airway during treadmill exercise

Radiography

Radiography of the throat region
Radiography of the nose and sinuses

Arterial Blood Gas Analysis

Resting values of PaO_2
Arterial blood samples during exercise (requires a treadmill)

DIFFERENTIAL DIAGNOSES

Laryngeal hemiplegia*
Soft palate dislocation (dorsal displacement)*
Epiglottic entrapment
Subepiglottic cysts
Atheroma of the false nostril
Pharyngitis
Rostral displacement of the palatopharyngeal arch
Tracheal stenosis

*Denotes most likely diagnosis.

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Tooth-root tumors
Nasal tumors
Retropharyngeal abscess
Guttural pouch tympany
Hypoplasia of the epiglottis
Deviated nasal septum

SUDDEN DEATH

SIGNALMENT
Age of the Horse
No significant effect
Breed and Use
No significant effect
Sex
No significant effect
HISTORY
Was the horse observed at the time of death?
Have there been any recent changes in management?
Has the horse received any medication recently?
Was the death related to exercise?
Was there evidence of struggling?
Have there been any other recent horse deaths?
Has the horse been ill recently?
Was the horse at pasture or in a box stall?
If a mare, has it recently foaled?
Has there been recent storm activity?
PHYSICAL EXAMINATION
General Inspection
Note the position of the horse
Note any signs of struggling
Detailed Inspection
Age of horse
Presence of blood from any of the body orifices
Evidence of trauma
Evidence of diarrhea or blood in the feces
Note evidence of abdominal distension
Evidence of skin abrasions
DIAGNOSTIC PROCEDURES
Necropsy
Note distension or torsion of intestine or severe enteritis
Presence of hemorrhage (thoracic or abdominal)
Trauma (fractures of vertebrae, ribs, etc.)
Samples of blood, gut contents, liver, kidney, or heart for toxicologic profile
Sampling of pasture and/or feed for toxins
DIFFERENTIAL DIAGNOSES
Rupture of the aorta
Acute respiratory distress after adverse drug reaction
Mare (rupture of the uterine artery at parturition)
Electrocution/lightning strike
Acute fulminant colitis
Trauma (e.g., gunshot)
Snakebite
Pneumothorax
Skull fractures
Toxic plants (e.g., yew)
Cantharidin toxicosis
Endotoxemia
Monensin toxicity
Organic/chemical toxins

URINE OUTPUT CHANGES

SIGNALMENT
Age of the Horse
Ruptured urinary tract—foals
Chronic renal failure—older horses
Urolithiasis—older horses
Cystitis—older horses
Paralytic bladder—older horses
Psychogenic polydipsia—older horses
Breed and Use
No significant effect
Sex
No significant effect
HISTORY
When was the change in urine output first noticed?
Has the horse had a change in water consumption?
Does the horse strain to urinate?
Is the urine discolored?
Is there blood in the urine and if so at what stage of urination does it occur?
Is the horse losing weight?
Is the change in urine output related to exercise?
Has the horse received any medication (e.g., gentamicin or NSAIDs)?
PHYSICAL EXAMINATION
General Inspection
General body condition
Attitude—signs of depression or bright
Detailed Inspection
Vital signs: HR, RR, temperature, gut sounds, capillary refill time
Rectal examination, particularly for examination of urinary tract
Clinical Pathology
Urinalysis
Serum/plasma urea nitrogen and creatinine

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Fractional electrolyte excretions
Total serum/plasma protein
Urinary creatinine ratio
CBC
Abdominocentesis
Peritoneal fluid: serum creatinine ratio—foals

Water Deprivation Test
Check urinary concentrating ability in selected cases

Bacteriology
Bacterial culture of urine

Endoscopic Examination/Cystoscopy
Lower urinary tract and bladder

Ultrasound
Renal
Bladder
Abdominal (foals, suspected ruptured bladder)

Abdominal Radiography
Plain and contrast (foals, suspected ruptured bladder)

Injection of Nontoxic Dye (Methylene Blue, Fluoroscein)
Via urethral catheter (foals, suspected ruptured bladder)

Renal Biopsy
For prognosis/diagnosis, ultrasound guided (if possible)

DIFFERENTIAL DIAGNOSES

Oliguria
Acute renal failure*
Chronic renal failure*
Severe dehydration*
Shock
Urolithiasis
Equine herpesvirus myelitis
Ruptured urinary bladder—foals

Polyuria
Chronic renal failure*
Psychogenic polydipsia*
Cushing's syndrome
Fluid administration
Frusemide administration
Steroid administration
Tumors of the pars intermedia of the pituitary gland
Diabetes mellitus
Cantharidin toxicosis

WEIGHT LOSS OR FAILURE TO THRIVE

SIGNALMENT

Age of the Horse
Parasites—young horses
Gastrointestinal ulcers—young horses
Diarrhea—young horses
Internal abscessation—young horses
Tooth problems—older horses
Inflammatory bowel disease—older horses
Neoplasia—older horses
Liver disease—older horses
Renal disease—older horses

Breed and Use

ATHLETIC/PERFORMANCE HORSES
Chronic infections/internal abscessation
Pleuropneumonia
Diarrhea
Gastrointestinal ulcers
Abdominal abscesses
Peritonitis
Inflammatory bowel disease
Neoplasia (e.g., lymphosarcoma)

PLEASURE HORSES
Malnutrition
Teeth problems
Chronic infections (e.g., pleuritis)
Chronic diarrhea
Gastrointestinal ulcers
Internal abscessesation
Peritonitis
Inflammatory bowel disease
Neoplasia (e.g., lymphosarcoma)
Liver disease
Chronic colic (e.g., sand, enterolith)
Renal disease
Heart failure

FOALS
Diarrhea (e.g., rotavirus)
Chronic infections
Congenital gastrointestinal abnormalities
Malnutrition/maldigestion
Chronic intussusception
Internal abscessation
Immunodeficiency

Sex
No significant effect

HISTORY
Is the horse eating normally?
How long has the horse been losing weight?
Is the appetite increased or decreased?
Is the appetite capricious?
What is the composition of the diet?
If the horse is grazing pasture, is there access to sand?
Are there any behavioral changes?
How frequently is the horse dewormed?
Are there any signs of abdominal pain?
Are there signs of systemic disease?
Has there been a disease outbreak in the herd (e.g., "strangles," rotavirus, etc.)?
Are the feces normal in color, volume, and consistency?

*Denotes most likely diagnosis.

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Have any medications been given and what is the response?
Where is the horse housed?
Is the horse low on the social structure within the herd?

PHYSICAL EXAMINATION

General Inspection
Estimation of weight loss: mild, moderate, or severe
Attitude—signs of depression or bright
Consistency and composition of feces
Signs of pain
Evidence of systemic disease
Evidence of dysphagia or problems with prehension

Detailed Inspection
Vital signs: HR, RR, temperature
Examination of teeth and tongue
Body weight
Capillary refill time (mucous membrane color)
Skin turgor
Gut sounds
Auscultation of heart and thorax
Palpation of superficial lymph nodes

DIAGNOSTIC PROCEDURES

Clinical Pathology
Hematocrit and total serum/plasma protein
White cell count and differential
Plasma/serum fibrinogen
Liver enzymes/biochemistry: GGT, AP, bilirubin, bile acids
Renal function tests: blood urea nitrogen, creatinine, urinalysis
Abdominal fluid analysis: total protein, nucleated cell count, cytology, bacteriology
Serum protein electrophoresis

Serology
Coggins test
Ehrlichia titers
Equine protozoal myeloencephalitis (EPM) testing

Fecal Examination
Fecal consistency
Parasite ova
Fecal occult blood
Fecal bacteriology, e.g., *Salmonella* (selective culture media used with samples over several days necessary; results improved if combined with culture of rectal mucosal biopsy)
Fecal/rectal mucosal mycology
Examination for sand

Nutritional Examination
History of eating off sandy soil
Quality and volume of feed

Rectal Examination
Abnormal masses
Distended/thickened bowel
Areas of localized pain

Abdominocentesis
Fluid color
Fluid turbidity
Presence of blood
Cytology and bacteriology

Small Intestinal Absorption Tests
D-Glucose
D-Xylose

Other Procedures (If Indicated)
Rectal mucosal biopsy
Liver biopsy
Renal biopsy
Exploratory laparotomy

DIFFERENTIAL DIAGNOSES

Adults
Malnutrition*
Teeth problems*
Parasitism (e.g., strongylosis)
Chronic infections
Chronic pleuritis
Abdominal abscesses
Peritonitis
Chronic diarrhea
Gastrointestinal ulcers
Liver disease
Chronic colic (e.g., sand, enterolith)
Renal disease
Inflammatory bowel disease
Neoplasia (e.g., lymphosarcoma)
Heart failure
Heavy-metal intoxication (e.g., lead)
Idiopathic

Foals
Malnutrition*
Bacterial septicemia*
Failure of passive transfer of immunity*
Strongyloides westeri infestation
Parascaris equorum **(roundworm)** infestation
Rhodococcus equi infection
Small intestine intussusception
Chronic diarrhea
Rotavirus diarrhea
Bacterial gastrointestinal tract infection: *Salmonella, E. coli, Clostridium, R. equi*
Idiopathic
Liver disease
White muscle disease

*Denotes most likely diagnosis.

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CHAPTER 3

Practical Diagnostic Imaging

Roger K. W. Smith, Celia Marr, and R. Christopher Whitton

Section 1. General Principles and Musculoskeletal Ultrasonography

Ultrasonography was developed in the medical field in the late 1960s. Unlike many imaging systems currently available to the medical profession, the equipment is affordable for veterinarians and so has made a rapid transfer into the veterinary field. It was first used for the diagnosis of pregnancy in the mare in the early 1970s and then used for the investigation of tendon and ligament injuries a decade later. Since then, there has been a decrease in the cost of ultrasound equipment, an improvement in the quality of the images they produce, and application to all major body systems. It is now considered a standard item of equipment in the diagnostic armory of any veterinarian undertaking a significant amount of horse work. The ability to obtain a diagnostic ultrasound image takes only a little time and practice. However, the interpretation of the acquired images requires experience, especially for the more subtle pathology, where the quality of the ultrasound machine and image are also important factors. There are many pitfalls into which the ultrasonographer can fall, and this chapter attempts to introduce the techniques and the basis of interpretation to minimize such occurrences.

PRINCIPLES OF ULTRASONOGRAPHIC IMAGING

Ultrasound refers to sound of frequencies higher than the audible range (over 20,000 Hz). For imaging, frequencies of more than 1 MHz are used.

Generation of the Ultrasound Beam

The high-frequency sound waves are generated by the application of an electrical pulse to a *piezoelectric crystal.* The crystal responds to the electrical current by contracting and expanding to generate the physical sound wave. The range of frequencies of the sound produced has a bell-shaped distribution, with the most prominent frequency emitted being the center frequency.

Generation of the Ultrasound Image

Ultrasound passes into the tissues, and reflections (echoes) are created by two different processes:

1. When sound meets interfaces between tissues of differing acoustic impedance (related to their density), sound is reflected from the interface *(specular reflection).* The greater the differences in acoustic impedance between the two tissues, the greater the amplitude of the reflected echo. Most sound is reflected at the interface of soft tissues with bone or air, leaving insufficient sound to produce an image of deeper structures. Ultrasound, therefore, can only image adequately non-osseous tissues and the surface of bone.
2. The second process involves the scattering of the ultrasound when it meets small reflectors within the tissues. This echo is known as *nonspecular reflection (scatter)* and allows the internal architecture of the tissues to be represented on the ultrasound image.

The reflection of the sound by specular or nonspecular reflection results in *attenuation* of the beam as it passes through the tissues. The beam is further attenuated by the generation of heat within the tissues, which, in diagnostic imaging,

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is very small. Attenuation is frequency dependent, so that the higher the frequency the greater the attenuation and therefore the less the penetrative depth.

The *resolution* of ultrasound is defined by lateral and axial components. Lateral resolution depends on the beam width, which is determined by the crystal size. In general, larger crystals produce lower frequencies, wider beam width, and therefore have lower lateral resolution. Lateral resolution is further improved by focusing the beam. Different focal zones can be selected dependent on the machine and the transducer type. Axial resolution depends on the length of the ultrasound pulse, which, in turn, depends on the frequency. The higher the ultrasound frequency, the higher the axial resolution. The axial resolution can be calculated from the frequency and the speed of sound in tissues. Wavelength (approximates to resolution) = velocity (1540 m/s) ÷ frequency. Therefore, an ultrasound frequency of 10 MHz will have an axial resolution of approximately 0.15 mm, whereas the resolution of a frequency of 3.5 MHz is 0.44 mm.

KEY POINT

The highest frequency which will give the necessary penetration is the frequency of choice. Many transducers operate broadband width technology, where a transducer can use the lower emitted frequencies for deeper structures and higher frequencies for the more superficial structures.

The reflected echoes return to the transducer and are received by the crystals during "listening" periods between the ultrasound pulses. These echoes are converted to electrical signals by the piezoelectric effect and are processed to give the ultrasound image. The display of this information can be in a number of ways:

1. **A-mode,** where the echo is displayed as an Amplitude on an oscilloscope screen; this is no longer used for imaging purposes.
2. **B-mode,** where the intensity of the echo is reflected by the Brightness of a dot on the screen. The image is built up by the simultaneous display of multiple B-mode elements to give a two-dimensional image. If a single element is updated and displayed adjacent to the previous display (i.e., scrolled across the screen), this is known as *M-mode.* If all the B-mode elements are continuously updated, it is known as *real-time* imaging and therefore can display movement, a particularly useful facet to diagnostic ultrasound imaging.
3. **Doppler ultrasonography** also relies on high-frequency ultrasound. However, it depends on the Doppler effect, where the frequency of sound is modified if reflected by a moving structure. In the heart and blood vessels, sound reflected by moving blood cells undergoes a frequency change that depends on the speed and direction of the blood flow. Velocities can only be determined accurately if the interrogating ultrasound beam is almost parallel to the direction of blood flow.

Spectral Doppler images represent a graph of velocity against time where flow toward the transducer is displayed above the baseline, whereas flow away from the transducer is below the baseline. *Continuous-wave Doppler* can record high velocities but cannot provide spatial data because it samples along the length of the ultrasound beam. *Pulsed-wave Doppler* echocardiography samples an operator-specified site, providing precise spatial information but is limited in the velocity that it can accurately record. *Color flow Doppler* echocardiograms consist of multiple pulsed-wave Doppler sites sampling simultaneously to provide a two-dimensional map of blood flow. Different velocities and directions of blood flow are encoded in color. Typically, blood flow toward the transducer is represented in shades of red, whereas blood flow away from the transducer is represented by shades of blue. Some color flow echocardiographic units can detect variance, or turbulent flow, and may depict this in other colors, such as green.

Power Doppler, or Doppler angiography, is a new modality in which the amplitude of the returning Doppler signal is quantified and displayed in color. This is particularly sensitive to low-velocity blood flow and, unlike conventional Doppler imaging, is not angle dependent. This technique is primarily used to detect peripheral blood flow and may have potential applications in abdominal imaging and in identifying increased blood flow in inflammation in the musculoskeletal system.

Types of Ultrasound Transducer

LINEAR

Multiple crystals are aligned along the flat surface of the transducer. This will give a rectangular image on the screen.

CURVILINEAR OR CONVEX

Multiple crystals are aligned along a curved transducer surface. This will produce an image on the screen intermediate between the pie-shaped image

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of the sector transducer and the rectangular image of the linear transducer. This transducer has the advantage of reducing the contact area, but far-field resolution is generally inferior to the sector transducer.

MECHANICAL SECTOR

A single crystal is oscillated back and forth or rotated. This will give a pie-shaped image on the screen. It has the advantage of reducing the necessary contact area of the transducer (ideal for imaging the heart between ribs) but has reduced near-field (narrow beam width) and far-field (widely spaced elements) resolution.

ANNULAR ARRAY

A concentric arrangement of ring-shaped elements is seen, which are mechanically oscillated or rotated to give a pie-shaped image. The concentric arrangement of the elements provides good focusing in two planes.

PHASED ARRAY

Multiple elements are present in a compact transducer. The elements are "fired" in sequence and the resulting beam steered electronically to give a sector-like display. These transducers are preferred for color Doppler but are expensive.

Sound Path Delay

For examining very superficial structures such as the superficial digital flexor tendon within the metacarpal region, a stand-off or "fluid offset" is placed between the transducer and the skin. This is composed of a substance that allows the transmission of sound with little attenuation and is usually either fluid or a commercially available silicon pad. It has the effect of moving the superficial structures away from the emission artifact at the surface of the transducer (the echoes seen at the top of the screen that arise from reverberation of sound from the transducer housing) and closer to the focal zone of the transducer, thereby optimizing resolution. In addition, it increases the width of the ultrasound "window" when the flat surface of a linear transducer is placed against the curved palmar aspect of the metacarpus. A "fluid offset" is integrated into the transducer. The benefit of a separate stand-off is that it can be removed for examining deeper structures, where the albeit low attenuation of the sound beam by the stand-off does not compromise the image quality.

Ultrasound Machine Controls

These vary in number and nature between ultrasonographic units. Some controls require modification for each examination to accommodate differences in the propagation and penetration of ultrasound in individual horses of different body types. Other controls depend on the particular application or area under examination and can be used every time that area is examined but may need to be changed to examine different anatomic regions. In general, for imaging static soft tissues such as the musculoskeletal system or abdominal organs, a soft image maximizing the gray scale is used to differentiate many different tissue types. Conversely, for echocardiography, where relatively large moving structures are examined, a fast frame rate is necessary, and an image with high contrast is selected to differentiate the solid cardiac structures from the fluid-filled chambers.

CONTROLS MODIFIED BY THE OPERATOR DURING EACH EXAMINATION TO ACCOMMODATE DIFFERENCES IN INDIVIDUAL PATIENTS

Power

This adjusts the energy delivered to the transducer so that increased power produces a brighter image. However, too much power can mask subtle details of the image. The operator sets the power control at the lowest level at which all detail of the image is clear.

Gain

This modifies the amplification of the returning echoes. Increasing the gain will increase the brightness of the image but will also amplify noise, and too much gain can mask subtle details of the image. The operator sets the gain control at the lowest level at which all detail of the image is clear.

Time Gain Compensation

The echoes coming from deeper in the body have lower amplitude than those from the superficial structures because of beam attenuation. Time gain compensation (TGC) compensates for this so that echoes from the deeper tissues are amplified more. TGC can be adjusted by the operator so that the brightness of echoes is uniform throughout the image.

Depth

The operator should select a depth of penetration that allows examination of all tissue or organ of interest but does not include areas that are not of diagnostic interest.

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Focusing

Linear and curvilinear probes may have variable focusing. The image should be focused on the tissue under examination. It may be necessary to adjust the focusing during an examination to focus on different structures.

KEY POINT

Excessively high power and gain settings are a common error, leading to an image that is so bright the subtle pathology is masked. When the power and gain are too low, the image is dark, increasing the risk of anechoic or hypoechoic artefacts.

CONTROLS SELECTED FOR SPECIFIC APPLICATIONS AND EXAMINATION ROOM ENVIRONMENT

Screen Brightness and Contrast

These refer to the television screen and have no direct effect on the generation of the ultrasound image. They should be set to accommodate the ambient light in the examination area but should not be used to compensate for an image that is too light or dark because of inappropriate power, gain, and TGC settings. A "light wedge" may be displayed on the side of the screen to assist in the setting of these controls so that the maximum range of gray scale is visible.

Reject

Increases in reject improve the signal-to-noise ratio. The reject control is used to improve image quality when very high power and gain have been used, for example, in abdominal imaging in large fat horses.

Gray-Scale Allocation

The range of grays used to display echoes of different amplitudes can be modified to produce images with more or less contrast. For soft-tissue imaging, a soft image with a maximum number of shades of gray often allows more subtle differences in tissue characteristics to be displayed. Functions such as postprocessing, gamma curve allocation, dynamic range, and compression alter gray-scale allocation and vary in sophistication between different machines. Usually, the operator can select and store different programs to be used for different applications.

Frame Rate

Fast frame rates are necessary for imaging moving structures, but lower frame rates improve image quality by providing more time for the ultrasound to collect returning echoes.

IMAGE MANIPULATION, MEASUREMENT, AND STORAGE

Split Screen

This allows sequential images to be displayed side by side. This facility is particularly useful to compare a suspected injury with the corresponding area on the contralateral limb or to record longitudinal and transverse images of a lesion simultaneously.

Measurement Calipers

Measurement of liner dimensions and cross-sectional areas, using a trackball system, is particularly useful to provide an objective assessment of individual structures.

Alphanumeric Keyboard

This allows comprehensive labeling of patient identify, date of examination, and details of region under examination.

Image Storage

It is important for maintenance of medical records and for monitoring the progress of individual patients that ultrasonographic images are recorded adequately. Options include thermal prints, video, Polaroid films, radiographic plates, and digital archiving systems. Thermal prints are the cheapest, but video is superior if movement is important, for example in echocardiography. It is important that stored images have no or minimal artifacts to avoid difficulty in subsequent interpretation. Ideally, images should be interpreted at the time of examination.

Common Artifacts

OPERATOR-INDUCED ERRORS

1. *Off-incidence artifact.* Failure of the transducer to be positioned at 90 degrees to the surface of an echogenic structure will result in the structure appearing hypoechoic.
2. *Inadequate patient preparation.* Poor preparation results in an increased number of contact artifacts that arise because of the presence of air between the transducer and the skin. This results in either a poor quality picture or dark anechoic bands in the image.
3. *Inappropriately adjusted gain and power.*

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ATTENUATION ERRORS

Acoustic Shadowing

Shadowing may be caused either by a highly reflective boundary (e.g., bone, foreign body, gas) or occurs at the edge of a reflective curved surface when the beam is tangential to it. The latter is called *refraction,* or *edge shadowing,* and is particularly evident when examining the suspensory ligament branches in the transverse fashion, where the digital arteries or borders of the flexor tendons cast a shadow through each branch.

Acoustic Enhancement

Enhancement occurs deep to an area of low attenuation (e.g., fluid), producing a more echogenic appearance to the structures beneath. Focal enhancement can arise in the middle of the image as a result of the focusing of the beam in this region.

The presence of either shadowing or enhancement can be used to the operator's benefit to help identify areas of mineralization, gas, fluid, or foreign bodies.

PROPAGATION ERRORS

Reverberation Artifact

If there is a strong reflector, such as an appreciable amount of air or a metallic foreign body, in the tissues or between the transducer and the skin, the shadowing artifact often is filled with a "reverberation artifact." This can be either a series of hyperechoic lines parallel to the hyperechoic line from the surface of the gas, as seen in the normal lung, or uniform echogenicity, as in the "comet-tail" artifact produced by some metallic foreign bodies.

Speckle

This produces a grainy appearance as a result of interference effects of scattered sound from multiple small reflectors within a tissue.

Mirror Image

A highly reflective surface can result in a second reflection that can produce a mirror image of the more superficial soft tissues deep to the reflective surface.

Positioning Errors

Positioning errors arise when the ultrasound beam is bent, or refracted, because the sound travels across a boundary between two areas with different acoustic impedance. Multipath refers to the positioning error created by the echo being reflected back to the transducer via a different path (second reflection from another strong reflector).

Range Ambiguity

In the presence of a large cystic structure, increased depth can be imaged because of reduced attenuation. In such cases, echoes from the deeper structures may return to the transducer after the emission of the next sound pulse and are therefore interpreted as coming from nearer the transducer. This may produce extra artifactual echoes within the cystic structure.

Ultrasonographic artifacts can frequently be differentiated from true abnormalities because many artifacts have characteristic appearances or sites, the position of contact artifacts will alter within the tissue with movement of the transducer, and replacing the transducer on the skin can result in the disappearance of the artifact.

Preparation and General Technique

KEY POINT

It is imperative that good contact is achieved between the transducer and the skin if good diagnostic images are to be produced. Time spent at this stage usually yields considerable reward in image quality.

The following procedure is recommended before ultrasonographic examination:

1. The hair over the area is clipped with fine-bladed electric clippers.
2. Surgical scrub solution is used to clean the area thoroughly and remove clipped hair.
3. The area is cleaned with alcohol to remove bubbles created by the surgical scrub and to help dissolve wax and greases on the skin.
4. Finally, the region is wiped dry. If left wet, the high-viscosity contact gel can run off the skin (especially the limbs).
5. Contact gel is applied liberally to the skin, transducer, and, if used, the stand-off.

Veterinarians frequently are requested to scan the horse's limbs without clipping. In fine-coated breeds (e.g., the Thoroughbred), this is possible if the skin and hair are cleaned well and contact gel is liberally applied to the skin and allowed to soak in. However, contact artifacts are more common without clipping, and it is important that the veterinarian inform the owner/trainer before the examination that it is possible to miss subtle pathology if clipping is not performed.

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Terminology

The terms "echolucent" and "sonolucent" are incorrect terms, because "lucent" refers to electromagnetic radiation and not to a physical wave such as sound. They should therefore not be used (Table 3-1).

MUSCULOSKELETAL ULTRASONOGRAPHY

Equipment

In the horse, most musculoskeletal structures that are examined ultrasonographically are within 5 to 8 cm of the skin, so that a center frequency of 7.5 MHz is the most applicable. Equipment should give detailed imaging of the flexor tendons and sufficient penetration to image the suspensory ligament.

The choice of transducer type depends on both operator preference and the area being imaged. In most cases, a small linear transducer is best for musculoskeletal work because it allows both transverse and longitudinal images to be most easily assessed. Small curvilinear or sector transducers are preferable when there is limited area for transducer contact (e.g., between the bulbs of the heels). Ideally, 5- and 3.5-MHz transducers should also be available for imaging deeper structures in the proximal regions of the limbs.

A stand-off pad or fluid-offset transducer is useful for examining the most superficial structures. However, the most modern transducers have very small emission artifacts and large focal zones, so they can be used without a stand-off for all except the most superficial structures. A hard-wearing stand-off, although expensive, is cost-effective.

Tissue Characteristics

Ultrasonography demonstrates the structure of the tissues rather than tissue type. Hence, some tissues may have different appearances depending on the frequency used and the adjacent tissues. As a basic principle, black areas (absence of echoes) represent homogeneous tissue or fluid, whereas white areas indicate nonuniform structures. Table 3-2 gives some examples of the common appearance of various tissues.

Principles of Interpretation of Tendon and Ligament Pathology

Tendon and ligament pathology gives rise to a number of ultrasonographic changes. Maximum information can be obtained by assessing a number of ultrasonographic signs.

CHANGES IN ECHOGENICITY

Anechoic/Hypoechoic Change

"Black holes" in acute tendon injuries represent the presence of hemorrhage and disrupted fibers, granulation tissue, or young fibrous tissue (in order of increasing echogenicity). Peritendinous inflammation is represented by a hypoechoic area surrounding the tendon. The inflammatory fluid acts as negative contrast and increases the definition of the tendon outline.

Hyperechoic Change

The brighter appearance of a healed tendon injury represents the formation of fibrosis. Alternatively, mineralization or foreign bodies cause acoustic shadowing deep to a strongly echoic area.

CHANGES IN SIZE

Increased cross-sectional area accompanies significant damage to tendons and ligaments, and an

TABLE 3-1. Commonly Used Terms Used to Describe Ultrasonographic Findings

Term	Explanation
Anechogenic/anechoic	Not producing any echoes (therefore appearing black on the screen)
Echogenic	Having 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)
Isoechogenic/isoechoic	Producing similar intensity echoes to another area
Normoechogenic/normoechoic	Producing the expected intensity of echoes for the structure in question
Ultrasonograph	Image produced by the ultrasound scanner

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TABLE 3-2. Ultrasonographic Appearances of Commonly Encountered Musculoskeletal Tissues and Substances

Tissue	Ultrasonographic Appearance
Tendons/ligaments	Dotted internal architecture when the transducer lies at right angles to the structure (transverse) and a striated pattern when the transducer is parallel (longitudinal). Ligaments may have deep and superficial parts with fiber orientation in different directions.
Muscle	Hyperechogenic interfascicular septa with interspersed hypoechogenic muscle fibers. Fascial planes appear as strongly hyperechoic lines.
Cartilage	Anechogenic (black). The cartilage-synovial membrane-fluid and cartilage-bone interface give an appearance of "tram-lines."
Bone	Highly reflective and produces acoustic shadowing deep to it. The thickness of the bone surface echo can increase with active bone formation and decrease in sequestra. Forming or resorbing bone casts a variable degree of shadowing.
Fat	Dependent on the connective tissue content of the fat. Frequently relatively hyperechogenic.
Blood	Varies between hypoechogenic and hyperechogenic. Normal blood vessels are hypoechogenic. Superficially, veins usually will show some degree of collapse because of the pressure exerted by the transducer, whereas arteries usually will maintain their circular shape.
Nerves	Echogenic. They can be difficult to identify but the larger ones have a coarse stippled pattern in transverse sections and a striated pattern in longitudinal views (similar to tendon but with a much coarser pattern).
Fluid	Anechogenic, often with acoustic enhancement (see section on artifacts) deep to it, provided the collection of fluid is sufficiently large.
Pus	Depends on its consistency but will have a variable number of internal echoes ± reverberation artifacts if gas is present.
Gas/air	Produces reverberation artifacts unless suspended in small bubbles, characterized by numerous bright specular reflections. Often casts a combination of acoustic shadow and reverberation artifact. Tends to collect in the uppermost area of the cavity containing it.
Foreign bodies	Are usually strongly hyperechogenic and produce either acoustic shadowing or reverberation artifact beneath (the so-called comet tail with metal foreign bodies). Many foreign bodies (especially metal) have very thin echoes at their surfaces (cf. bone).

increase in tendon cross-sectional area, without any alterations in its internal architecture, is one of the earliest signs of tendinitis. The size of the structure should be compared with the unaffected leg (beware of bilateral injuries) or published values for the cross-sectional area of a particular tendon at a specific level on the limb. The dorsopalmar and lateromedial dimensions are less accurate.

When enlarged, certain structures will fill areas not normally occupied by that structure. For example, desmitis of the accessory ligament of the deep digital flexor tendon (ALDDFT or inferior check ligament) will result in obliteration of the hypoechoic space on either side of the ALDDFT between the deep digital flexor tendon (DDFT) and the suspensory ligament (SL).

CHANGES IN SHAPE

Enlargement of a tendon, or an eccentric lesion, will tend to alter its shape. Adhesions also can distort the shape of the tendon.

CHANGES IN POSITION

Swelling of certain structures can alter the position of other structures. For example, in severe superficial digital flexor tendinitis, tendon laxity and enlargement result in the movement of the superficial digital flexor tendon (SDFT) medially, displacing the DDFT laterally. Transection of one of the branches of the SDFT within the pastern results in shifting of the more proximal tendon to the opposite side of the limb.

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CHANGES IN MARGINATION/OUTLINE

The formation of adhesions often is accompanied by loss of definition in a tendon's borders. Adhesions are identified easiest in the digital sheath where the negative contrast of the synovial fluid (± additional contrast provided by intrasynovial injection of local anesthetic) gives the best definition. Small avulsion fractures of tendon and ligament insertions will manifest as disruptions in the normal bony contour.

Indications for Ultrasonographic Examination of the Musculoskeletal System

1. Investigation of soft tissue injury.
2. Investigation of the surface of bone.
3. Investigation of joint abnormalities.
4. Investigation of wounds.
5. Investigation of an area causing lameness, as determined by diagnostic anesthesia, with negative radiographic findings or radiographic findings that suggest a soft tissue injury.
6. Evaluation of structures likely to be concurrently or previously injured.
7. Comparison with the contralateral limb.

Technique for Ultrasonographic Examination of the Palmar/Plantar Aspect of the Metacarpal/Metatarsal and Phalangeal Regions

METACARPAL/METATARSAL REGION

The horse should be standing squarely with even weight distribution on both forelimbs, otherwise the tendons may vary in size and shape. Generally, in the metacarpal/metatarsal region, a stand-off or fluid offset is used for investigating tendon and ligament injuries, but further investigation of the SL and the ALDDFT can be carried out without any sound path delay.

KEY POINT

The transducer should be moved through arcs both lateromedially and proximodistally to obtain the optimum imaging angle. If resolution of the structures is difficult, slight tilting of the transducer off-incidence will highlight the borders of tendons and ligaments, although this will also generate central hypoechoic artifacts within the tendons and ligaments.

The transducer, with or without the stand-off initially, is applied to the palmar aspect of the limb in a horizontal (transverse) fashion. The procedure adopted for the examination must be methodical. Two recording systems have been described, and all have advantages and disadvantages. The distance of the transducer distal to the accessory carpal bone provides the most accurate system for recording and remeasuring at subsequent examinations. A tape with centimeter divisions from 0 to 30 cm can be fixed to the lateral aspect of the limb to assist accurate location and recording of the position of the transducer.

An alternative system of levels or zones has been described, where the metacarpal region is divided into seven equidistant regions: 1A, IB, 2A, 2B, 3A, 3B, 3C. Each zone has characteristic anatomic features (Fig. 3-1) from which they can be recognized. This system allows easier identification of the normal organization and size of the individual structures, irrespective of the size of the animal. The size of the tendons within each zone varies much less than the variation between individuals, so that cross-sectional areas, especially in the midmetacarpal region, also can be recorded satisfactorily using this system for future comparison.

KEY POINT

It is important to obtain at least two different images in perpendicular planes.

Therefore, after the transverse images, longitudinal images should be obtained with the transducer in line with the tendons. In acute injuries, this second orientation helps to differentiate artifacts from pathology, and, in chronic tendon and ligament injuries, the longitudinal images provide useful information on the quality of the tendon repair.

The number of longitudinal images depends on the size of the transducer and the length of the limb. However, usually three longitudinal views (proximal, mid-, and distal metacarpal) are sufficient. If a sector/curvilinear transducer is used, the beam only will be "on-incidence" in the center of the field of view so that only a small "ultrasound window" can be assessed.

KEY POINT

If injury to the suspensory ligament branches is considered, it is essential that the branches are evaluated by placing the transducer on the medial and lateral aspects of the limb, directly over the branches.

Both transverse and longitudinal images are obtained from the medial and lateral aspects. The

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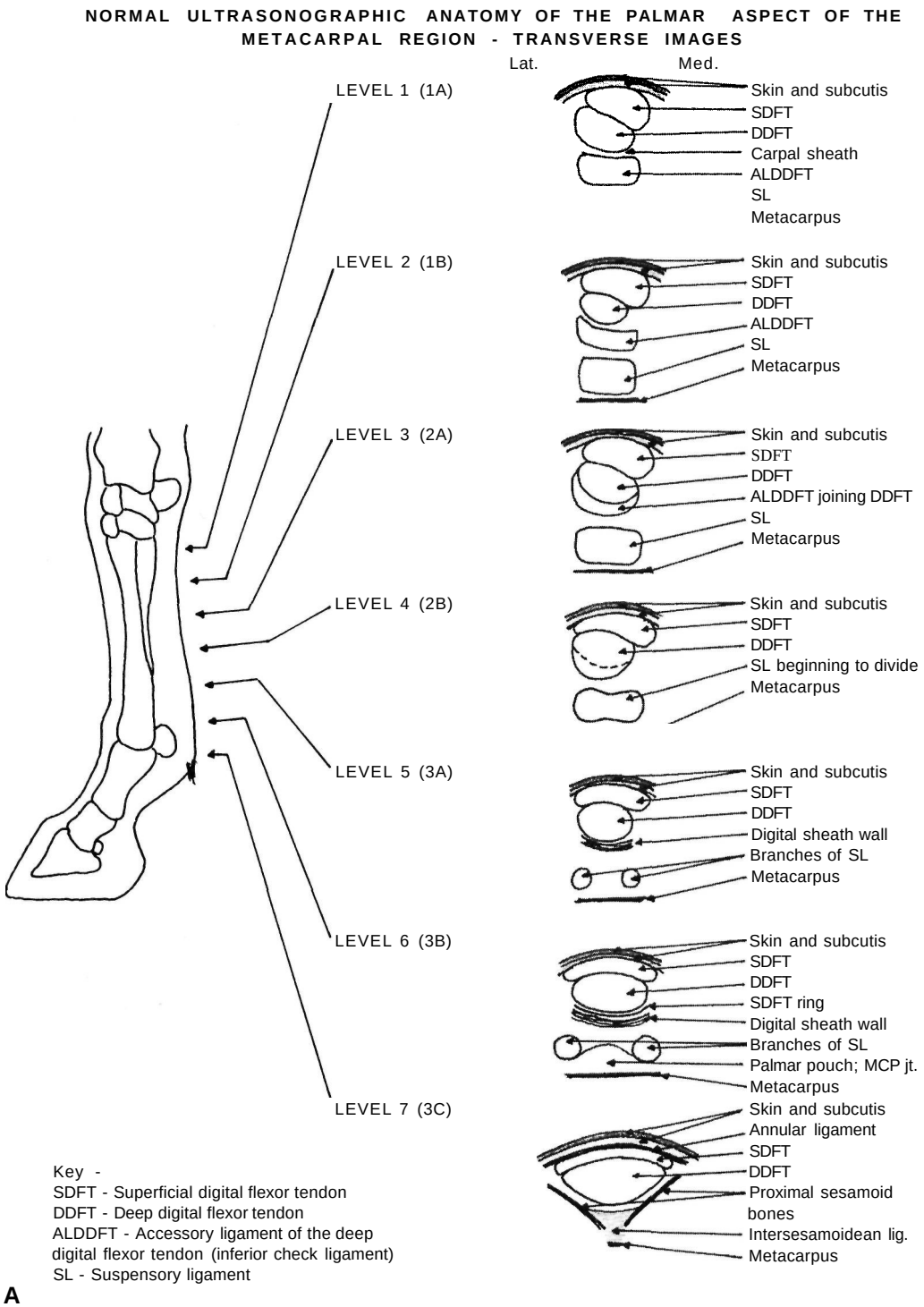
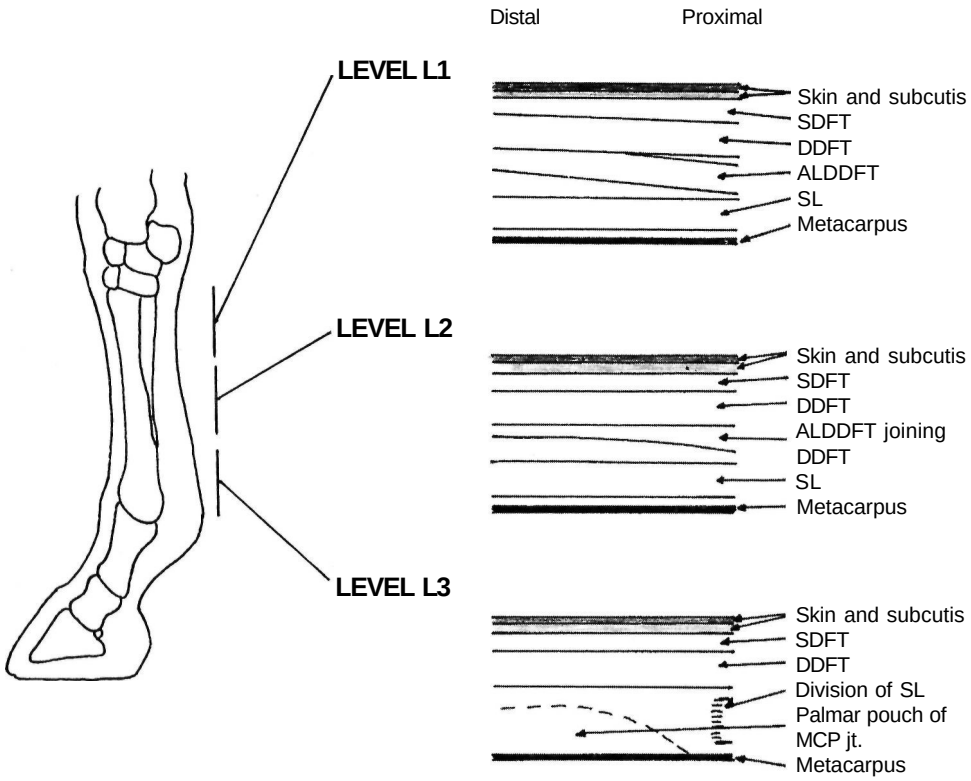


Figure 3-1. Diagrammatic representation of the normal ultrasonographic anatomy of the distal limb. *A,* Transverse probe orientation of the metacarpus from the palmar aspect; *B,* Longitudinal probe orientation of the metacarpus from the palmar aspect; and C, Transverse probe orientation of the palmar aspect of the fetlock and pastern. (A *B* From Hodgson, D.R., Rose R.J.: The Athletic Horse. Philadelphia, W.B. Saunders, 1994, pp. 300-301. C From Robinson, N.E.: Current Therapy in Equine Medicine 4. Philadelphia, W.B. Saunders, 1997, p. 64.)

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NORMAL ULTRASONOGRAPHIC ANATOMY OF THE PALMAR ASPECT OF THE METACARPAL REGION - LONGITUDINAL IMAGES



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

B

Figure 3-1 *Continued*

latter is sometimes termed the "ski-jump" view because of the shape of the abaxial surface of the proximal sesamoid bone. These extra views are necessary because edge refraction from the borders of the flexor tendons interferes with the assessment of the suspensory ligament branches from the palmar aspect of the limb, and the "ultrasound window" is frequently not wide enough to image the full extent of the branches. The branches increase in size in a proximodistal direction so that any comparison of cross-sectional area made between branches has to be made at the same level.

In the metatarsal region, the examination procedure is similar to the metacarpal region, using the seven zones, beginning immediately distal to the tarsometatarsal joint, with two extra levels included proximal to the tarsometatarsal joint for assessment of the plantar ligament and the superficial digital flexor tendon.

KEY POINT

It is recommended to examine both limbs for comparison and because of the high incidence of bilateral soft tissue injuries.

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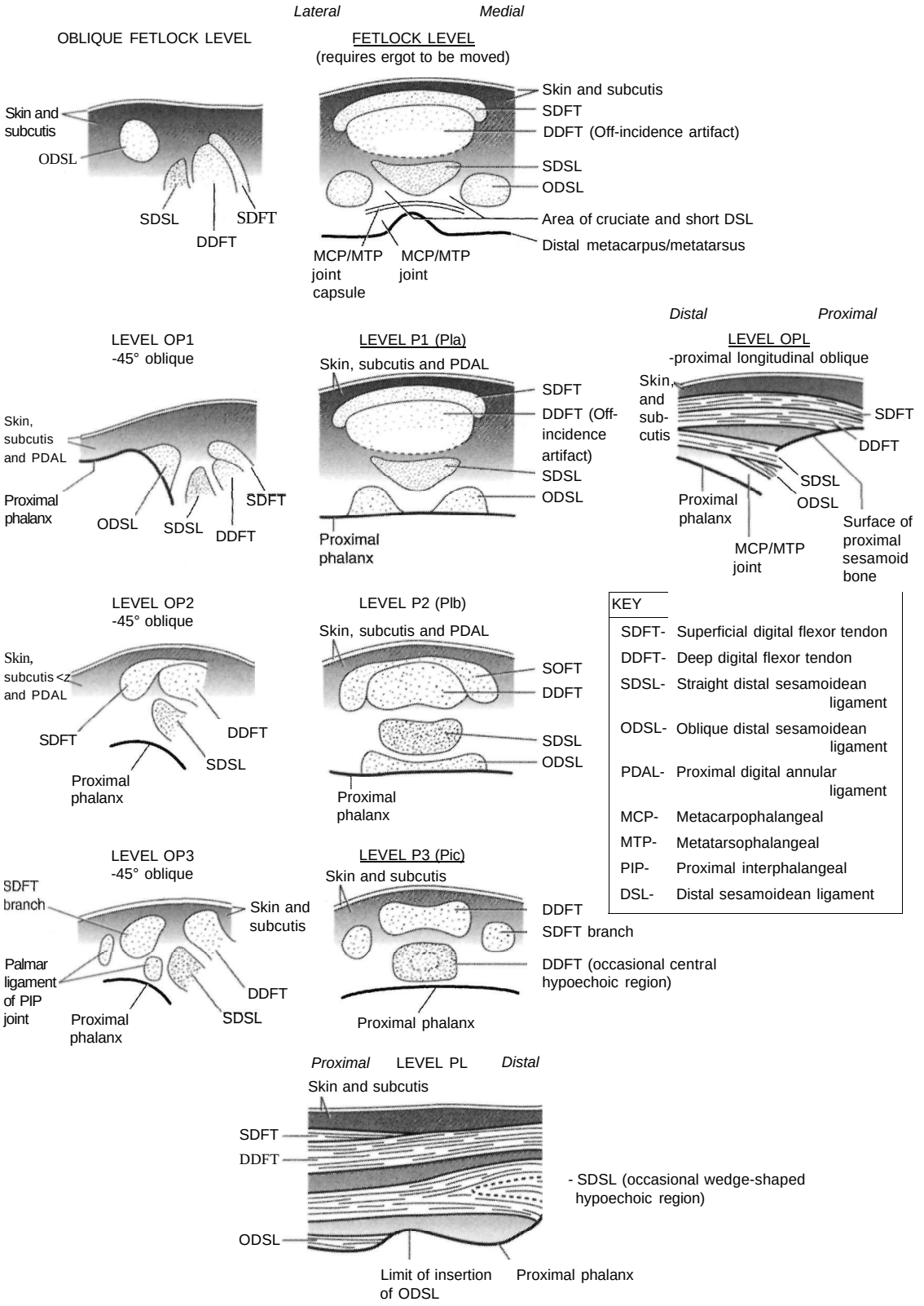


Figure 3-1 *Continued*

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PHALANGEAL REGION

The transducer is applied to the palmar/plantar aspect of the limb and three transverse levels assessed. Further distal levels have been described using a sector transducer, but they are difficult to obtain with a linear transducer in a limb bearing weight normally. The region further distal than the proximal interphalangeal joint can sometimes be imaged with the limb raised or, depending on the conformation of the distal limb (easier with a severely broken-back foot-pastern axis), with the limb placed further caudally with respect to the contralateral limb to hyperextend the distal interphalangeal joint. One longitudinal view also is usually assessed. The design of the transducer can prevent the correct "on-incidence" angle being achieved. Transducers with long handles can contact the ground, which can be avoided by placing the foot on a block, and the rectal probe can be turned so that the lead is proximal to prevent the interference of the lead with the bulbs of the heel. Oblique views are necessary to image the structures running obliquely in the pastern (e.g., the branches of the SDFT distally and the oblique distal sesamoidean ligaments proximally).

Conditions of the Palmar/Plantar Aspect of the Metacarpus/Metatarsus

SUPERFICIAL DIGITAL FLEXOR TENDINITIS

Acute Tendinitis

"Subclinical" or Early Signs of Tendinitis. Often the only finding in these cases is enlargement and/or change in shape of the tendon. This can be accompanied by peritendinous edema, which is not specific for tendinitis and can also result from local trauma. Providing there is no evidence of tendon injury and the edema disappears, work can be recommenced after only a short period of rest. However, persistent edema suggests the presence of tendinitis.

Clinical Strain Injuries. *Concentric (core) lesions* (Fig. 3-2) are the most common SDFT injury in racehorses and sports horses. A variable size hypoechoic (occasionally anechoic) lesion is visible in the center of the tendon, usually centered on the midmetacarpal region (adjacent to the bifurcation of the suspensory ligament). If the injury is localized to the region of the metacarpophalangeal joint or distally, then there is often evidence of previous injury to the midmetacarpal region.

Eccentric (noncentral) lesions are a less common manifestation.

Generalized hypoechogenicity may represent a tendon that is healing in which the core lesion has disappeared. If the injury is recent, however, this represents diffuse damage to the tendon and/or intratendinous edema.

Mixed echogenicity is most commonly seen with recurrent or partially healed injuries. There is a combination of hyper- or normoechogenicity associated with healed tendon and scattered hypoechoic foci representing more recent injury.

Rupture of the SDFT (Fig. 3-3) is manifested by an almost totally anechoic region of the SDFT surrounded by a thin echogenic line (the paratenon). Evidence of damage will also be apparent proximal and distal to the rupture. If the tendon ends have retracted, the outline of the paratenon at the site of the rupture may not be particularly enlarged. Bunched-up retracted fibers will be identifiable proximal and distal to the rupture site.

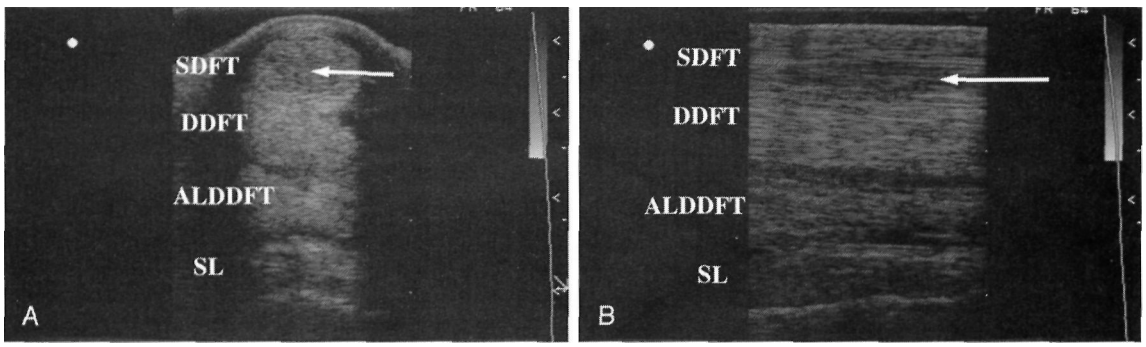


Figure 3-2. *A,* Transverse view from the proximal metacarpal region. Central hypoechoic lesion within the superficial digital flexor tendon, characteristic of superficial digital flexor tendinitis. Note the thin ring of edema around the tendon, giving better definition to the tendon. *B,* Longitudinal view from the proximal metacarpal region of the same limb as in *A.* Superficial digital flexor tendinitis is represented in longitudinal view by loss of the normal linear striated pattern with the superficial digital flexor tendon. SDFT, superficial digital flexor tendon; DDFT, deep digital flexor tendon; ALDDFT, accessory ligament of the deep digital flexor tendon; SL, suspensory ligament.

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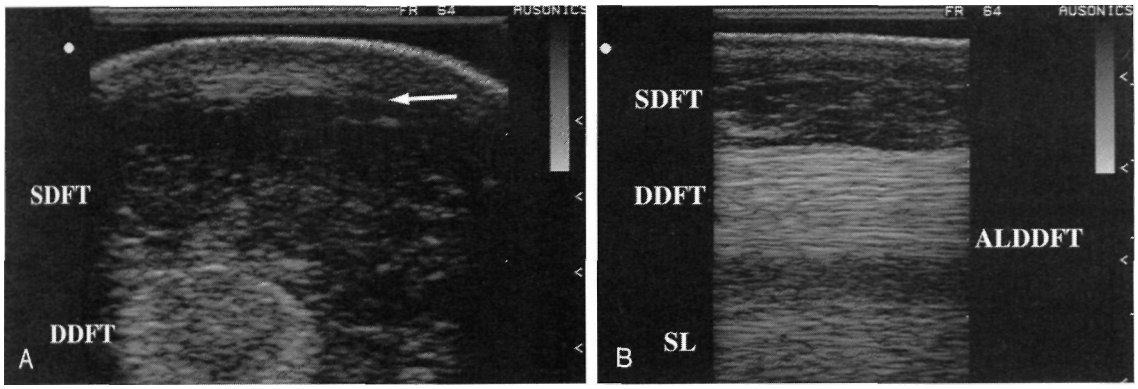


Figure 3-3. Rupture of the superficial digital flexor tendon. These scans were obtained from the midmetacarpal region and show almost complete disruption of the superficial digital flexor tendon in both *(A)* transverse and *(B)* longitudinal images. The damaged tendon is surrounded by an intact paratenon as represented by the thin echogenic line. 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.

Local Trauma. The effects of local trauma can vary from localized peritendinous edema with no evidence of intratendinous damage to localized hypoechoic/anechoic lesions on the palmar surface of the tendon. Local traumatic injuries do not extend far proximodistally.

Sepsis. Sepsis of the tendon tissue is rare and usually gives an anechoic lesion, often with a communicating tract to the periphery of the tendon. Aspiration of the lesion will yield a sample containing large numbers of degenerate neutrophils. These lesions do not usually cause gross enlargement of the affected tendon and change rapidly in time in comparison with the core lesion in a tendon strain. If the lesion is present within a tendon sheath, there will usually be an accompanying septic tenosynovitis.

Chronic Tendinitis

The ultrasound characteristics of chronic tendinitis are more subtle. The tendon often is enlarged, but its echogenicity varies from hypoechogenic through normoechogenic to hyperechogenic if the injury was severe and substantial fibrosis has occurred. The intratendinous pattern is usually more coarse, with a lack of striations in the longitudinal images. It is these, and not the transverse, images that are most helpful in diagnosing chronic tendinitis. In some cases, the outline of the original core lesion can still be seen. Mineralization may occur that causes acoustic shadowing. If the calcification is florid, previous intratendinous injection of depot corticosteroids should be suspected.

With the increased definition of the newer machines and the higher frequencies used (10 MHz upward), subtle changes in both the transverse and longitudinal patterns can be identified in horses without any history of clinical signs of tendinitis (Fig. 3-4). The significance of these changes is unclear at this time, but they may represent cumulative microdamage associated with aging and/or exercise.

Semiquantitative Assessment of Tendon Injury

Objective measurements allow a better determination of prognosis and assessment of healing. The following measurements have been suggested:

1. Cross-sectional area (transverse image). Most horses should have cross-sectional areas of less than 1.2 to 1.6 cm^2, although measurements can vary among software packages.
2. Percentage of damaged tendon (transverse image) (see Table 3-3).
3. Type of lesion (i.e., degree of echogenicity): *type 1,* lesion is hypoechoic, more white than black; *type* 2, lesion is hypoechoic, same amounts of white and black; *type 3,* lesion is hypoechoic, more black than white; *type 4,* lesion is anechoic, totally black.
4. Fiber alignment score (longitudinal image): 1 to 3 (1, best, to 3, no fiber alignment).

In addition, the values for each individual level can be summed to give a "total" value.

Such semiquantitative assessment requires a machine with the appropriate software application, to allow the measurements to be made, or separate digitizing equipment. These measurements provide a more sensitive indicator of the progress of the case but are time consuming.

Assessment of Healing

All tendon injuries should ideally be monitored ultrasonographically at three monthly intervals or before a change in exercise level. At each examination, the following indicates good progress:

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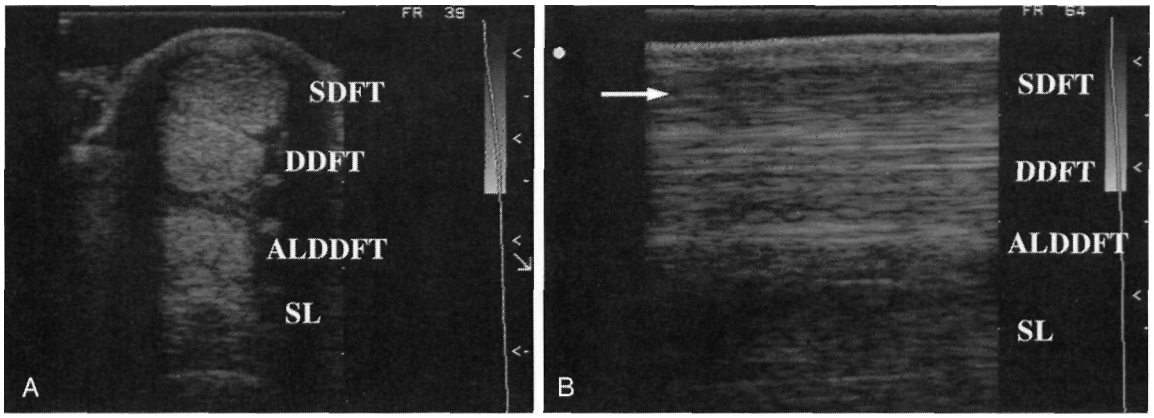


Figure 3-4. These images were obtained from the proximal metacarpal region in the contralateral limb to that in Figure 3-2. Note the more coarse appearance of the tendon with hypoechoic and hyperechoic areas. These changes can represent chronic tendinitis or reinjury of a chronic tendinitis. Aging changes within the superficial digital flexor tendon (SDFT) can look very similar. *A.* Transverse view, proximal metacarpal region. *B.* Longitudinal view, midmetacarpal region. Note the loss of the linear striated pattern in the superficial digital flexor tendon. DDFT, deep digital flexor tendon; ALDDFT, accessory ligament of the deep digital flexor tendon (inferior check ligament); SL, suspensory ligament.

1. Stable or decreasing cross-sectional area;
2. Increase in the lesion echogenicity and a homogeneous texture;
3. Improvement in the striated pattern seen longitudinally (fiber alignment);
4. Absence of peritendinous fibrosis and adhesions.

If such improvements are seen, then the exercise level can be increased. An increase in the cross-sectional area probably provides the most sensitive indicator that the tendon is not able to withstand the work being delivered to it, and therefore any increase in cross-sectional area should initiate a decrease of the exercise level.

Horses suffering from tendinitis are constantly at risk of reinjury. Healing, determined histologically, takes at least 15 months. The mean interval between injury and return to training in racehorses depends on the severity of the initial injury. Studies have shown this to average between 9 and 18 months, as shown in Table 3-3. Sports horses may be able to return to full work in a shorter time, but event the mildest ultrasonographically detectable injuries should have at least 6 months off. Occasionally, horses are returned to full work before full resolution of the ultrasonographic lesion; however, this success may be due to the horse being capable of sustaining work despite the presence of a tendon injury.

Formulation of Prognosis

Prognosis depends on a number of factors, of which the major component is the severity of the

TABLE 3-3. Intervals Between Injury and Return to Training in Racehorses

Severity of Tendinitis	Criteria for Severity (Marr 1993)	Criteria for Severity (Genovese et al. 1996)	Time off Training
Slight/mild	<50% of CSA affected and/or < 100 mm in length	<15% total hypoechoic*	9-10 mo
Moderate	50-75% of CSA affected and/or 100-160 mm in length	16-25% total hypoechoic*	11 mo
Severe	>75% of CSA affected and/or >160 mm in length	>25% total hypoechoic*	12-18 mo

*% total hypocchoic $= \frac{\text{sum of the cross-sectional areas of the lesion at seven equidistant levels}}{\text{sum of the tendon cross-sectional areas at seven equidistant levels}}$

CSA, cross sectional area.

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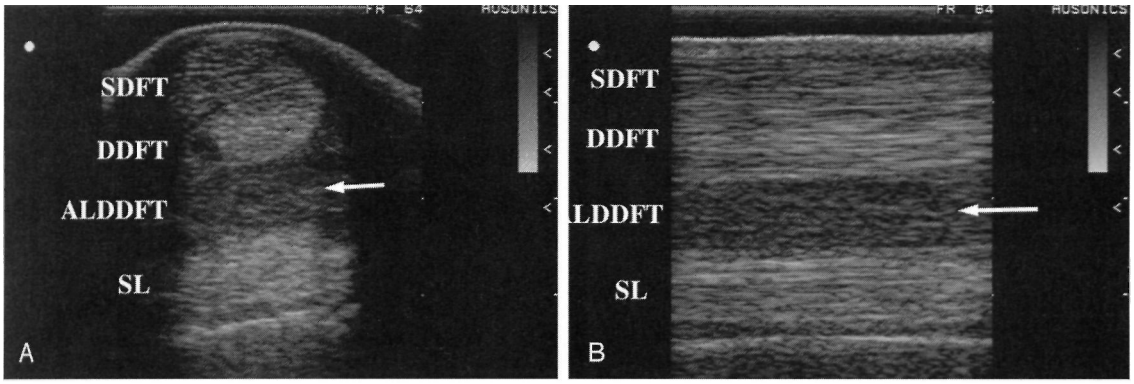


Figure 3-5. Desmitis of the accessory ligament of the deep digital flexor tendon. *A,* Transverse image from the proximal metacarpal region. Note the enlarged and hypoechoic ligament deep to the deep digital flexor tendon *(arrow). B,* Longitudinal image from the midmetacarpal region. Note the obliteration of the normal "space" between the accessory ligament of the deep digital flexor tendon and the suspensory ligament by the swollen check ligament *(arrow).* There is a loss of the normal striated pattern in the ligament.

initial injury. Ultrasound is essential for providing the most accurate assessment of the degree of damage. In a study of National Hunt and Point-to-Point racehorses undergoing conventional treatment with or without additional therapy with glycosaminoglycans or laser therapy, a successful return to racing was achieved in 63%, 30%, and 23% of mild, moderate, and severe injuries respectively (Marr et al. 1993a).

There are other variables that may affect the prognosis, such as the nature of the work intended, age, breed, treatment, episodes of reinjury, and the rest period.

ACCESSORY LIGAMENT OF THE DEEP DIGITAL FLEXOR TENDON DESMITIS

The ultrasonographic appearance of this condition is variable. Changes range from diffuse hypoechoicity to focal hypoechoic areas. The ligament is enlarged and will usually obliterate the space either side of the ALDDFT, between the DDFT and the SL (Fig. 3-5). There may be extension of the ALDDFT to the borders of the SDFT, indicating adhesion formation and/or concurrent superficial digital flexor tendinitis. Such adhesions can result in secondary flexural deformities of the distal limb. The prognosis for isolated desmitis of the ALDDFT is better than for superficial digital flexor tendinitis (77% return to work) and requires 3 to 9 months (typically 4 to 6 months) off work. The prognosis appears to be unrelated to the initial severity, although a longer convalescent period may be required for the most severely affected horses. A successful outcome can be achieved even in the presence of poor quality healing, as judged by the criteria described above for superficial digital flexor tendinitis. Ultrasonography is useful during the convalescent period to determine when the horse can return to work.

Desmitis of the ALDDFT in the hindlimb (subtarsal check ligament) is rare but exhibits similar changes to its forelimb counterpart (Fig. 3-6). Clinically, the swelling it causes in the proximal metatarsal region can be mistaken for plantar ligament desmitis, deep digital flexor tendinitis, or desmitis of the proximal suspensory ligament, and

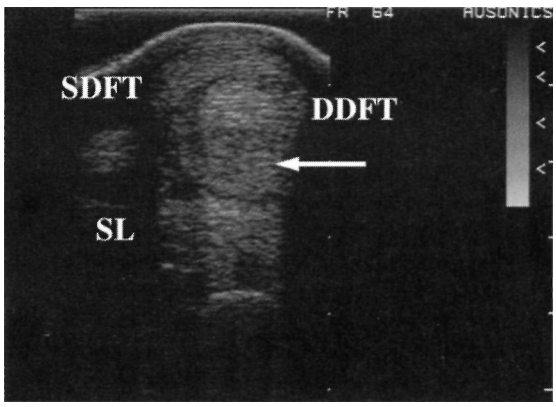


Figure 3-6. Desmitis of the accessory ligament of the deep digital flexor tendon in the hindlimb. The subtarsal check ligament is normally a thin echogenic structure immediately deep to the deep digital flexor tendon. In this transverse scan from the proximal metatarsal region of a horse suffering from desmitis of the accessory ligament of the deep digital flexor tendon, the ligament is enlarged and has altered echogenicity *(arrow).* SDFT, superficial digital flexor tendon; DDFT, deep digital flexor tendon; SL, suspensory ligament.

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ultrasonography is very useful to differentiate these conditions.

DESMITIS OF THE SUSPENSORY LIGAMENT (MUSCULUS INTEROSSEUS MEDIUS)

There are three regions of injury: proximal, body, and branch.

1. Proximal suspensory desmitis—also called "high suspensory disease," proximal metacarpal syndrome. The ultrasonographic appearance of this injury has considerable overlap with the normal appearance. The presence of hypoechoic areas in the proximal suspensory ligament is common in normal horses, and therefore the significance of such findings must be interpreted in the light of clinical findings (swelling, pain on palpation) and diagnostic local analgesia. Those lesions considered to be a true suspensory ligament desmitis will vary in time, and so repeat examinations are useful to confirm their significance.

Ultrasonographic features of injury include enlargement of the suspensory ligament, poor definition to the margins (especially dorsally), single or multiple poorly defined focal areas of hypoechoicity, diffuse hypoechoicity, and irregularity of the palmar surface of the proximal metacarpus/metatarsus, indicative of enthesiophytosis. In addition to the ultrasonographic abnormalities, radiographic changes, such as sclerosis and altered trabecular pattern at the origin of the suspensory ligament, also may be present, with or without concurrent increased radionuclide uptake on gamma scintigraphy in the proximal metacarpal/metatarsal regions. Differential diagnoses include palmar cortical fractures, which usually have no abnormalities within the proximal suspensory ligament and higher uptake on gamma scintigraphy, and avulsion fractures of the head of the suspensory ligament where abnormalities in the suspensory ligament often are confined to an area immediately adjacent to the site of the avulsion fracture.

The period of rest required is similar to that for desmitis of the ALDDFT (3-9 months), and ultrasound is used to direct the ascending exercise regimen that horses should undergo. Uncomplicated cases in the forelimb have a greater than 80% chance of returning to work, whereas those in the hindlimb carry a worse prognosis, with only 17% returning to work in one study (Dyson 1994).

2. Desmitis of the body of the SL. If this area is injured, usually there is generalized hypoechogenicity and enlargement to the ligament. In competition/sports horses, the injury often extends into the branches of the SL.

There is some controversy over the link between suspensory desmitis and splints. Some aggressive exostoses on the second or fourth metacarpal/metatarsal bones may impinge on the body of the suspensory ligament and cause a localized suspensory desmitis, but this probably occurs only in some cases. Many exostoses grow around rather than into the suspensory ligament. Careful assessment by oblique positioning of the ultrasound transducer is necessary because the ultrasound "window," when the transducer is placed on the palmar/plantar aspect, does not usually extend sufficiently abaxially to image these areas adjacent to the splint bones.

3. **Desmitis of the suspensory ligament branches.**

KEY POINT

The suspensory ligaments of both limbs should be evaluated ultrasonographically because biaxial and bilateral involvement is common.

This is the most common of the suspensory ligament injuries in sports horses. In the forelimbs, biaxial desmitis has the highest incidence, whereas lateral branch desmitis is the most common manifestation in the hindlimbs. A core lesion or generalized involvement of the branch, together with enlargement, is seen ultrasonographically (Fig. 3-7). The longitudinal image from the abaxial aspect gives an excellent assessment of the abaxial surface of the proximal sesamoid bones where any associated enthesiopathy is demonstrated by steps in the S-shaped surface of the bone. The size of the suspensory ligament branches should be compared with both contra-axial and contralateral branches at the same level as the branches increase in size in a proximodistal direction.

KEY POINT

One of the most sensitive indicators of suspensory branch desmitis is periligamentar fibrosis, which is extremely common in this condition. It has the effect of "moving" the suspensory ligament branch away from the skin (see Fig. 3-7).

Suspensory desmitis is frequently associated with other abnormalities of the limb. Horses with straight hocks and long sloping pasterns are thought to be predisposed to degenerative lesions, typified by branch enlargement. Desmitis can occur concurrently with other soft-tissue injuries, such as superficial digital flexor tendinitis or distal sesamoidean ligament pathology.

Clinical and radiographic examination of the metacarpo/metatarsophalangeal joint is also rec-

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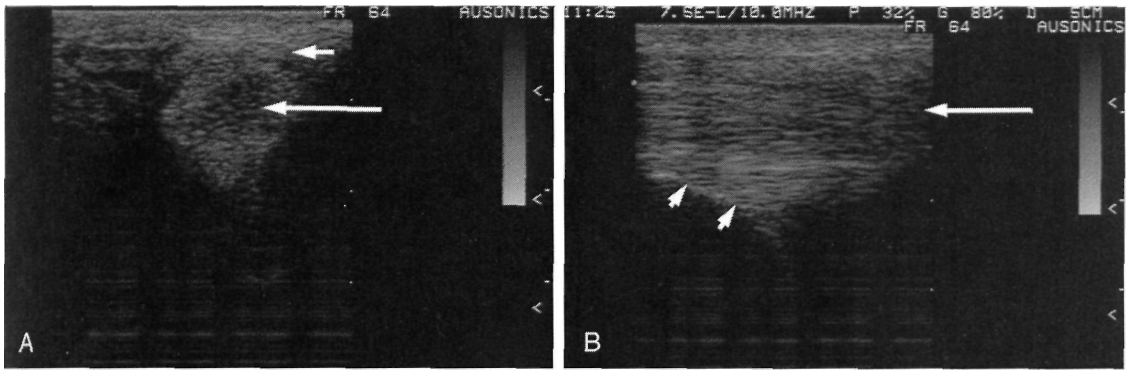


Figure 3-7. *A,* Transverse ultrasonograph obtained from the medial side of the limb, directly over the medial suspensory ligament branch, in a horse with suspensory ligament branch desmitis. Note the ring of periligamentar fibrosis *(short arrow),* characteristic of suspensory ligament branch desmitis, and the enlarged, and centrally hypoechogenic, suspensory ligament branch *(long arrow). B,* Longitudinal image of the same suspensory ligament branch, with a reduced striated pattern *(long arrow),* inserting on the abaxial surface of the proximal sesamoid bone *(short arrows).*

ommended in cases of suspensory ligament desmitis because concurrent pain and pathology in this joint is frequently present, because of the nature of the injury (hyperextension). Furthermore, radiography will reveal bony pathology that frequently is associated with the suspensory ligament body and branch desmitis, such as enthesiopathy of the proximal sesamoid bones ("sesamoiditis") and distal splint bone fractures.

The management of suspensory ligament body and branch desmitis benefits from regular ultrasound examinations similar to that carried out for the SDFT. Similar criteria are used to direct the ascending exercise regimen, although the quality of healing generally is inferior to that seen in the SDFT. Between 3 and 12 months is needed for the ligament to heal, but reinjury and injury to previously unaffected branches can occur later, so that suspensory desmitis must always carry a guarded prognosis. Some lesions may persist despite careful management, and in those cases, horses can be returned to work after an appreciable period of rest when the lesions fail to alter on sequential ultrasound examinations. However, the owner should be made aware that these animals represent an increased risk of reinjury.

DEEP DIGITAL FLEXOR TENDINITIS

KEY POINT

Deep digital flexor tendinitis is extremely rare in the metacarpal region as a primary injury. Its usual site of injury is within the digital sheath (see below).

Localized damage can be identified associated with ALDDFT desmitis, although this often represents extension of the damaged check ligament fibers into the body of the DDFT. DDFT tendinitis can occasionally be seen in the metatarsal region, especially proximally, where it is associated with tarsal sheath distension.

Conditions Spanning the Metacarpo/ Metatarsophalangeal Joint

DIGITAL SHEATH TENOSYNOVITIS

Tenosynovitis of the digital sheath is relatively common and can be either primary (e.g., sepsis, repeated overextension) or secondary to injury of associated tendon and ligaments. When examining the digital sheath ultrasonographically, the whole length of the digital sheath should be evaluated from distal metacarpal/metatarsal region to foot. The ultrasonographic signs of tenosynovitis are

1. *Effusion*—especially lateral and medial to the flexor tendons in the proximal digital sheath and distally in the pouch on the palmar aspect of the distal pastern.
2. *Thickening of the synovial sheath wall*—best assessed dorsal to the flexor tendons in the distal metacarpal region.
3. *Adhesions*—care should be taken to avoid misdiagnosing the normal synovial plicae in the proximal digital sheath as adhesions (Fig. 3-8). They are normal structures joining the medial and lateral borders of the DDFT to the sheath wall in the proximal digital sheath and are usually only visible ultrasonographically when there is distension of the sheath. However, although normal structures, they are a useful indicator of synovial membrane hypertrophy or edema.

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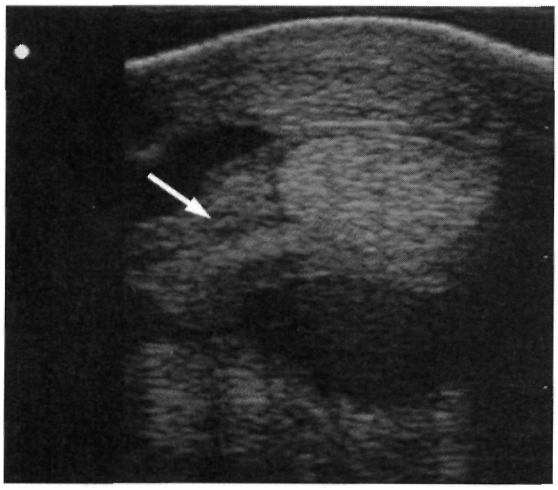


Figure 3-8. Transverse ultrasonograph from the distal metatarsal region in a horse with digital sheath tenosynovitis. Note the fluid distension of the sheath and the thickened lateral synovial plica, stretching from the wall of the digital sheath to the deep digital flexor tendon *(arrow).* This is a normal structure but grossly thickened because of the presence of inflammatory tenosynovitis. SDFT, superficial digital flexor tendon; DDFT, deep digital flexor tendon.

4. *Mineralization in the wall of the digital sheath*—will manifest ultrasonographically as focal hyperechoic foci casting acoustic shadows. They are more common if the sheath has been medicated in the past with corticosteroids.

5. *Concurrent thickening of the fetlock annular ligament* (see below)—section of this ligament, especially if thickened, may help these cases.

The prognosis relates to the degree of lameness and its chronicity. The presence of adhesions warrants a guarded prognosis, and recurrent episodes of lameness are common in these cases.

Sepsis of the digital sheath will show similar changes to that described for aseptic tenosynovitis, but they will be more severe. There is usually considerable thickening of the synovial membrane and subcutaneous edema. The epitenon surrounding each tendon swells and results in a hypoechoic "halo" around the tendon, which is especially evident on the DDFT in the pastern (Fig. 3-9). The effusion may have a floccular nature associated with the collection of fibrin. A synovial fluid sample is necessary to confirm the diagnosis (see Chapter 17). Prognosis is poor if sepsis is established and there are adhesions present. Immediate aggressive treatment, including lavage of the sheath and antibiotics, is essential to have the best chance of a successful outcome.

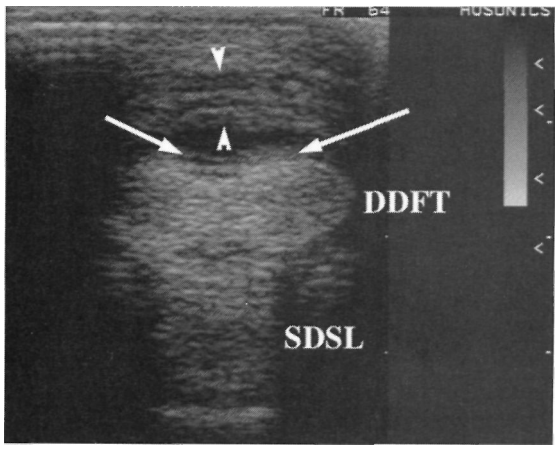


Figure 3-9. Transverse scan from the distal pastern region in a horse with septic tenosynovitis of the digital sheath. There is considerable thickening of the subcutaneous tissues and digital sheath wall *(between arrowheads),* and a hypoechoic "halo" around the deep digital flexor tendon *(long arrows),* which accompanies severe inflammatory tenosynovitis.

DEEP DIGITAL FLEXOR TENDINITIS

Deep digital flexor tendinitis usually occurs within the digital sheath and is a rare injury. Ultrasonographic changes vary from severe disruption of the tendon to localized hypoechoic lesions proximal or distal to the fetlock joint, with or without focal areas of mineralization (Fig. 3-10). The lameness associated with deep digital flexor tendinitis is moderate to severe and of long duration.

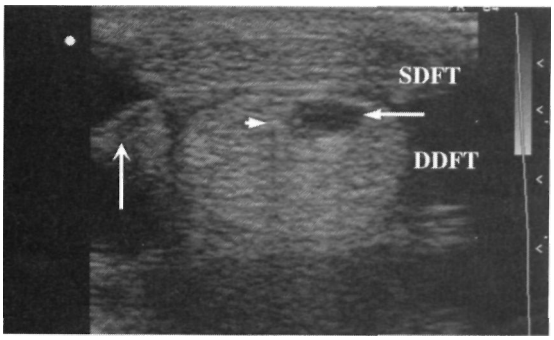


Figure 3-10. Transverse ultrasonograph from the distal metatarsal region showing a localized hypoechoic lesion within the deep digital flexor tendon *(long horizontal arrow),* with a small focus of mineralization casting an acoustic shadow *(short arrow).* The pain was localized to this region clinically. Note also the accompanying tenosynovitis with a thickened lateral synovial plica *(long vertical arrow).* SDFT, superficial digital flexor tendon; DDFT, deep digital flexor tendon.

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There is usually concurrent digital sheath tenosynovitis and subcutaneous fibrosis. The prognosis is poor, and the lesions can take up to 12 to 18 months to resolve. Adhesion formation is common.

Penetrating wounds to the region of the phalanges are common in the horse, and these injuries should always be examined ultrasonographically early on for signs of DDFT injury and digital sheath violation (Fig. 3-11). Occasionally, sepsis of the DDFT can develop, causing localized hypoechoic/anechoic areas with tracts to the periphery of the tendon. Synovial distension of the sheath indicative of septic tenosynovitis usually is present. As in all cases of sepsis within a tendon, these lesions are rapidly destructive, and surgical debridement is indicated as soon as possible.

Small curvilinear transducers have been used between the bulbs of the heel to image distally to the level of the proximal border of the navicular bone. However, at this level the tendon is "off-incidence" and appears hypoechoic. Healing penetrations can be identified at this site by the presence of echoes (haphazardly arranged scar tissue collagen), but the DDFT directly over the flexor surface of the navicular bone cannot be reliably imaged. Therefore it is rare to document ultrasonographic changes in cases of navicular disease. Mild enlargements without alterations in the internal architecture of the tendon occasionally are seen.

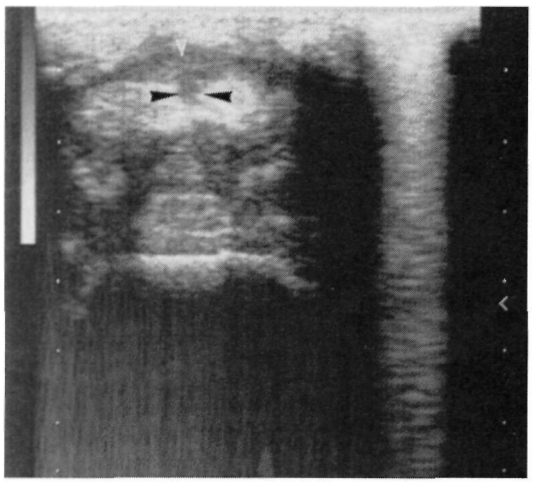


Figure 3-11. Transverse ultrasonography from the distal pastern region in a horse that had suffered a penetrating injury to the plantar pastern. Note the defect on the plantar surface of the deep digital flexor tendon *(black arrowheads)* and the overlying echogenic material *(white arrowhead)*. The latter may progress to form a restrictive adhesion.

ANNULAR LIGAMENT SYNDROME

This syndrome is characterized by thickening of the palmar/plantar annular ligament (PAL) and its associated structures and tenosynovitis of the digital sheath. These abnormalities vary in extent between cases. The syndrome can be divided into primary and secondary forms. *Primary annular ligament syndrome* (ALS) is characterized by pathology within the PAL, caused by local trauma or chronic overstretching. Ultrasonography reveals no damage to the flexor tendons, and in the early stages of the condition, there may be signs of desmitis of the annular ligament (regions of hypoechoicity and anechoicity). *Secondary ALS* occurs as a result of primary pathology within associated soft tissue structures, such as the digital sheath (chronic aseptic or septic tenosynovitis) or digital flexor tendons (tendinitis). The pain arises from a relative constriction of the structures within the fetlock canal, although this has recently been disputed in some cases where enlargement of the tendons is not present. An appreciation of the restriction to the normal gliding movement of the tendons within the fetlock canal can be obtained by examining the area clinically and ultrasonographically while the digit is flexed and extended.

In addition to injuries to the flexor tendons within the sheath in secondary ALS, the ultrasonographic characteristics are digital sheath tenosynovitis (including effusion and variable thickening of the digital sheath wall, with or without adhesions) and variable thickening of the palmar aspect of the fetlock, variably involving three components:

1. The digital sheath synovial membrane, which is relatively hypoechogenic immediately palmar to SDFT.
2. The PAL, which has an echogenic structure with striations running transversely. In the normal horse, the annular ligament can be difficult to identify, especially in the midline where a vinculum attaching the PAL to the SDFT obscures its borders. It can be more easily identified if the transducer is moved to the palmarolateral or palmaromedial aspects to observe its attachment to the palmar border of the proximal sesamoid bone. Thickening of the annular ligament to over 2 mm is considered diagnostic of ALS, although, because of the other components, the distance between the palmar surface of the SDFT and the skin surface may be a better measure. This has been measured as 3.6 ± 0.7 mm in normal horses. Therefore, a distance of greater than 5 mm should be considered abnormal. It is usually possible to make both measurements to differentiate real enlargement of the PAL and associated fibrosis or

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synovial hypertrophy. In chronic cases of ALS, enthesiophytosis of the PAL attachment zones may be seen either radiographically or ultrasonographically as irregular new bone on the palmar borders of the proximal sesamoid bones (Fig. 3-12).

3. The subcutaneous tissues, which have variable echogenicity but usually less than the annular ligament.

It is important to evaluate the whole of the digital sheath and the PAL to determine the therapeutic approach and prognosis. In uncomplicated primary PAL thickening, surgical transaction of the PAL results in approximately 70% return to work. If adhesions are observed ultrasonographically, the prognosis is worse. In secondary ALS, with solely digital tenosynovitis, aggressive treatment of the tenosynovitis is warranted in the first instance, although surgical transection of the PAL should still be considered in unresponsive cases. Symptomatic relief can be achieved through sectioning the PAL in secondary ALS associated with superficial or deep digital flexor tendinitis, although the prognosis relates more to the tendon injury than the PAL thickening.

Conditions of the Palmar/Plantar Aspect of the Phalanges

KEY POINT

Most soft tissue injuries of the pastern create considerable edema and/or subcutaneous fibrosis, making detailed palpation difficult. Ultrasonography is therefore useful to differentiate the soft tissue structures involved.

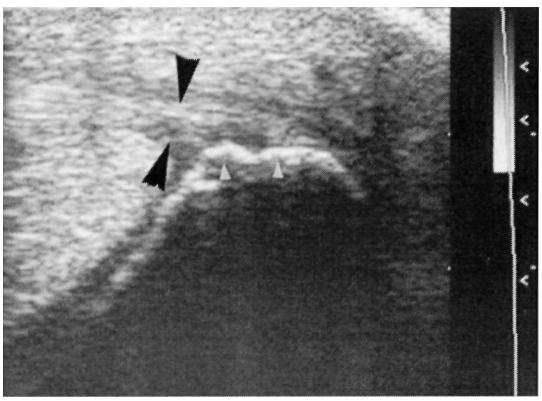


Figure 3-12. Oblique transverse ultrasonograph of the insertion of the palmar annular ligament *(between black arrowheads),* showing enthesiopathy at the insertion of the ligament *(white arrowheads).*

TENDINITIS OF THE SDFT BRANCHES

Injury to SDFT branches often occurs subsequent to injury to the midmetacarpal region of the SDFT, so this area should also be examined ultrasonographically. Clinically, there is swelling of the palmaroproximal aspect of the phalanges, uniaxial if only one branch is affected, which can be difficult to distinguish from distal sesamoidean desmitis. Ultrasonographically, there is enlargement of the SDFT branches.

Treatment of this condition is similar to that for superficial digital flexor tendinitis; the mainstay is initial rest, followed by a controlled ascending exercise regimen, with regular ultrasound monitoring. Prognosis depends on the presence of concurrent pathology but must remain guarded because reinjury is common.

DISTAL SESAMOIDEAN LIGAMENT DESMITIS

Injury to these structures is rare. Enlargement and hypoechogenicity of either or both of the oblique and straight distal sesamoidean ligaments can be identified ultrasonographically. The short and cruciate distal sesamoidean ligaments cannot be reliably identified. There is a normal hypoechoic central region at the insertion of the straight distal sesamoidean ligament on the middle scutum that should not be misdiagnosed as a desmitis. In true desmitis, there will be concurrent enlargement of the ligament, an abnormal longitudinal striated pattern, and subcutaneous fibrosis. There can be concurrent injury to other soft tissue structures on the palmar/plantar aspect of the limb. Radiographic evidence of enthesiophytosis in these ligaments also may be present, although enthesiophytosis at the insertion of the oblique distal sesamoidean ligaments on the palmar/plantar aspect of the proximal phalanx may not be clinically significant.

These injuries usually require an extended period of rest, and reinjury is common, thus warranting a guarded prognosis. Ultrasonography can be used to monitor the healing in an attempt to modify exercise programs, although persistent lesions are common.

Ultrasonography of Other Soft Tissues

TENDONS AND TENDON SHEATHS OVER THE DORSAL ASPECT OF THE CARPUS

Each tendon and its accompanying sheath can be imaged over the dorsal aspect of the carpus. Tendinitis and tenosynovitis usually are induced by percutaneous trauma. Chronic hypertrophy of the tendon sheaths, which limit carpal flexion and/

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or cause lameness, are suitable candidates for surgical debridement. Ultrasonography can help to determine the nature of the swelling over the carpus, identify which tendon sheaths (or acquired bursa) are involved, and locate any foreign bodies.

GASTROCNEMIUS TENDON, DEEP TARSAL TENDONS, AND SDFT OVER THE POINT OF THE HOCK

The gastrocnemius tendon wraps around the SDFT laterally proximal to the point of the hock and comes to lie deep to the SDFT at the point of the hock. The calcaneal bursa separates the SDFT from the gastrocnemius tendon, but it is only identifiable ultrasonographically medial and lateral to the tendons. An acquired subcutaneous bursa is variably present superficial to the SDFT over the point of the hock. Tendinitis of the gastrocnemius tendon and/or the deep tarsal ligaments has been described and is manifest as enlargement and heterogeneity of the affected tendons. Gastrocnemius tendinitis commonly is associated with calcaneal bursal distension. A period of 6 to 12 months is necessary for the condition to heal, but lesions can persist.

The retinaculae binding the SDFT to the point of the hock can be easily identified ultrasonographically. Rupture of the medial band or a longitudinal split in the SDFT can be identified in cases of persistent or intermittent dislocation of the tendon from the point of the hock. Intermittent dislocation carries a poorer prognosis.

BICIPITAL TENDON AND BURSA

These structures are easily imaged over the cranial aspect of the shoulder. Enlargement and disruption of the bicipital tendon is rare, whereas bicipital bursal distension is more common, frequently associated with trauma and sepsis, and can be identified caudal to the tendon. Calcification of the bicipital tendon has been identified cranial to the shoulder joint, where it causes acoustic shadowing.

Ultrasonography of Other Structures

JOINTS

Ultrasonography is becoming an increasingly useful imaging modality for the investigation of joint abnormalities. Ultrasonography is useful to identify the following:

1. *Joint effusion,* where the nature of the effusion can be determined (e.g. floccular in the case of joint sepsis).
2. *Synovial hypertrophy,* especially evident in joint sepsis (Fig. 3-13). Villonodular synovitis can be identified on the dorsal aspect of the metacarpophalangeal (fetlock) joint.
3. *Articular cartilage defects*—many of the joints of the limb have very thin cartilage and the resolution of ultrasound limits its usefulness to identify subtle cartilage lesions. However, the technology is advancing rapidly and better definition is being achieved. Younger animals have thicker cartilage and abnormalities such as osteochondrosis of the stifle are easily identified, with or without underlying subchondral bone defects.
4. *Chip fractures of the margins of the joints.*
5. *Soft tissue structures*
   a. *Collateral ligaments*—such as avulsion or desmitis. Many collateral ligaments ascribe a curved path around the joint, so that most of the ligament will be "off-incidence" and dark.
   b. *Menisci*—the menisci of the stifle are easily imaged longitudinally from the medial and lateral aspects of the femoropatellar joint and are fine-stippled triangular-shaped

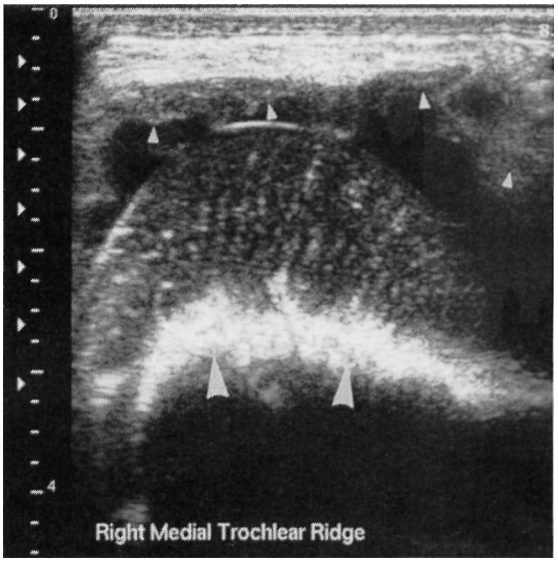


Figure 3-13. Transverse ultrasonograph obtained from over the medial trochlear ridge of a foal with chronic sepsis of the femoropatellar joint. Note the fluid distension of the joint and the thickened synovial membrane *(small arrowheads).* The medial trochlear ridge is cartilaginous at this age, so that the bone surface echo is a considerable distance from the cartilage surface *(large arrowheads).*

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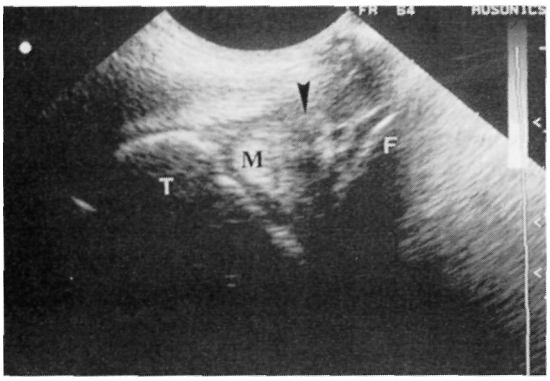


Figure 3-14. Longitudinal ultrasonograph from the medial aspect of the stifle showing a horizontal medial meniscal tear *(arrowhead).* T, tibial crest; F, medial femoral condyle; M, medial meniscus.

structures with their apex directed toward the center of the joint. Meniscal tears, most frequently affecting the medial meniscus, can be identified in areas of the meniscus not visible arthroscopically, allowing better assessment of the extent of the tear (Fig. 3-14).

c. *Intra-articular ligaments* (e.g. cruciate ligaments of the stifle)—these ligaments can be identified if the limb is maintained in a flexed position and 3 to 5 MHz sector or curvilinear transducers applied to the cranial and caudal aspects of the stifle. The transducer is directed caudodistally toward the tibial plateau for the cranial cruciate or craniodistally from the popliteal region for the caudal cruciate.

BONE

Ultrasound has been considered only able to image soft tissue structures because of its failure to penetrate bone sufficiently. However, many orthopedic injuries in the horse involve the surface of the bone, which can easily be assessed ultrasonographically. The following bony lesions can be imaged with ultrasound:

1. *Enthesiophytosis* at the insertion of tendons, ligaments, and fibrous joint capsules.

2. Fractures—small chip fractures are readily identified ultrasonographically where their size can be measured and their exact location marked on the skin preoperatively. In the upper limb and

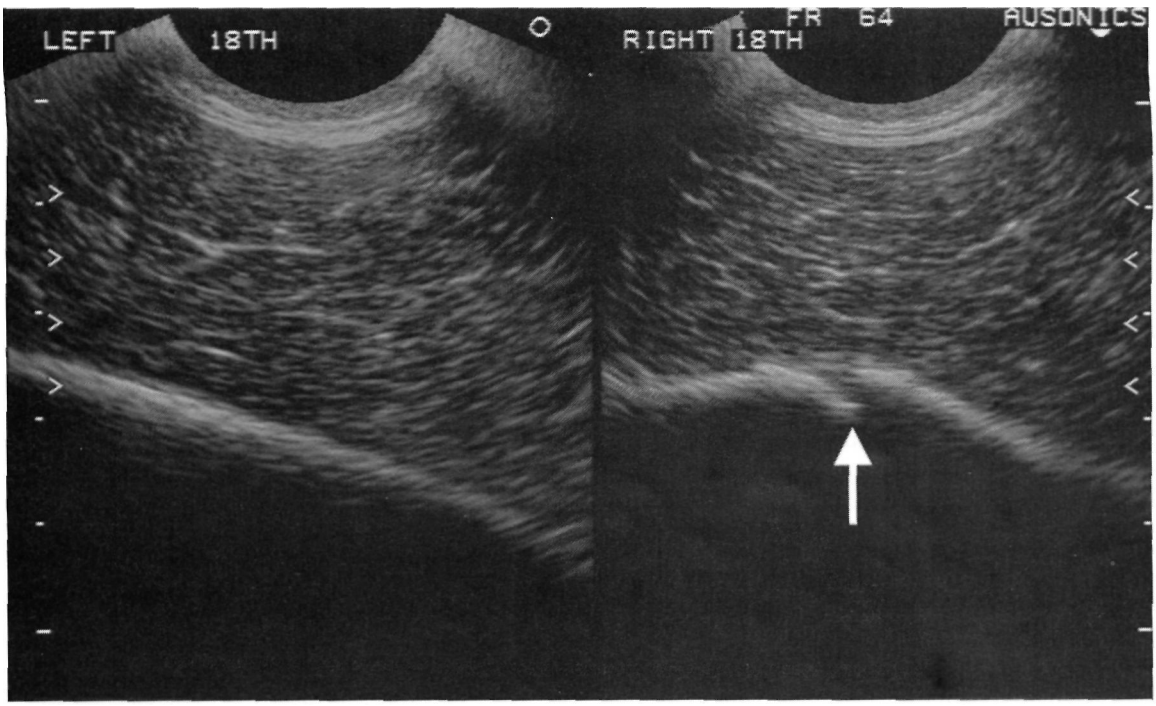


Figure 3-15. Longitudinal scans from the left and right 18th ribs, showing a rib fracture *(arrow).* The horse presented as suffering from back pain, had reduced back reflexes, and gamma scintigraphy revealed marked focal uptake over the proximal parts of the 17th and 18th ribs. Radiography was negative, but ultrasonography confirmed the presence of fractures in both ribs, illustrating the advantage of ultrasonography for the diagnosis of fractures in areas not particularly amenable to radiography.

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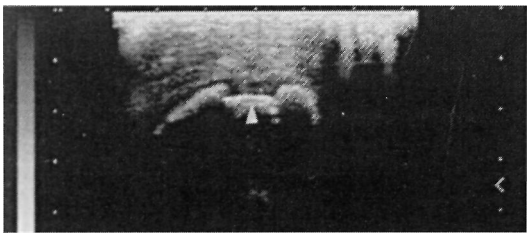


Figure 3-16. Transverse ultrasonograph obtained over a medial distal radial swelling. There is a sequestrum *(arrowhead)* lying in an involucrum, with anechogenic fluid immediately superficial to it.

body, where large body mass makes radiography of poor definition, ultrasonography is particularly useful, as in fractures of the wing of the ilium, ribs, deltoid tuberosity, and third trochanter (Fig. 3-15). However, in large or multiple fractures, multiple shadowing artifacts makes appreciation of the exact fracture configuration difficult.

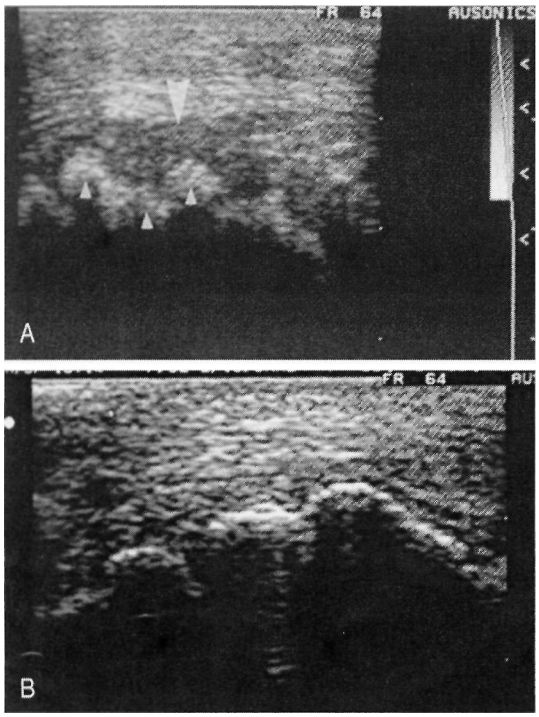


Figure 3-17. Transverse scans obtained over the distal radius in a horse that suffered a nondisplaced distal radial fracture. *A,* Scan obtained 2 months after the injury. There is a thickened, convoluted, bone surface echo *(small arrowheads)* and a hypoechogenic thickened periosteum *(large arrowhead)* indicative of immature callus. *B,* Scan obtained 1 month after *A.* The callus has matured and the bone surface echo has reduced in thickness towards normal.

3. Osteitis/osteomyelitis—the presence of anechogenic fluid adjacent to the surface of bone is diagnostic for septic osteitis (Fig. 3-16). There usually is also an irregular surface to the underlying bone. Sequestra can be identified lying in an incomplete involucrum (see Fig. 3-16) and their length recorded to determine if they are resorbing under conservative management.

4. Callus formation—the amount of callus and extent of bridging of the fracture gap can be identified ultrasonographically. Immature callus does not cause acoustic shadowing so that active callus on the surface of a bone shows a thickened bone surface echo (BSE). Once the callus has matured, the BSE returns to normal width (Fig. 3-17).

MUSCLE

Ultrasonography is particularly useful for the diagnosis of muscle abscessation and hematoma. Acute intramuscular hemorrhage is usually echogenic, so that the only apparent change is a loss of the normal coarse striated pattern of muscle. Once the hemorrhage organizes into a hematoma, anechoic areas are visible as a multiloculated structure with jelly-like consistency when balloted (Fig. 3-18). Subsequently, the hematoma will either resorb or become a seroma, characterized ultrasonographically as an anechoic area within the muscle, with or without loculations. A muscle abscess has a similar appearance except that it usually has an obvious echogenic fibrous capsule and floccular fluid. Most seromas and abscesses cause acoustic enhancement deep to them. If the abscess contains large numbers of white cells and debris, they settle in the most dependent part.

The use of ultrasonography for the diagnosis

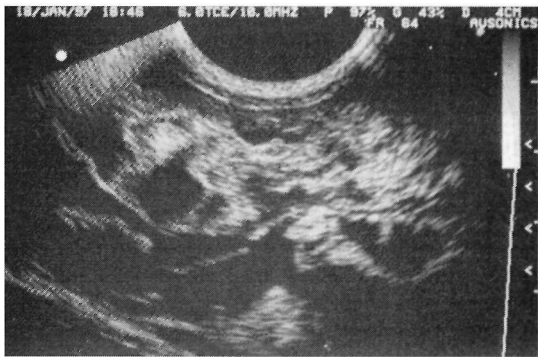


Figure 3-18. Ultrasonograph from a swelling on the lateral aspect of the thigh. The scan shows the classical appearance of an organizing hematoma. Ballottement with the probe would exhibit the jelly-like consistency of the swelling.

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of other muscle injuries in the horse is in its infancy, and its usefulness remains to be determined. In most cases, such as postoperative myopathy, muscle damage results in an increase in echogenicity of the muscle tissue.

WOUNDS

KEY POINT

Ultrasonography has particular benefits in the assessment of wounds and discharging tracts because many penetrating equine wounds involve soft tissues and also the surface of bone. However, it is not a substitute to radiography and both are used together to obtain a full evaluation.

The tract of the penetrating wound, as well as more established discharging tracts, can be imaged and followed to their limit. This can indicate the cause of the discharging tract (e.g., sequestration, foreign body) and demonstrate if any adjacent structures have been damaged (e.g., joints, underlying bone) (Fig. 3-19). Ultrasound is a sensitive detector of foreign bodies, including radiolucent foreign bodies such as wood, all of which cast acoustic shadows or reverberation artifact (Fig. 3-20). Although the acoustic shadowing is helpful in locating foreign bodies, it also obscures underlying structures and prevents the dimensions of the foreign body from being determined. Hence, the ultrasonographic examination should involve as many planes as possible around the foreign body. Air also casts an acoustic shadow with a variable amount of reverberation artifact. It too can obscure detail, and therefore, large gaping wounds with or without aspirated air under the skin are not good candidates for ultrasonographic examination. In these cases, digital palpation, contrast radiography, or surgical exploration are better alternatives.

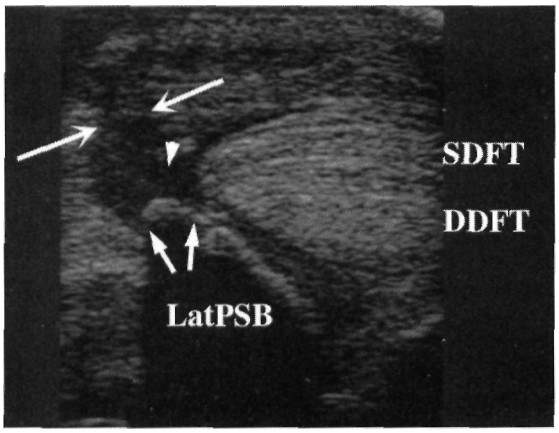


Figure 3-19. Transverse ultrasonograph from the palmar aspect of the fetlock region in a horse with a chronically discharging wound over the lateral proximal sesamoid bone. Ultrasonography clearly demonstrates the tract extending from the wound to the palmar border of the lateral proximal sesamoid bone *(between long arrows).* The tract appears to communicate with the digital sheath *(arrowhead)* and there are defects within the surface of the lateral proximal sesamoid bone *(connected arrows),* indicating septic osteitis. Note also the considerable subcutaneous thickening superficial to the flexor tendons. LatPSB, lateral proximal sesamoid bone; SDFT, superficial digital flexor tendon; DDFT, deep digital flexor tendon.

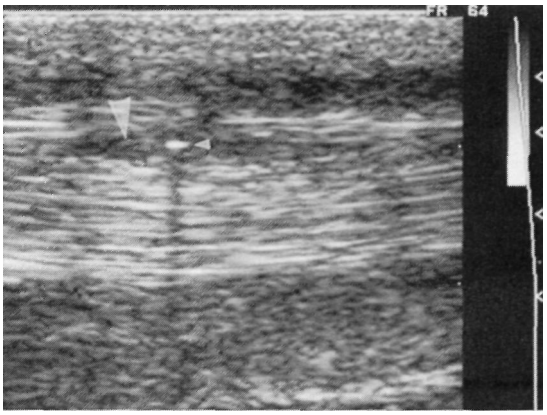


Figure 3-20. Longitudinal ultrasonograph over the superficial digital flexor tendon of the hindlimb following the removal of a "blackthorn" 2 weeks previously. The swelling around the small puncture wound had persisted, initiating an ultrasound examination of the area. The scan shows a small echogenic foreign body *(small arrowhead),* which casts a thin acoustic shadow, lying within a relatively hypoechoic cavity *(large arrowhead).* Immediately superficial to and to the right of (distal) the foreign body, a defect in the surface of the tendon can be identified. A 0.5 cm piece of the tip of a blackthorn was successfully removed at surgery.

Section 2. Echocardiography

Echocardiography has greatly enhanced the ability to assess the horse's cardiovascular system. The technique is indicated for investigation of horses with cardiac murmurs, cardiac arrhythmias, pulmonary or dependent edema, and in poor performance, when musculoskeletal and respiratory disease has been ruled out. A complete evaluation includes real-time B-mode imaging, and M-mode and Doppler echocardiography. *B-mode echocar-*

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diography is used to examine the internal anatomy of the pericardium, cardiac chambers, heart valves, and great vessels. *M-mode echocardiography* enables the precise assessment of cardiac dimensions and movement at specific points in the cardiac cycle, and *Doppler echocardiography* determines the velocity and direction of intracardiac blood (low.

KEY POINT

Two-dimensional real-time, M-mode, and Doppler echocardiography are used together to identify cardiac lesions and assess their severity.

TECHNIQUE AND EQUIPMENT

Imaging of the adult horse's heart requires a depth of penetration of approximately 30 cm; therefore, transducers frequencies of 2 to 3.5 MHz are used. In foals and small ponies, higher frequencies can be suitable. Phased array or mechanical sector transducers provide a wide field of view from a small contact point. There should be a simultaneous electrocardiograph recorded during the echocardiographic examination. Suitable equipment has measurement packages for M-mode and Doppler echocardiography.

A series of standardized images should be obtained in every case, and each structure should be examined in at least two perpendicular planes (Table 3-4; see Chapter 6).

KEY POINT

A standardized approach should be adopted in every case to avoid omissions.

TABLE 3-4. Normal Two-Dimensional and M-Mode Echocardiography Values for Adult Horses

	Range (mean)
Right ventricular internal dimensions: diastole	32-35 (38.6) mm
Ventricular septal thickness: diastole	19-43 (30.6) mm
Ventricular septal thickness: systole	35-62 (48.1) mm
Left ventricular internal dimension: diastole	80-130 (110.6) mm
Left ventricular internal dimension: systole	43-79 (61.1) mm
Left ventricular free wall thickness: diastole	20-40 (29.2) mm
Left ventricular free wall thickness: systole	32-56 (44.5) mm
Left ventricular fractional shortening	32-55 (44.1) %*
Septal-left atrioventricular valve E point separation	<10 mm
Left auricular dimension	35-75 (56.4) mm
Aortic root dimension	55-92 (73.1) mm
Left ventricular ejection time	<045 s
Left atrial diameter (two-dimensional)	<13.5 mm
Pulmonic artery diameter: aortic root diameter (two-dimensional)	<1

*The left ventricular fractionlal shortening is normally less than 40% at heart rates of less than 50 bpm.

Data from Bonagura et al. 1985, Lescure and Tamzali 1984, and Reef 1990.

CARDIAC MURMURS

As many as 80% of normal horses have cardiac murmurs, and these are particularly common in athletic breeds. Most of these are physiologic and are not associated with cardiac pathology. In some horses, the murmurs are due to valvular regurgitation, but this is often mild and of no clinical significance. Color flow Doppler echocardiographic studies have revealed that small jets of valvular regurgitation can be detected in normal horses with no cardiac murmurs, and therefore mild degrees of valvular regurgitation are considered to be a physiologic rather than pathologic phenomena. However, in some horses, a cardiac murmur can be indicative of significant cardiac pathology, leading to athletic impairment or even severe cardiac dysfunction.

The most common pathologic conditions associated with cardiac murmurs are valvular insufficiency and ventricular septal defects. Other congenital cardiac defects, such as patent ductus arteriosus and the complex defects, are less common and usually become clinically apparent early in life. In horses in which cardiac pathology is suspected, echocardiography is used to determine the precise nature of any changes in valvular structure and to visualize ventricular septal defects or other congenital anomalies. Echocardiography also is useful to document and semiquantify regurgitant blood flow and intracardiac shunts and to detect dilation of cardiac chambers and changes in cardiac function that reflect the severity of the underlying cardiac disease. By considering all of

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this information, an assessment of the hemodynamic effects of any lesions can be made and a suitable management plan and prognosis can be formulated. Repeated examinations allow the clinician to document the rate of progression of lesions more accurately than an assessment made based oh one examination.

Insufficiency of the Semilunar Valves

The pulmonic valve is a rare site for cardiac pathology, whereas the aortic valve is the most common site for degenerative valvular lesions in the equine heart. The murmur of aortic insufficiency is pan, holo-, or early diastolic with its point of maximal intensity in the left or right fourth intercostal space. The murmur can be decrescendo and coarse or occasionally have a musical or creaking quality. Most affected horses are middle aged or older. As left ventricular volume overload develops, the arterial pulses become hyperkinetic with a strong systolic component and rapid diastolic drop-off. Degenerative aortic insufficiency is usually a slowly progressive lesion, and signs of severe cardiac disease do not occur until the left ventricle dilates to the point that the left atrioventricular valvular annulus dilates and causes left atrioventricular insufficiency. Lesions that are associated with more rapid progression to severe aortic insufficiency include bacterial endocarditis and rupture of one of the valve cusps.

KEY POINT

Aortic insufficiency is common, and when associated with degenerative lesions, it is usually well tolerated in middle-aged and older horses.

Degenerative lesions typically appear as echogenic nodules, often most obvious on the left coronary cusp (Fig. 3-21). Bacterial endocarditis causes larger more irregular nodules. Left ventricular volume overload leads to dilation of the chamber and rounding of the left ventricular apex (Fig. 3-22). The movement of the ventricular septum becomes more vigorous, which is evident subjectively on B-mode echocardiograms and can be documented more precisely using M-mode echocardiography. The left ventricular lumen diameter becomes progressively larger. In degenerative aortic valvular disease, the regurgitation is often well tolerated and the end diastolic left ventricular lumen diameter increases gradually over a period of years. Rapid enlargement of the left ventricle indicates progressive deterioration. With more aggressive forms of aortic valvular pathology, severe regurgitation can develop before the left ventricle dilates markedly.

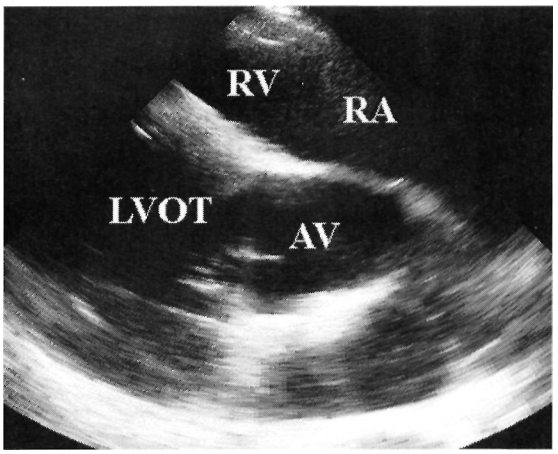


Figure 3-21. A long-axis echocardiogram from a 12-year-old Thoroughbred gelding with aortic insufficiency with marked nodular thickening of the aortic valve (AV). LVOT, left ventricular outflow tract; RV, right ventricle; RA, right atrium.

The mitral valve should be assessed carefully in horses with aortic insufficiency, because concurrent mitral insufficiency can lead to heart failure. Turbulence in the left ventricular outflow tract causes diastolic vibrations of the aortic valve, septum, and left atrioventricular valve, which are most readily detected in M-mode images (Fig. 3-23). Jets of aortic insufficiency, which occupy the area immediately adjacent to the valve, are considered mild, whereas jets occupying one- to

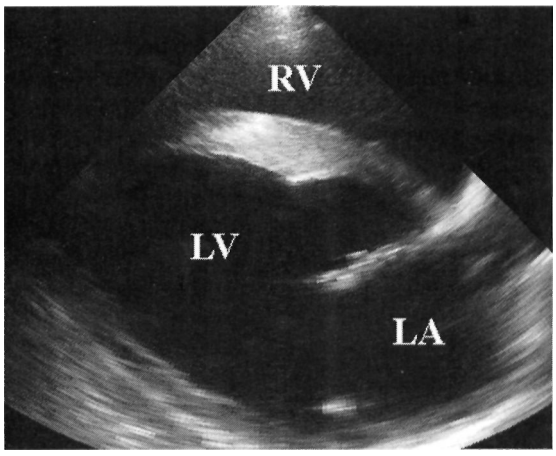


Figure 3-22. A long axis echocardiogram of the left (LV) and right (RV) ventricles from a 12-year-old Thoroughbred with aortic insufficiency. The LV is rounded at the apex, indicating that there is left ventricular volume overload. LA, left atrium.

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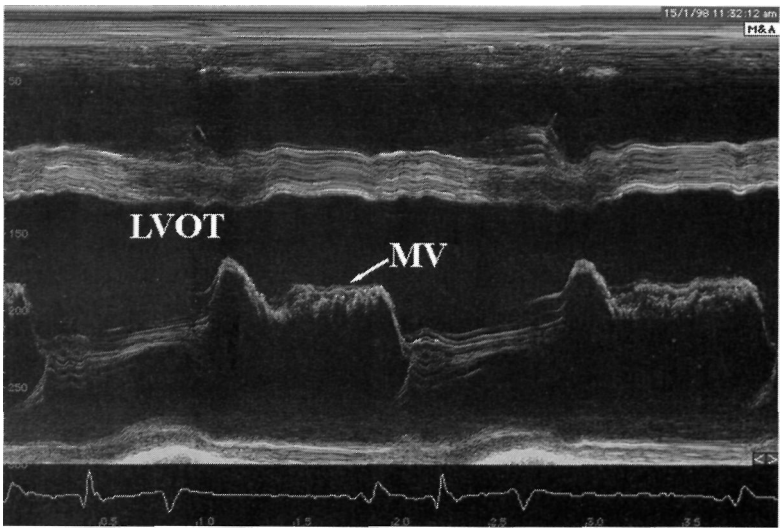


Figure 3-23. An M-mode echocardiogram at the level of the mitral valve from a 12-year-old Thoroughbred gelding with aortic insufficiency. The mitral valve (MV) is vibrating during diastole as it comes into contact with regurgitant blood flow in the left ventricular outflow tract (LVOT).

two-thirds of the left ventricular outflow tract are moderate. Larger jets are considered severe.

KEY POINT
The severity and importance of aortic insufficiency is determined by the nature of valvular changes, the area occupied by the regurgitant jets, and the degree of left ventricular dilation.

Insufficiency of the Atrioventricular Valves

Right atrioventricular (tricuspid) insufficiency is a common cause of murmurs in equine athletes and is rarely clinically significant in this type of horse. In less athletic breeds, it is less common and more likely to indicate cardiac pathology. *Left atrioventricular (mitral) insufficiency* can be associated with exercise intolerance, predispose the horse to the development of arrhythmias such as atrial fibrillation, or lead to heart failure if severe. Lesions of the atrioventricular valve include degenerative changes, inflammation, bacterial endocarditis, and rupture of the chordae tendineae. Rapidly progressive conditions carry a poor prognosis.

Typically, atrioventricular insufficiency leads to a pan or holosystolic murmur with the point of maximal intensity over the right fourth (tricuspid) or left fifth (mitral) intercostal spaces. Mitral insufficiency frequently causes harsh, band-shaped murmurs that may radiate dorsally or caudodorsally, whereas the murmur of tricuspid insufficiency is usually less harsh and more localized. The clinical significance of atrioventricular insufficiency is determined by the specific site, the severity of associated regurgitation, and the rate of progression. Echocardiography can assist the clinician in evaluating all of these factors.

KEY POINT
Tricuspid insufficiency is frequently well tolerated in horses, whereas mitral insufficiency is more likely to be clinically significant.

Echocardiographic findings in artrioventricular valvular insufficiency include nodular or diffuse thickening of the valve cusps, indicating degenerative or inflammatory changes. Rupture of a chorda tendinea allows portions of the valve to flail upward into the atria. Mild to moderate tricuspid insufficiency in the absence of obvious structural changes in the valve may have no clinical consequence. Likewise, prolapse of either of the atrioventricular valves with minimal or mild regurgitation rarely progresses or causes clinical signs.

Doppler echocardiography is used to determine the area occupied by the regurgitant jet as a semiquantitative guide to its severity. Tricuspid jets can usually be readily detected, but, unfortunately, jets of mitral regurgitation often run perpendicular to the available imaging planes in the horse, which can limit accurate assessment of the area occupied.

Ventricular volume overload occurs with more severe lesions and provides a useful quantitative guide to the severity of regurgitation. The diameter of the left ventricle can be measured accurately with M-mode echocardiography, but, because of the crescent shape of the right ventricle, it is difficult to measure this ventricle in a repeatable manner with M-mode echocardiography. Severe

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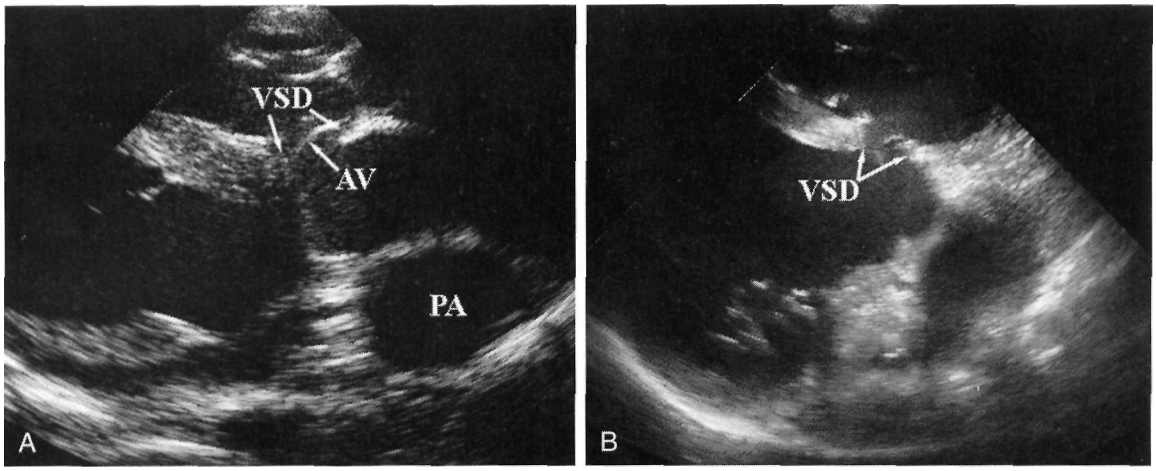


Figure 3-24. Long- *(A)* and short- *(B)* axis echocardiograms of the left ventricular outflow tract (LVOT) in a 2-year-old Welsh mountain pony with a large ventricular septal defect (VSD). The defect is located in the membranous portion of the septum and a portion of the aortic valve (AV) is disrupted. The pulmonary artery (PA) is dilated secondary to pulmonary hypertension.

right ventricular volume overload can be detected by subjective assessment of the size and shape of the right ventricle in two-dimensional images. Normally, the right ventricle is approximately half the size of the left ventricle. Rounding of the apex of the right ventricle occurs with severe dilation. Likewise, it is difficult to measure the right atrium, and it is assessed subjectively by comparing its size with the left atrium. The diameter of the left atrium can be determined from left parasternal long-axis images, and this is a useful guide to the severity of mitral insufficiency. In severe mitral insufficiency, the pulmonary artery can filiate. This is a very poor prognostic indicator because it may indicate impending pulmonary artery rupture.

KEY POINT

Severe tricuspid regurgitation is associated with large regurgitant jets, structural changes of the valves, and dilation of the right atrium and ventricle. Severe mitral regurgitation is associated with large regurgitant jets, structural changes of the valves, and dilation of the left atrium and ventricle and pulmonary artery.

Ventricular Septal Defects

The ventricular septal defect (VSD) is the most common form of congenital heart disease in large animals. The defect is usually located in the membranous portion of the septum beneath the aortic valve, but any portion of the septum can be involved (Fig. 3-24). The VSD causes a coarse pansystolic murmur, loudest over the right hemithorax. There is frequently an additional holosystolic crescendo-decrescendo murmur, loudest over the pulmonic valve, in the left third intercostal space, caused by the left to right shunt and relative pulmonic stenosis. Because of the location of the defect, beneath the aortic annulus, the aortic valve can become incompetent, leading to a third murmur in diastole (Fig. 3-24).

The size of the defect determines the magnitude of the intracardiac shunt, the severity of clinical signs, and the ultimate prognosis. Defects of less than 2.5 cm in Thoroughbreds and Standardbreds, or less than one-third of the aortic diameter in other breeds, are restrictive with mild degrees of shunting. These horses can have useful careers in less demanding sports. Larger defects lead to ventricular volume and pressure overload, cardiac compromise, and heart failure. Small defects are associated with high velocity shunts because a normal or near normal pressure difference between the two ventricles exists. Velocities of greater than 4.5 m/s indicate a restrictive lesion. Echocardiography is used to identify and measure the VSD in two perpendicular planes, to measure the intracardiac shunt velocity, to document left ventricular volume overload, and to investigate concurrent valvular lesions.

CARDIAC ARRHYTHMIAS AND MYOCARDIAL DISEASES

Primary causes of equine myocardial diseases include toxic, viral, bacterial, neoplastic, nutritional,

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and immune-mediated processes. Occasionally, dissecting hematomas disrupt the ventricular conduction system after the formation of aortocardiac fistula, causing sudden death or signs of distress and sustained ventricular tachycardia. Myocardial dysfunction can also arise secondary to processes such as metabolic disturbances, hypoxia, or endotoxemia. In the latter situation, echocardiography is rarely required. However, in horses suspected of having primary myocardial disease, echocardiography can be used to detect localized lesions and assess global myocardial function. However, it is important to recognize that a normal echocardiogram does not rule out the existence of focal or microscopic myocardial pathology.

In cardiomyopathy, ventricular dilation and hypomotility of the ventricular septum and ventricular walls can be detected subjectively on two-dimensional echocardiograms and more objectively with M-mode echocardiography (Fig. 3-25). Fractional shortening (left ventricular lumen in diastole — left ventricular lumen in systole ÷ left ventricular lumen in diastole) is reduced in the hypomotile ventricle. Increased pre-ejection period, decreased left ventricular ejection time, increased mitral E point-septal separation, and reduction in the systolic movement of the aortic root are also signs of compromised ventricular function. Occasionally, hyperechoic nodules are visible within the myocardium, but this is not a consistent finding in myocardial disease and these may be incidental findings because they can be observed in horses with no history of cardiac arrhythmias or myocardial dysfunction.

Postexercise echocardiography can be used in conjunction with maximal exercise testing on high-speed treadmills as part of comprehensive diagnostic evaluations of poor performance. In normal horses, left ventricular dimensions are minimally affected by exercise. Reductions in fractional shortening by greater than *5%* after exercise are considered to be abnormal.

In horses with atrial fibrillation, echocardiography is invaluable in identifying individuals with significant underlying cardiac pathology. These are less likely to convert to sinus rhythm after treatment with quinidine sulfate and have a higher incidence of adverse signs during treatment and a higher likelihood of recurrence of the arrhythmia. Slight reductions in fractional shortening are frequently observed in atrial fibrillation. This reflects reduction in preload and will resolve when the rhythm returns to normal. It does not indicate underlying myocardial dysfunction.

KEY POINT

Echocardiography is used to detect ventricular dilation, reduced fractional shortening, and occasionally focal myocardial lesions in some horses with myocardial disease.

THE PERICARDIUM

Pericarditis and pericardial effusions are uncommon in horses. Thoracic trauma can lead to hemorrhage into the pericardium. Pericarditis is usually effusive and fibrinous and occurs sporadically in

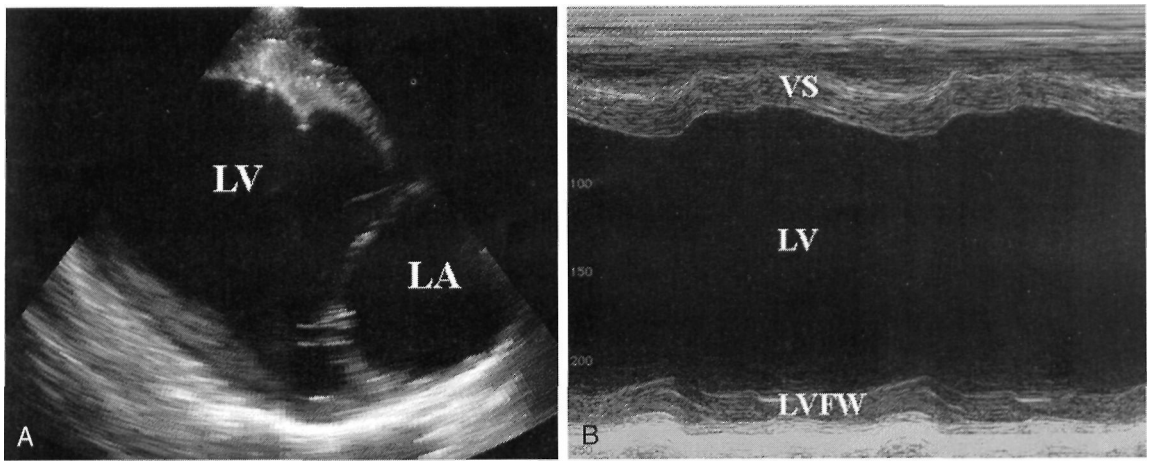


Figure 3-25. Long-axis *(A)* and M-mode *(B)* echocardiograms of the ventricles from a 8-year-old Arabian gelding with dilated cardiomyopathy. The left ventricle (LV) is markedly dilated and hypomotile, with very little movement of the ventricular septum (VS) and left ventricular free wall (LVFW) visible on the M-mode echocardiogram. LA, left atrium.

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all types of horses. It is caused by bacterial or viral infection or can be idiopathic in nature. Pericardial tamponade reduces ventricular filling and leads to signs of systemic venous distension, marked dependent edema, and accumulation or effusion in the thoracic and abdominal cavities. Reduction in cardiac output causes signs of low output failure such as pallor, weakness, and reduced urinary output. The pericardial effusion is readily apparent on two-dimensional and M-mode echocardiograms, and the dimensions of the cardiac chambers are reduced. Collapse of the right atrium is one of the earliest signs of cardiac tamponade, and, if present, pericardiocentesis is indicated. In most cases of pericarditis, large amounts of fibrinous debris are adherent to both the parietal and visceral surfaces of the pericardium. Extension of the inflammation into the epi- and myocardium can cause focal lesions within the myocardium. In acute cases, the prognosis is fairly good, provided aggressive therapy, including pericardial lavage, is undertaken. In chronic cases, the pericarditis can become constrictive and the prognosis deteriorates.

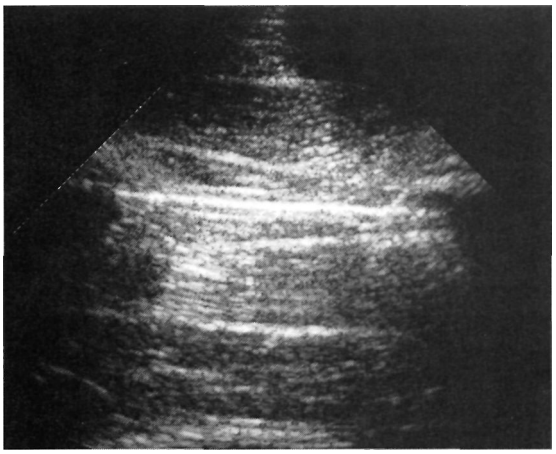


Figure 3-26. An ultrasonogram showing the normal surface of the lung produces a straight echogenic line with reverberation artifacts deep to the surface echo.

Section 3. Thoracic Ultrasonography

The contents of the mediastinum and pleural cavity are readily examined with ultrasonography. The large difference in tissue density that arises at the interface between air in the lung and the pleural cavity produces a strong echo at the surface of the lung with reverberation artifacts deep to the surface echo (Fig. 3-26). Therefore, it is impossible for ultrasound to provide images of normal aerated lung. However, ultrasound can penetrate areas of consolidation or abscessation, providing the lesions extend to the periphery of the lung.

TECHNIQUE AND EQUIPMENT

Transducers of frequencies ranging from 4 to 7.5 MHz are most suitable for examining the thorax, although in the presence of large pleural effusions and extensive pulmonary pathology, when more depth of penetration is required, lower frequencies can be used. Sector or microconvex transducers are the most convenient, because the intercostal spaces provide a narrow point of contact for the ultrasound beam. Limited images of the thorax can be obtained using linear transducers. To examine the lungs and pleural cavity, the transducer is placed between the ribs with the beam oriented in a dorsal to ventral plane. The cranial mediastinum can be examined from the right third intercostal space, which usually requires the horse to stand with the right forelimb drawn forward. The cranial mediastinum can also be examined by imaging caudally and ventrally from the thoracic inlet.

Comparison of Thoracic Ultrasonography and Radiography

Diagnostic ultrasonography can be regarded as complementary to conventional radiography for imaging the thorax. Ultrasonography is noninvasive to both patient and veterinary personnel. It is less likely to require sedation of the patient and the equipment is more portable, and frequently less expensive, than radiographic units that are capable of obtaining thoracic radiographs of diagnostic quality. Ultrasonography is superior to radiography for the identification and characterization of pleural effusions. Both radiography and ultrasonography can readily identify large areas of consolidation and abscessation. Ultrasonography may be more sensitive for smaller peripheral areas of consolidation, particularly in the ventral lung lobes. However, ultrasonography will not demonstrate lesions that do not extend to the periphery of the lung and it is not particularly helpful in lower airway disease, where cytologic examination of tracheal aspirates or bronchoalveolar lavage is of more diagnostic value. The real-time

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nature of ultrasonographs allows guided biopsy or aspirates of lung lesions.

KEY POINT

Ultrasonography is superior to radiography for evaluation of the pleural space and the identification of small peripheral lung lesions.

THE PLEURA

In the normal horse, a very small amount of fluid is visible in the ventral portions of the pleural cavity. Causes of pleural effusion include pleurisy/pleuropneumonia, neoplasia (e.g., cranial mediastinal lymphosarcoma, metastatic squamous cell carcinoma), and right-sided heart failure. Fluid will settle in the ventral thorax, and its quantity can be documented by measurement of the fluid level with respect to the point of the shoulder (Fig. 3-27). This will allow changes in fluid volume to be documented more precisely by sequential examinations. The echogenicity of pleural effusions is variable. Transudates are usually anechoic, but exudates, with a high cellular content, can be echogenic. Fibrinous debris often is present in pleuropneumonia. Gas bubbles free in the fluid or adherent to fibrinous material should alert the clinician to the possibility of anaerobic infection. Cytologic examination is necessary to confirm the precise nature of a pleural effusion. Ultrasonography can be helpful in selecting the best site for pleurocentesis.



Figure 3-27. A thoracic ultrasonogram from the sixth intercostal space at the level 6 cm dorsal to the point of the shoulder from a 13-year-old Thoroughbred stallion with pleuropneumonia. Hypoechoic fluid is free within the pleural space and the ventral portions of the lung are consolidated, allowing sound to penetrate.

Pleural abscesses are usually a consequence of chronic pleuropneumonia. Ultrasonography is used to identify that a pleural abscess is walled off from the remainder of the thorax before thoracotomy to drain the abscess is considered. Pneumothorax is characterized by an air interface with a sharp differentiation from the underlying lung and the free air will not move with respiration.

KEY POINT

Ultrasonography is used to identify, characterize, quantify, and aspirate pleural effusions.

THE CRANIAL MEDIASTINUM

In cases of cranial mediastinal lymphosarcoma, there is usually a large lobular or multilobular mass that may exit the thoracic inlet and typically is associated with moderately large volumes of pleural effusion. Occasionally, it is possible to obtain a fine-needle aspirate of the mass via the thoracic inlet with ultrasound guidance. Abscesses and lymphadenopathy in the cranial mediastinum may be multilocular or have a solid appearance.

THE LUNGS

KEY POINT

Ultrasonography is used to identify lesions that extend to the periphery of the lung.

The surface of the lung is normally smooth and unbroken and glides freely over the diaphragm and parietal pleural suface. The deeper portions of the lung are obscured by reverberation artifact (see Fig. 3-26). Diseased portions of lung that are not aerated allow the ultrasound to penetrate and therefore have an ultrasonographic appearance similar to other solid soft tissues. Abnormalities that can be identified ultrasonographically include atelectasis, consolidation, hepatization, abscessation, and nodular and diffuse lesions. Atelectasis normally involves the ventral portions of the lungs that are nonaerated and collapsed into thin compressed slivers. Consolidated lung is also nonaerated, but because of infiltration by inflammatory cells, the affected portion of lung maintains its wedge shape (see Fig. 3-27). Hepatized lung represents a more advanced inflammatory and necrotic lesion; the lung is nonaerated and fluid within the airways produces an appearance that is similar to that of liver. Pulmonary abscesses are

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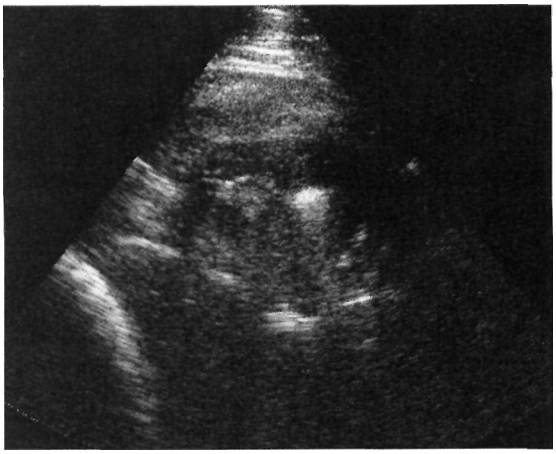


Figure 3-28. A thoracic ultrasonogram from a 13-year-old Thoroughbred stallion with chronic pleuropneumonia. An encapsulated pulmonary abscess has formed with free gas within it.

walled off from surrounding lung. The abscess cavity can have variable echogenicity, depending on the precise nature of its contents (Fig. 3-28). Gas within the cavity causes hyperechoic areas with acoustic shadowing. Multiple abscesses are observed in *R. equi* pneumonia.

Pulmonary nodules can be associated with primary or metastatic neoplasia or granulomatous infiltration (Fig. 3-29). Lung biopsy is the preferred method of diagnosis. Diffuse lesions are nonspecific, and differential diagnoses include pneumonia, tuberculosis, pulmonary edema, and diffuse interstitial fibrosis. Mild disruption of the periphery of the lung field is also a nonspecific change and can be observed in a wide variety of lung diseases, from chronic obstructive pulmonary disease to early pneumonic lesions or scarring associated with previous pneumonia.

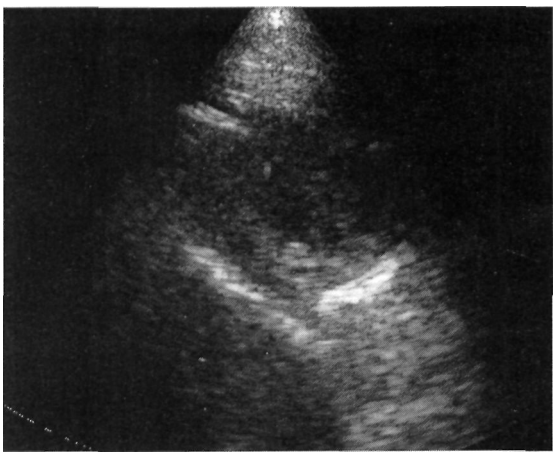


Figure 3-29. A thoracic ultrasonogram from a 14-year-old cob with pulmonary granular cell tumor. There is a discrete mass within the lung with fairly uniform echogenicity.

Section 4. Abdominal Ultrasonography

Ultrasonography is the principal imaging technique for visualization of abdominal organs. Its use in hepatic, renal, and splenic disease is now well-established. Increasingly, its value in a variety of equine intestinal disorders is recognized. Sector transducers are necessary for examination of most of the abdomen. In the adult horse, frequencies of 3.5 to 5 MHz are used. Ideally, the highest frequency that provides adequate penetration should be used to optimize image quality.

HEPATIC ULTRASONOGRAPHY: TECHNIQUE, EQUIPMENT, AND NORMAL ANATOMY

In most horses, the liver can be examined with a 4- to 5-MHz transducer. For larger horses, 3.5 MHz is used, whereas in foals and small ponies, 6.5 to 7.5 MHz may provide adequate penetration and superior image quality. On the right side, the liver can be identified ventral to the lung margins from the 14th to the 7th intercostal space, whereas on the left side, the liver is visible ventral to the lung margins and lateral to the spleen in the left 5th to 8th intercostal space.

The liver has a uniform echogenicity that is greater than that of the kidney and less than that of the spleen (Fig. 3-30). Hepatic and portal vessels course through the liver. The edges of the liver are sharp and pointed.

Diffuse Hepatic Diseases

It is not possible to visualize the entire liver in the horse nor to accurately measure its size. However, hepatic enlargement (e.g., in hepatitis, cholangiohepatitis, hyperlipemia) leads to extension beyond the normal boundaries, whereas if the liver is decreased in size (e.g., in ragwort toxicity, hepatic fibrosis), it may not be! visible in all the normal imaging field. In conditions associated with enlargement or decrease in liver size, the edges of the liver can become rounded. Hepatitis and hyp-

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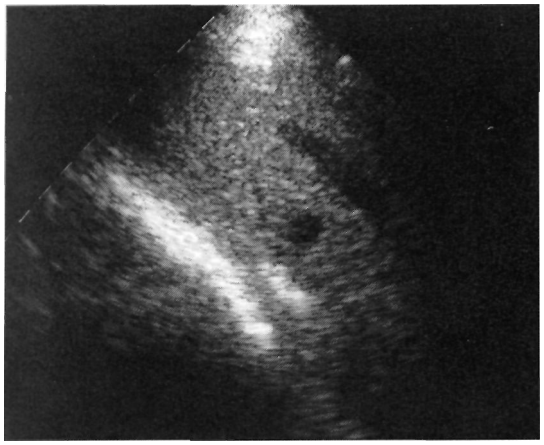


Figure 3-30. Normal liver imaged from the right 14th intercostal space. Hepatic vessels are visible and the liver has a uniform echogenicity.

erlipemia usually decrease the echogenicity of the liver, whereas ragwort toxicity can produce subtle increases in echogenicity. However, changes in echogenicity are not always apparent in diffuse hepatic disease. Liver biopsy is required to definitively diagnose the precise nature of hepatic pathology in diffuse conditions. Ultrasonography can be helpful in identifying suitable sites for biopsy.

KEY POINT

In diffuse liver disease, ultrasonography is most useful for assessing liver size and facilitating hepatic biopsy.

Localized Lesions of the Liver

Choleliths are the most common form of localized hepatic lesion in the horse. Choleliths are echogenic with strong acoustic shadowing, and multiple choleliths usually are present. With biliary obstruction, dilated bile ducts run parallel to hepatic vessels. Choleliths are often associated with cholangiohepatitis, and therefore biopsy is indicated to determine the extent of underlying pathology-

Hepatic abscesses are variable in echogenicity, depending on the nature of their contents. Gas within the abscess causes hyperechoic areas. *Primary hepatic tumors* are uncommon, but lesions such as bile duct carcinoma occasionally are identified ultrasonographically as localized circumscribed echogenic areas.

SPLENIC ULTRASONOGRAPHY

The equine spleen is very variable in size. The spleen occupies most of the left side of the abdomen, lying immediately medial to the body wall. It is examined in the paralumbar fossa, ventral to the lung in the 8th to 17th intercostal spaces, and medial to the liver in the 6th and 7th intercostal space. It can extend to the right side in the craniоventral abdomen, and occasionally it lies lateral and cranial to the left liver lobe in the cranial abdomen. Depending on the size of the horse, sector or microconvex transducers with frequencies of 3.5 to 5 MHz are ideal for examination of the spleen.

The spleen has a very uniform appearance with an echogenicity that is greater than that of the liver (Fig. 3-31). Blood vessels within the spleen are visible. Ultrasonography can be used to identify hematomas, abscesses, and splenic lymphosarcoma.

URINARY TRACT: TECHNIQUE AND NORMAL FINDINGS

The right kidney is examined transcutaneously from the right 14th to 17th intercostal space. It lies immediately beneath the body wall caudal and ventral to the caudal portions of the liver. In most horses it can be examined with a sector transducer of 4 to 5 MHz. In larger or fat horses, lower frequencies may be required. The left kidney is examined in the 16th and 17th intercostal spaces and paralumbar fossa (see Fig. 3-31). The left

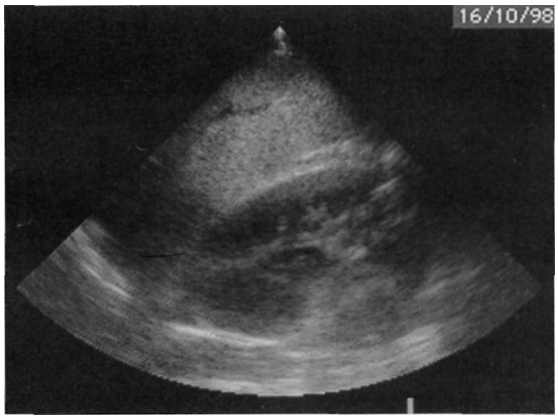


Figure 3-31. An ultrasonogram from the left paralumbar fossa in a normal horse showing the spleen and left kidney. The spleen has a uniform echogenicity and is more echogenic than the kidney.

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kidney is more variable in position than the right. It lies deep to the spleen and therefore transducers with frequencies of 3.5 MHz or lower are routinely used. The left kidney can also be examined per rectum using a sector transducer placed directly on the caudal pole.

The kidney is always less echogenic than the spleen and is usually less echogenic than the liver. In normal adult horses, the kidneys are 15 to 17 cm in length with a smooth outline. The cortex and medulla are visible with a distinct corticomedullary junction (Fig. 3-32). The cortex is slightly more echogenic than the medulla. Arcuate vessels at the corticomedullary junction cause pinpoint echogenic foci with reverberation artifacts. The fat in the renal pelvis is echogenic and often causes acoustic shadowing, which can be confused with mineralization or renal calculi.

In the adult horse, the bladder is examined per rectum using either linear or sector transducers of around 5 MHz. The foal's bladder is examined transcutaneously by placing a sector or linear transducer on the caudoventral midline of the abdomen. Urine is anechoic in foals but in adults is echogenic because of the high mucous and crystalline content. The urine may separate into two distinct layers or swirls of echogenic material may be visible, mixing with anechoic fluid.

Renal Diseases

Acute renal failure is most commonly caused by toxic or vasomotor tubular disease in horses. Ultrasonographic changes suggestive of acute renal disease include increases in kidney size, decreases in echogenicity, and perirenal edema (Fig. 3-33). However, ultrasonographic changes are not always present, and a normal ultrasonographic appearance does not rule out the existence of renal pathology. Renal enlargement is the most consistent feature of acute renal disease. *Chronic renal failure* can be associated with interstitial nephritis, renal fibrosis, glomerular nephritis, or neoplasia. Typically with nephritis or fibrosis, the kidneys are small with a nodular outline. The echogenicity either is increased or unchanged. Loss of the distinct corticomedullary junction can occur in both acute and chronic renal diseases (see Fig. 3-33). *Renal tumors* are associated with localized masses, usually of mixed or increased echogenicity. *Renal calculi* are echogenic with strong acoustic shadows. Renal calculi are often associated with glomerulonephritis or other renal pathology. In horses with clinical and clinicopathologic signs of chronic renal disease, renal biopsy can be helpful in establishing the prognosis. The right kidney can be biopsied from the 17th intercostal space. Ultrasonographic guidance reduces the risk of laceration of blood vessels. The left kidney is more difficult to biopsy, but it may be possible to position it against the body wall by rectal palpation.



Figure 3-32. An ultrasonogram showing part of the right kidney in a normal horse. The cortex is slightly more echogenic than the medulla and there is a distinct corticomedullary junction. The renal pelvic fat is extremely echogenic and renal vessels are visible.

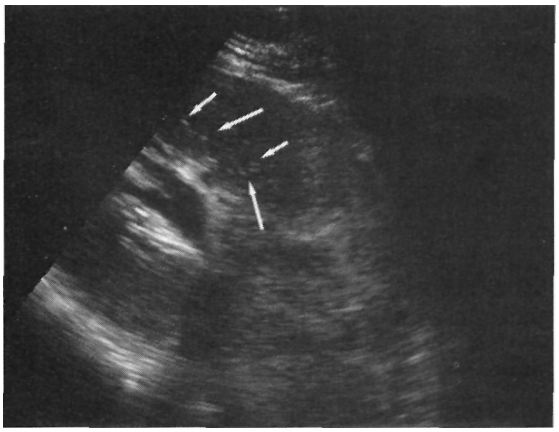


Figure 3-33. An ultrasonogram of the right kidney from a horse with acute aminoglycoside toxicity. The kidney is enlarged with reduced echogenicity and loss of the normal distinct corticomedullary junction. Pinpoint echogenic foci are visible within the renal parenchyma *(arrows).*

Diseases of the Lower Urinary Tract

The most common disease of the bladder in the adult horse is cystic calculi. Typically, a large single calculi or a sludge of sabulous material is

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present. Single calculi are extremely echogenic with strong acoustic shadowing and a roughened irregular surface.

Disruption of the lower urinary tract is rare in adults but can occur in parturient mares or after obstruction of the urethra, for example, with stricture formation. Lower urinary tract disorders are more common in foals. As a primary condition, bladder rupture occurs during birth, usually in colts. The clinical signs of progressive depression, lethargy, and abdominal distension begin to appear within 48 to 72 hours of birth. Disruption of the urachus, bladder, or, rarely, one of the ureters can occur secondary to infection in young foals within the first few weeks of life. These foals often have pre-existing clinical signs associated with umbilical infection or septicemia. In uroperitoneum, ultrasonography reveals copious quantities of free fluid within the peritoneal cavity. Usually, this is anechoic but can be echogenic if there is severe peritonitis. The bladder will often contain some urine. With bladder rupture, the bladder is small, flaccid, and irregular in shape, and occasionally a rent will be visible in the cranial or craniodorsal aspect. If the urachus or ureter is ruptured, the bladder will be small but will maintain its spherical shape. Sites of sepsis or necrosis may be visible as localized thickened areas of the urachus or bladder.

UMBILICAL ULTRASONOGRAPHY: TECHNIQUE AND NORMAL FINDINGS

Ultrasonography is the ideal means of assessment of the internal umbilical structures in foals. The structures are examined from the ventral abdomen, placing the transducer transversely, using 7.5- to 10-MHz linear or sector transducers. The umbilical vein lies immediately dorsal to the linea alba, running cranially from the external remnant to the liver and measures 0.3 to 1.1 cm (mean, 0.6 cm). The umbilical arteries run caudally from the external remnant to the cranial pole of the bladder where they separate to lie either side of the bladder. The arteries and vein have distinct walls with anechoic or echogenic material in the lumen. Each artery is normally 0.5 to 1.4 cm (mean, 0.85 cm) in diameter in transverse images. The urachus is not visible in the normal foal but lies between the two arteries. Both arteries and the urachus together measure 1.2 to 2.4 cm (mean, 1.75 cm) at the cranial pole of the bladder in transverse images. The umbilical structures are visible in most foals up to around 1 month of age. They are visible if they are diseased in older foals.

Umbilical Diseases

KEY POINT

Ultrasonography can demonstrate infection of internal structures of the umbilicus in foals with normal findings on palpation.

Infection of the umbilicus is readily diagnosed with ultrasonography. The condition should not be ruled out on the basis of palpation of the external remnant because infection of the internal structures can occur without external signs. The arteries and urachus are more commonly infected than the umbilical vein. Infected structures are enlarged and may contain anechoic or echogenic pus (Fig. 3-34). Gas echoes can be present. Ultrasonographic findings can aid in the selection of appropriate treatment. If multiple structures are affected or if the infected structures are more than twice their normal size, surgical resection should be considered. Conversely, with less severe infection, antibiotic therapy is warranted. Ultrasonographic monitoring is useful to monitor the response to therapy, and if the umbilical structures continue to enlarge after antibiotic treatment is initiated, the antibiotic selection should be changed or surgery should be performed.

Persistent urachus refers to a urachus that has been open since birth. *Patent urachus* is defined as a urachus that has been closed but opens secondary to infection or in response to excessive recumbency of the foal with failure to micturate properly. Ultrasonography is used in foals with urine dripping from the urachus to investigate the possibility of infection.

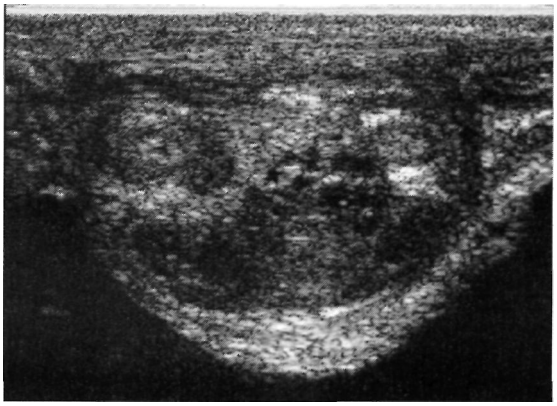


Figure 3-34. An umbilical ultrasonogram from the caudal abdomen in a 7-day-old Thoroughbred colt with infection of the urachus and left umbilical artery. The umbilical arteries lie on either side of the urachus, which is enlarged and contains hyoechoic material. The left artery is enlarged.

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Urachal cysts and diverticuli occur fairly commonly in young foals. They can be asymptomatic or cause increased frequency of urination in otherwise healthy foals. Ultrasonographically, small collections of urine are visible within the urachus. Normally, these resolve spontaneously as the foal grows.

INTESTINAL DISEASE

KEY POINT

Ultrasonography is particularly helpful in examination of the intestine in animals that are too small to permit rectal examination.

The intestines are examined from the ventral and lateral aspects of the abdomen using 3.5-MHz sector transducers in adults and 5- to 7.5-MHz sector or linear transducers in foals. In the adult horse, it is difficult to identify most of the intestine specifically. Portions that can be recognized are the stomach wall, immediately below the spleen, and the ventral colon, which has haustra, in the ventral abdomen. Portions of small intestine can often be recognized in the caudal ventral abdomen. Normal small intestine is collapsed and empty, although movement of gas and ingesta may be appreciated. In the foal, the colon is less well developed so that small intestine is present in most of the ventral abdomen.

Ultrasonography is useful in selected cases of abdominal catastrophe or colic. To some extent, this diagnostic tool can be regarded as an alternative to rectal examination in animals that are too small to examine safely. This is particularly true of foals, because small intestinal conditions are common in this age group, and the less well-developed colon allows a relatively greater proportion of the abdomen to be examined.

Distended edematous loops of intestine are heavy and fall to the ventral abdomen so that the foal should be examined from the ventral abdomen if it is standing or from the most dependent portion of the abdomen if it is recumbent. Obstructed small intestine is dilated and fluid filled, with edematous walls and absent motility. In enteritis, the intestine is also distended, but some motility will usually be present. The presence of both distended (proximal to obstruction) and collapsed (distal to obstruction) loops of intestine is highly suggestive of a surgical lesion, because in most cases of enteritis, the entire small intestine will be involved. Jejunojejunal, cecocecal and cecocolic intussusceptions can be visualized ultrasonographically as a donut appearance consisting of portions of intussuscepted intestine within a thickened edematous loop. Ileocecal intussusceptions are not usually visible because the intussuscepted portion is masked by gas within the cecum so that typically only the prestenotic loop of distal small intestine is seen. In adult horses with small intestinal obstruction, ultrasonography may reveal distended edematous loops of small intestine before there is sufficient small intestinal distension to be palpable per rectum and before nasogastric reflux occurs.

KEY POINT

Distended nonmotile loops of small intestine with thickened walls and adjacent loops of collapsed intestine are suggestive of a lesion that requires surgical intervention.

Ultrasonography is used to confirm the left lateral and dorsal displacement of the large colon. In lateral displacement, the colon is present between the body wall and spleen but is not dorsal to the kidney. In nephrosplenic entrapment (left dorsal displacement) of the colon), the large intestine is visible in the nephrosplenic space, obscuring the caudal pole of the kidney. Care must be taken not to confuse small colon with large colon because it is normal for portions of small colon to be found in this location. Small colon is smaller and does not run cranially between the spleen and the body wall.

Ultrasonography demonstrates gastric squamous cell carcinoma as a large heterogeneous echogenic mass between the spleen and the stomach wall. In other forms of intestinal neoplasia such as lymphosarcoma, the diffuse nature of the infiltrate is difficult to appreciate ultrasonographically. However, as the resolution of ultrasonographic equipment improves, there may be potential for identification of infiltrative intestinal diseases in future.

THE PERITONEAL CAVITY

The peritoneal cavity is examined from the ventral body wall with transducers of 3.5 to 7.5 MHz, depending on the size of the horse and the volume of peritoneal fluid to be examined. Ultrasonography can be used to identify and characterize peritoneal effusions. In horses in which it has been difficult to obtain peritoneal fluid by conventional means, it can be helpful to search for a pocket of fluid that is accessible for aspiration. This can be done with any high-frequency linear or sector

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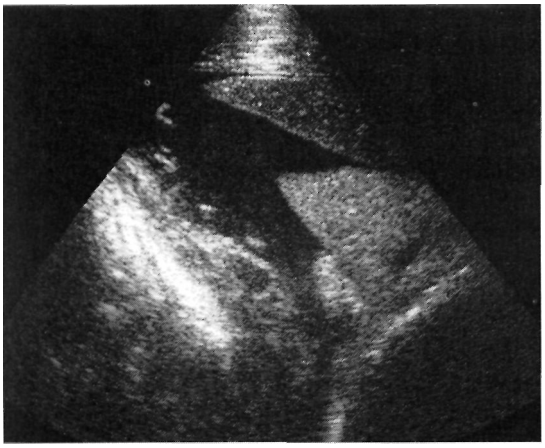


Figure 3-35. An ultrasonogram obtained from the left cranioventral abdomen in an aged mare with hemoperitoneum. There are large quantities of slightly echogenic peritoneal fluid. The strands of material floating within the fluid are omentum.

transducer. Increased volumes of peritoneal fluid occur with inflammation (septic and nonseptic peritonitis), uroperitoneum, hemorrhage (Fig. 3-35), or transudates (e.g., in ascites due to congestive heart failure or secondary to neoplasia).

In peritonitis, ultrasonography is used to search for underlying causes and to assess the volume of fluid, thus monitoring the response to therapy. If large amounts of flocculent material and/or gas echoes are in the dorsal abdomen, intestinal rupture may be present. Hemoperitoneum should be suspected in horses with abdominal pain in which multiple hemorrhagic samples of peritoneal fluid are obtained. Ultrasonography is a convenient means to distinguish splenic puncture from a true hemorrhagic peritoneal effusion. It usually is difficult to identify the precise source of hemorrhage, but possibilities include bleeding from a neoplasm such as granulosa cell tumor or vascular rupture, particularly if the signs of pain are associated with strenuous exercise or occur in a periparturient mare where rupture of the uterine artery is a possibility. Hemoperitoneum usually is associated with increased quantities of anechoic or sometimes echogenic fluid, but aspiration will easily confirm its nature (see Fig. 3-35). Neoplasms such as mesothelioma are associated with huge quantities of peritoneal fluid.

MESENTERIC AND VASCULAR LESIONS

Ultrasonography is the technique of choice for examination of the aortoiliac quadrification. This area is examined per rectum using high-frequency, linear, or sector transducers. Aortoiliac thrombosis produces echogenic masses within one or more of the vessels. Early thrombi are hypoechoic and increase in echogenicity as they mature. Changes in the size and extent of the thrombus can be used to monitor response to therapy. The cranial mesenteric artery also can be examined per rectum by placing the transducer directly on the mesenteric root. It is usually easiest and safest to do this with a forward-pointing transducer (i.e., one that is normally used for transcutaneous imaging) because the beam can be directed cranially toward the vessels without the operator having to push excessively to reach the affected area. In cranial mesenteric arteritis, multiple vessels are affected. The vessels are tortuous with thickened walls that often are surrounded by fibrous material.

The main differential diagnosis for mesenteric masses are neoplasia, abscessation, or granuloma. Any intra-abdominal mass that is palpable per rectum is amenable to ultrasonographic examination via this route. Use of a standard forward-pointing transducer often facilitates the examination of these masses because it extends the reach of the ultrasound beam. Microconvex transducers are particularly helpful because their small size makes them easy to manipulate within the confines of the rectum. In foals and weanlings in which abdominal abscessation is suspected, the abdomen is examined transcutaneously.

Ultrasonography is used to distinguish solid from fluid-filled structures and determine any attachment to abdominal organs. Neoplasms and granuloma are typically solid, well encapsulated, and echogenic (Fig. 3-36). They may be either uniform or of mixed echogenicity. It is not possible to definitively diagnose specific forms of neoplasia based on ultrasonographic appearance, but ultrasonography may aid in the collection of transcutaneous biopsies. Abscesses are usually fluid filled. The fluid is often extremely echogenic and a dorsal gas cap may be visible.

Section 5. Ultrasonography of the Head and Neck

KEY POINT

Ultrasonography can be used to examine a wide variety of soft tissue structures in the head and neck.

Ultrasonography can be used to examine the muscles, lymph nodes, salivary glands, and ves-

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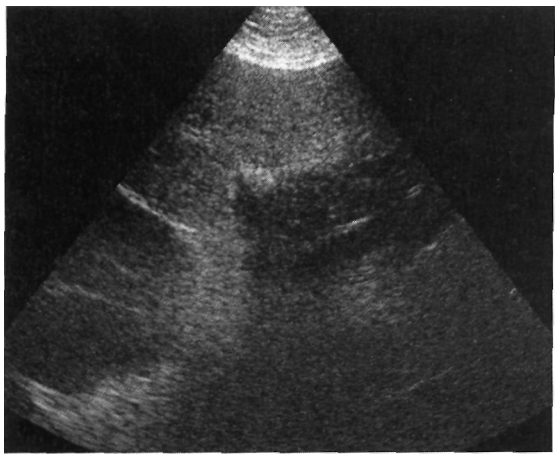


Figure 3-36. An ultrasonogram from the left paralumbar fossa in an aged pony with tuberculosis. A large hypoechoic mass is visible dorsal to the kidney that is a granulomatous mesenteric lymph node.

sels of the head, the eye, the retropharyngeal area, the thyroid gland, the jugular veins, cervical portions of the esophagus, and the cervical muscles. High-frequency transducers are used to obtain high-quality images. Sector transducers are more versatile because they allow the operator to "look around" bones and gas in the airways to reach the soft tissues. Ultrasonography is an extremely useful diagnostic tool in a variety of specific conditions of the soft tissues of the head and neck, and it has many applications in ophthalmology. In general, it is not helpful in conditions of the airways.

Ultrasonography is indicated in horses presenting with swelling in the retropharyngeal, cheek, and parotid areas. Diffuse swelling can be caused by edema, cellulitis, or hemorrhage, for example, from the guttural pouch or secondary to rapture of the longus capitis muscle. Hemorrhage and edema cause clear anechoic pockets of fluid, whereas the presence of gas and echogenic debris is suggestive of infection. Abscessation of the retropharygneal lymph nodes creates masses that are visualized by placing the transducer behind the vertical ramus of the mandible and directing the ultrasound beam dorsally and cranially. Bilateral swelling of the parotid glands is an allergic condition or rarely can be associated with viral infection. In these horses, the parotid glands are enlarged but their internal architecture is normal, composed of multiple uniform echogenic lobules. Salivary calculi usually are associated with unilateral parotid swelling and echogenic shadowing masses within the parotid duct or gland itself (Fig. 3-37). Ultrasonography can be helpful in exploring discharging tracts or localized masses such as dentigerous cysts and can be used to confirm thyroid gland enlargement.

All contents of the globe can be examined. Ultrasonography is particularly helpful in horses if corneal opacity prevents a full ophthalmoscopic examination. Foreign bodies, cataracts, posterior senechiae, and retinal detachments can be identified. Examination of the retrobulbar area is also possible. Ultrasonography provides a simple means of diagnosis of glaucoma by demonstration of enlargement of the affected eye.

In the neck, ultrasonography is used to identify lesions such as intramuscular injection abscesses, thrombophlebitis, and to assess the esophagus. Thrombophlebitis can occur after intravenous or perivascular injection or venous catheterization. Swellings associated with the jugular veins are assessed to determine if the swelling is perivascular rather than intravascular, to identify thrombi, to attempt to distinguish septic from nonseptic thrombi, and to assess patency of the vein. A nonseptic thrombus has uniform echogenicity (Fig. 13-38), whereas with sepsis, the thrombus is more heterogeneous with pockets of fluid or pus. Aspiration of a nonseptic thrombus should be avoided, but ultrasonography allows identification of a suitable fluid-filled site for aspiration for bacterial culture if sepsis is present. Patency of the vessel is assessed by observation of blood flow by Doppler ultrasonography or simply by observing that a lumen appears and changes size as the vessel is held off and released with two-dimensional ultrasonography.

Ultrasonography is most useful in the esophagus when there is rupture or an extraluminal mass

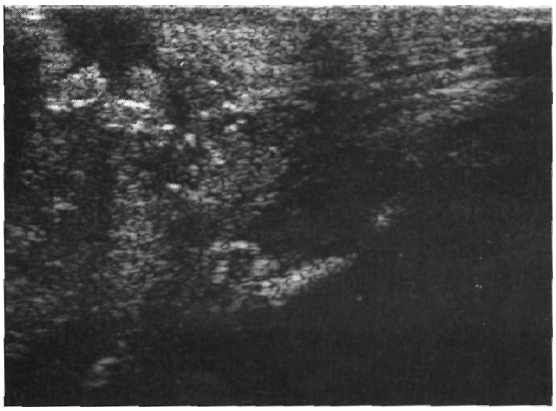


Figure 3-37. An ultrasonogram of a discharging tract and swelling in the left parotid area. Two calculi are visible in the tract and the area of the parotid duct, proximal to the calculi, is distended with hypoechoic material representing pus.

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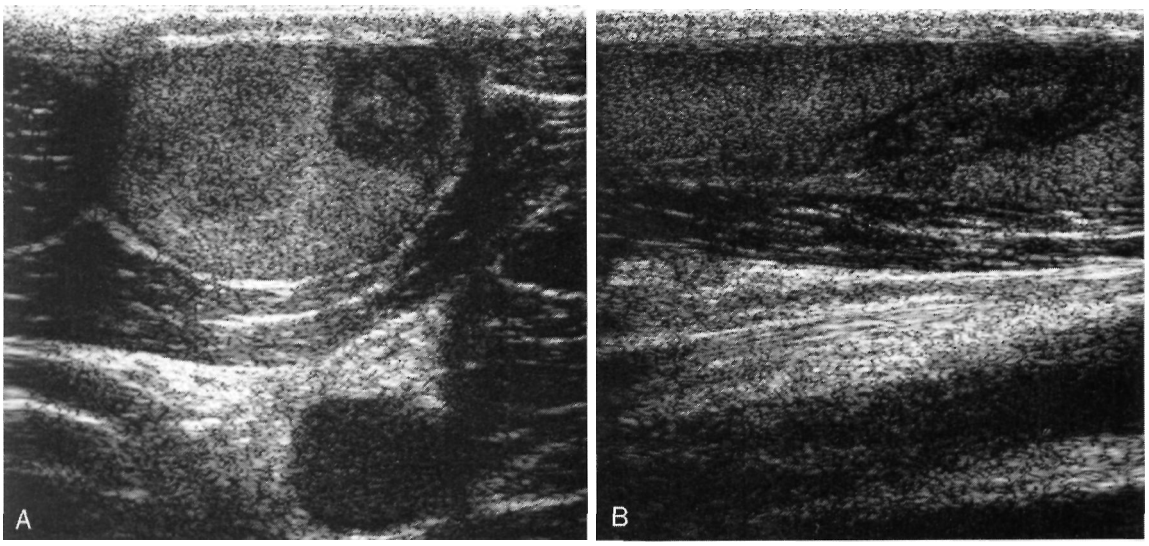


Figure 3-38. Transverse (A) and longitudinal (B) ultrasonograms of the jugular vein in a horse with a nonseptic thrombophlebitis. The thrombus is uniform and hypoechogenic. It has formed along the site of a previous jugular catheter. CA, carotid artery.

in the cervical region. The normal esophagus lies dorsal to the carotid artery and lateral to the trachea on the left side. Small fibrous bands around the esophagus can be demonstrated and, with esophageal rupture, the site may be visible and gas and fluid can be seen tracking along the facial planes adjacent to the esophagus.

Section 6. Radiography

Radiography remains the mainstay of diagnostic imaging in the horse. It is widely used in the diagnosis of orthopedic conditions, generally after the use of nerve blocks to localize the source of pain to an area or on its own where there are obvious localizing signs. Radiography also is used for examination of the upper respiratory tract and the lungs. Imaging of the gastrointestinal tract with radiography in adults is limited to the teeth and esophagus but also may be used to identify enteroliths. Radiographic examination of the abdomen is possible in foals. Plain radiography is used to assess the bony structures surrounding the brain and spinal cord. Myelograms are used to determine the degree of spinal cord compression in cases with suspected spinal cord compromise.

EQUIPMENT

Most equine radiography can be performed using a portable x-ray machine. Better images of the proximal limb will be obtained with machines with a higher mA output. These include large mobile x-ray machines and ceiling-mounted units. There are a wide variety of screen film combinations, and advice should be sought as to what is best suited to the x-ray equipment.

TECHNIQUE

The horse should be adequately restrained to allow positioning of the plates and to prevent movement of the body part undergoing examination. Light sedation generally is adequate for most horses. Where long exposure times are required, general anesthesia is preferable. General anesthesia generally is used for radiographs of the pelvis, although a technique for standing views has been described. Myelograms of the spine always are taken under general anesthesia to minimize the risk of adverse reactions and to allow manipulation of the neck for flexed and extended views.

Radiographic exposure depends on the total amount of x-rays (mA), the energy of the x-rays (kV), the distance from the x-ray tube to the film, and the thickness and density of the tissues. It is important to maintain a constant film focal distance for each anatomic location, usually 70 to 100 cm. To improve penetration of tissues, the kV is increased. In general, lower kV tends to give better contrast and is used for imaging bones, whereas higher kV reduces contrast and is most useful for soft tissue detail. Exposure time, which

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is limited by the mA output of the x-ray machine, is critical in conscious horses where blurring due to movement is a problem. Long exposure times and even double exposures can be used in anesthetized horses. Exposures for each body part and for various horse sizes should be recorded to allow an exposure chart to be built up.

Grids are used to minimize the effects of scattered radiation on the image. They are generally used for tissue thickness of greater than 11 cm. When using grids, the exposure must be increased and the cassette must be perpendicular to the primary beam. Coning down of the primary beam also improves detail by reducing the amount of scattered radiation.

It is extremely important to minimize the exposure of personnel to radiation during every radiographic procedure. The person holding the cassette should wear a lead apron and gloves and a cassette holder should be used where possible.

KEY POINT

No part of the body should be within the primary x-ray beam, even if it is protected by lead material.

SPECIFIC EXAMINATIONS

Foot

KEY POINT

When radiographing the foot, the hoof must be properly prepared.

The sole and the frog should be trimmed and the hoof wall brushed to ensure that all debris is removed. The sulci of the frog are then packed lightly and evenly with molding clay to prevent air shadows.

A number of views of the foot have been described. Four views are generally the minimum required: a lateromedial and dorsopalmar view of the distal phalanx, a coned down dorsopalmar view of the navicular bone, and a palmaroproximal-palmarodistal view (special navicular view) of the navicular bone. The lateromedial view is taken with the foot resting flat on a block so that the primary beam can be centered on the distal margin of the distal phalanx.

KEY POINT

A radiopaque marker placed on the dorsal surface of the hoof wall makes it easier to assess the degree of rotation of the pedal bone, in the lateromedial view.

The dorsopalmar views are taken with the toe resting in a block and the sole at 15 degrees to the plate. A grid is used for the dorsopalmar view of the navicular bone. Additional views include a weight-bearing dorsopalmar view, oblique views of the distal phalanx, and a palmaroproximal-palmarodistal view of the distal phalanx. The weight-bearing dorsopalmar view allows assessment of mediolateral foot balance and further assessment of the distal interphalangeal joint. Oblique views of the distal phalanx must be taken when a fracture of the distal phalanx is suspected and are also useful for assessing modeling of the extensor process.

Fetlock

A standard set of views include a standing lateromedial, flexed lateromedial, dorsolateral palmaromedial and dorsomedial palmarolateral obliques, and dorsopalmar/plantar views. To elevate the sesamoids above the joint space for the dorsopalmar view, the x-ray beam is angled down 10 degrees from the horizontal. A number of other views have been described. Elevated obliques (e.g., dorsal 35 degrees proximal 55 degrees lateral-palmar distal lateral oblique) are used to identify basal sesamoid fractures and proximal plantar/palmar first phalanx fragments and may also be used to identify plantar/palmar condylar lesions of the third metatarsus/metacarpus. An elevated lateromedial view (50 degrees proximal lateral-distal medial) will highlight the abaxial surface of the sesamoid bones. A flexed dorsopalmar or plantar-dorsal view with the limb drawn forward (most easily performed with the foot resting in a navicular block) is used to highlight the palmar/plantar aspect of the condyle of the third metacarpus/metatarsus. A contrast arthrogram (which can be performed either with air or radiopaque contrast agents) of the fetlock joint is useful for confirming proliferative synovitis.

Carpus

A minimum of five views are taken of the carpus: flexed lateral, dorsolateral palmaromedial and dorsomedial palmarolateral obliques, dorsopalmar, and flexed proximodistal skyline of the third carpal bone. Skylines of the proximal row of carpal bones or the distal radius also may be included when indicated. For weight-bearing views, the x-ray beam is centered on the midcarpal joint. The flexed lateromedial view separates the articular surfaces of the radial and intermediate carpal bones, which assists identification of the individual bones affected by pathology. The least useful

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view for assessing carpal pathology is the dorsopalmar view.

Tarsus

Most conditions of the tarsus can be adequately investigated with dorsoplantar, lateromedial, and dorsolateral-plantaromedial and dorsomedial-plantarolateral oblique views. For the lateromedial view, the x-ray beam is angled down 5 degrees and centered on the centrodistal joint. A flexed proximodistal view of the calcaneus is useful for assessing the sustentaculum tali. A flexed lateromedial view allows evaluation of the proximoplantar aspect of the trochlear ridges of the talus.

Stifle

Caudolateral-craniomedial oblique and caudocranial views are adequate for most conditions of the stifle. An oblique rather than a true lateromedial view is easier to position and highlights the lateral trochlear ridge of the femur, which is a common site affected by osteochondrosis lesions. The x-ray beam should be centered on the femorotibial joint, which is identified by palpating the proximal extent of the tibial crest. For the caudocranial view, the beam is angled down 10 degrees. To better highlight the apex of the patellar and the insertion of the cranial cruciate ligament, a flexed lateromedial view is used. If a patellar fracture is suspected, a flexed cranioproximal-craniodistal skyline view is necessary because these fractures often have a sagittal configuration.

Pelvis

Although a technique for taking radiographs of the pelvis, with the horse standing, has been described, pelvic radiographs generally are taken with the horse in dorsal recumbency under general anesthesia. If a pelvic fracture is suspected, the horse should be stall rested for 6 weeks to reduce the risk of complications associated with recovery from anesthesia with an unstable fracture. High output radiographic equipment (more than 100 kV, 200 mA) and a focused grid are required. Ventrodorsal views are obtained of the tuber ischii, pelvic canal, sacroiliac joints, and iliac wing. The horse is then rocked to each side for views of the coxofemoral joint.

Head

Obtaining radiographs of the head is greatly facilitated by a plate-holding device either attached to a wall or on a stand. Sedation will lower the head and allow easier head positioning. A rope halter should be used. Lateral radiographs of the sinuses and guttural pouches require relatively low exposures due to the small amount of tissue involved, and thus a portable x-ray machine can be used. For the sinuses, the x-ray beam is centered over the maxillary sinus and the beam angled horizontally. Oblique views are necessary for evaluation of the cheek teeth to prevent superimposition of the tooth roots of the opposite side. The side being radiographed should be positioned against the plate. The beam is angled up or down 30 degrees to highlight the mandibular and maxillary teeth, respectively. Higher exposures are used for tooth roots than the sinuses. In general, good quality well-aligned dorsoventral views will require general anesthesia. Lateral radiographs of the guttural pouch, larynx, and pharynx also can be obtained using portable equipment. The beam should be centered over the caudal aspect of the vertical ramus of the mandible.

Thorax

Radiography of the thorax requires a large x-ray machine and movable cassette holder. A grid will greatly improve the image quality. In foals, radiographs can be taken in lateral recumbency. Four overlapping views generally are required to cover the whole thorax in adults. The ventral views require high output equipment due to the muscle mass that must be penetrated.

Abdomen

In adults, radiographs of the abdomen are rarely of use except to confirm the presence of enteroliths. Abdominal radiographs in foals are used in the investigation of esophageal, gastric, and intestinal obstructions. Standing lateral radiographs are of most value in these cases. The area of interest should be positioned nearest to the cassette. Contrast agents may be helpful for evaluation of esophageal function and also commonly are used to assess foals with suspected gastroduodenal obstruction.

Spine

CERVICAL SPINE

Lateral radiographs usually are obtained standing. Portable equipment can be used for evaluation of the cranial and midcervical regions, but high output equipment is needed for the caudal cervical vertebrae. A grid will improve image quality. As

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with the head, a wall-mounted cassette holder or a mobile stand is of great benefit. Generally, four views are taken centering over the atlas, C2, C4, and C6. It is important that the neck is perpendicular to the beam so that the vertebrae are correctly aligned.

THORACOLUMBAR SPINE

High output equipment is needed to take adequate radiographs of the thoracolumbar spine, although adequate radiographs of the dorsal spinous processes of the withers can be obtained with lower output units. A cassette-holding device, preferably linked to the x-ray tube, reduces exposure of personnel and greatly facilitates the procedure. An aluminum wedge filter improves visualization of the dorsal spinous processes.

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CHAPTER 4

Musculoskeletal System

R. Christopher Whitton, David R. Hodgson, and Reuben J. Rose

Problems involving the musculoskeletal system are common reasons for veterinary attention to all breeds and types of horses. In racehorses, surveys in all countries have indicated that musculoskeletal problems are the most common reason for interruptions in training. In evaluation of horses before sale, considerable attention should be paid to the musculoskeletal system because this is likely to be the major cause of future disputes between the seller, purchaser, and veterinarian.

Most horses are presented because of lameness, with or without some degree of swelling in one or more of the limbs.

KEY POINT

A systematic examination procedure should be adhered to in all cases of lameness because, on occasion, the most spectacular swellings of the limbs may be of little functional significance.

GENERAL CONSIDERATIONS

The examination should commence after obtaining a precise history that should indicate whether the onset was sudden or insidious, the relationship of the presenting signs to exercise, details of medications received, and shoeing. It is important to remember that the history may not be reliable because many owners or trainers will provide a history that is consistent with their view of the problem.

Before a detailed examination commences, an evaluation of the conformation and symmetry of the musculoskeletal system should be undertaken. Particular note should be made of any swellings or muscle atrophy for later detailed investigation.

The horse should be walked, initially, on a hard surface and note should be taken of limb coordination and any indications of lameness. Special note should be made of any signs of incoordination, indicating a possible neurologic problem. These signs may be less apparent at the trot. *Forelimb lameness* is most easily diagnosed by watching the horse walk and trot on a firm even surface both toward and away from the observer.

As weight is borne on the affected forelimb, the head is lifted; the head is dropped when weight is placed on the unaffected forelimb.

KEY POINT

For the inexperienced observer, it is easier to watch for the forelimb on which the head drops because this allows concentration on one phase of the gait.

In the case of bilateral forelimb lameness, it may be difficult to detect any head movement. However, affected horses will usually show a reluctance to stretch forward and will have a stilted or shuffling gait. Many owners and trainers will describe such horses as being "tied up in the shoulders." Although some books emphasize classification of the lameness into a "swinging leg" or "supporting leg" lameness, it is very difficult to determine from the gait of the horse whether the problem is in the upper or lower limb. Only a detailed physical examination together with nerve blocks will definitively localize the lameness to a particular site.

Hindlimb lameness is often more subtle, and an affected horse will usually show no signs of head movement during trotting. Many owners or trainers will be unaware that their horse is lame, and the horse may be presented because of reduced performance or for having a "rough" or "choppy" gait. In the case of Standardbred trot-

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ters and pacers, trainers sometimes describe the horse as "jumping out of its gear." Diagnosis is best undertaken by watching the horse walk and trot on firm even ground away from the observer.

KEY POINT

When looking for lameness in a hindlimb, it is important to focus on a point in the midline (base of tail or tuber sacrale) as even in sound horses the hindquarters rock from side to side.

As weight is taken on the lame hindlimb, the hindquarters, particularly on the affected side, appear to rise, whereas when weight is taken on the sound limb, the hindquarters will drop.

In this section, particular emphasis is placed on diagnosis, prognosis, and treatment. One of the important early lessons learned by all clinicians is that often it is extremely difficult to provide an accurate prognosis for the client. We have all suffered damage to our egos when the racehorse with multiple limb problems, degenerative joint disease in several joints, and a strained tendon, which we have told the owner or trainer will never race again, ends up winning a major race. The important lesson from this is that forecasting likely outcomes is always a matter of probabilities, with some injuries carrying a better prognosis than others.

KEY POINT

Never tell the owner or trainer of a horse that it will never win another race or compete successfully, unless you know the horse is dead! This is the only way you can be sure that you will be correct.

EXAMINATION PROCEDURE

When examining a horse for either a forelimb or hindlimb lameness, the examination always should commence at the foot and progress proximally.

KEY POINT

It is important to remember that the majority of conditions causing lameness occur in the carpus or distally in the forelimb and in the tarsus or distally in the hindlimb.

Therefore, the examination should concentrate on the distal limb. There is an old saying that "the foot is the cause of lameness until proven otherwise," and this saying has considerable truth.

KEY POINT

In any lameness investigation, the foot should always be inspected and eliminated as a potential source for the lameness before any further examination.

With the busy schedule of practice, most of us ignore this sooner or later—to our cost. A typical story is as follows: A horse is presented with obvious carpal swelling, and radiographs are taken that show soft-tissue swelling. Advice is to treat with phenylbutazone for 7 days and rest the horse. The swelling disappears, but the horse is still quite lame. An examination performed by another practice shows that the horse has considerable pain on application of hoof testers and there is a fracture involving the wing of the distal phalanx (pedal bone). If the veterinarian performing the initial examination failed to examine the foot, he or she can appear incompetent. Clients always remember your mistakes more than your successes.

Forelimb

FOOT

Inspection and Assessment of Foot Conformation and Shoeing. The foot is inspected for signs of cracks, discharge, uneven wear, poor conformation, and improper shoeing. Particular note should be made as to how the shoe lies on the foot.

KEY POINT

The most common shoeing fault is where the shoe has excessive contact with the sole inside the white line, resulting in extra pressure on an area that was not designed for weight bearing.

The shoe normally should maintain contact with the hoof wall and a small amount of sole around the area of the white line. Uneven trimming of the foot may be evident when one stands behind the horse and evaluates the symmetry of the heels. If ground contact is not even, the heels may assume a "sheared" appearance, one of the bulbs of the heel being pushed higher than the other. A common shoeing fault in Thoroughbred horses occurs when the toe of the foot is left too long and the heel is cut too short, although in many horses this is due to poor heel growth rather than a fault of the farrier.

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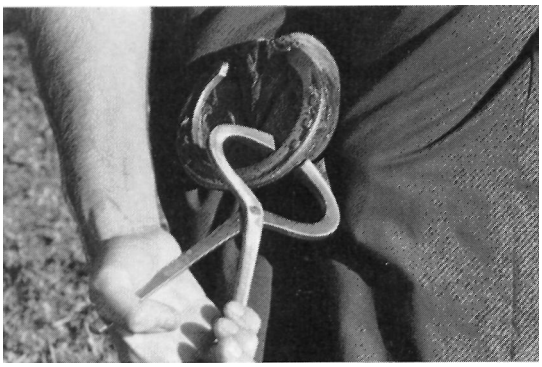


Figure 4-1. Examination of the foot using hoof testers. The testers are applied between the hoof wall and the sole to find whether pain is present.

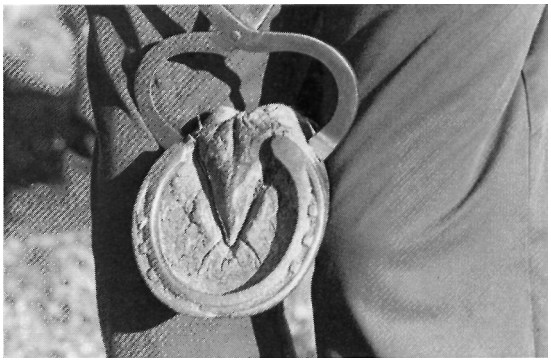


Figure 4-3. Examination of the foot using hoof testers. The hoof testers are being applied across the heels of the foot to localize pain to the palmar aspect of the foot.

Detailed Examination. Signs of increased heat around the coronary band and hoof wall should be determined by comparing with the opposite foot. With weight off the foot, the palmar region is examined to determine if there has been any loss of elasticity of the lateral cartilages, indicating the development of sidebone. Hoof testers can then be used to attempt to determine if pain is present in any part of the foot (Fig. 4-1).

KEY POINT

If signs of pain are found on hoof tester application, the area should always be rechecked and compared with the opposite foot.

Some horses with a unilateral forelimb lameness will show evidence of pain with hoof testers applied to the affected hoof, which initially can be thought to be the site of the problem. However, examination of the unaffected foot may disclose a similar painful response to hoof testers. Careful use of hoof testers applied around the hoof wall and periphery of the sole may localize the pain to a particular region of the foot, which is especially useful in dealing with problems such as foot abscesses. The hoof testers also should be applied across the frog (Fig. 4-2) and across the heels (Fig. 4-3) to determine if there are signs of pain. With the foot on the ground, the hoof wall is then tapped with a hoof hammer (Fig. 4-4) or closed hoof testers to determine if any painful areas are present, which may have been unapparent with the hoof testers.

PASTERN

Most problems in the pastern are due to direct trauma or concussion. Injuries to the proximal or middle phalanges usually result in swelling and



Figure 4-2. Examination of the foot using hoof testers. The hoof testers are applied over the middle third of the frog. Horses with pain in the navicular region may show pain on application over this area.



Figure 4-4. Tapping of the hoof wall with a hoof hammer, with the foot on the ground. Sometimes this will reveal pain when the use of hoof testers does not.

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Figure 4-5. Lateral view of the palmar pouch of the left fetlock showing distension of the pouch. The pouch is located between the suspensory ligament and the palmar aspect of the third metacarpal bone in the distal metacarpus.

pain, evident on various degrees of flexion or palpation of this region.

KEY POINT

Many normal horses will have swellings on the medial and lateral aspects of the distal part of the proximal phalanx, which are of little clinical significance.

However, in degenerative joint disease involving the proximal interphalangeal joint (ringbone), there can be severe changes present but little or no pain on flexion or palpation. Therefore, great care should be taken in assessment of pastern swelling and should include radiography of the pastern region if there is suspicion of joint involvement.

FETLOCK

Inspection of the fetlock should be undertaken to detect swelling over the dorsal region of the joint and the palmar pouches. The palmar pouches are located between the interosseous (suspensory ligament) and the palmar aspect of the third metacarpal bone. In the normal fetlock, this area will appear indented, but in joint disorders this will become distended with synovial fluid (Fig. 4-5). The fetlock joint should then be flexed as much as possible (Fig. 4-6) to determine the normal range of movement (usually 90 degrees) and to evaluate any pain that may be present in the joint. After this, the sesamoid bones are palpated over the abaxial surface of the fetlock to ascertain if pain is present.

A flexion test of the fetlock and lower joints should be performed. The fetlock is flexed for 1 minute and the horse trotted off (Fig. 4-7). Fetlock and phalangeal flexion tests are nonspecific and will accentuate lameness due to a variety of problems ranging from the foot to the fetlock. Note that the test should be performed with the carpus extended as much as possible so that lameness proximal to the fetlock is not emphasized.

METACARPUS

The structures of importance to the examination are the superficial and deep flexor tendons; infe-



Figure 4-6. Flexion of the left fetlock to determine a painful reaction.

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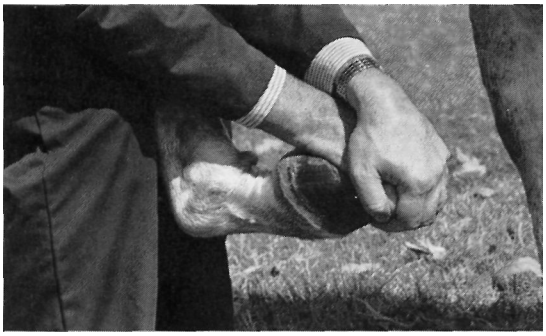


Figure 4-7. Flexion test demonstrating flexion of the fetlock and phalanges. Note that the carpus should be kept as extended as possible so that lameness can be localized to the distal limb. After 1 minute the horse is trotted off to determine if the extent of lameness is aggravated.

rior check ligament; interosseous (suspensory ligament); and second, third, and fourth metacarpal bones. The tendons and suspensory ligament should all be palpated individually with the weight off the affected leg (Figs. 4-8 and 4-9). The superficial flexor tendon is flat, whereas the deep flexor tendon is round. Particular note should be paid to any signs of swelling or pain on palpation.

KEY POINT

It must be kept in mind that all horses will show some pain on firm palpation of the suspensory (interosseous) ligament.

Significant pain generally is associated with thickening of the ligament or its branches. The inferior check ligament, situated in the proximal metacarpal region, should be palpated for signs of pain and swelling. The second, third, and fourth metacarpal bones should be palpated for signs of pain and swelling. Although the fourth metacarpal bone is rarely a source of lameness, the second metacarpal bone quite frequently is injured in pleasure horses and Standardbred racehorses.



Figure 4-9. Palpation of the suspensory (interosseous) ligament of the right foreleg to determine swelling and pain.

KEY POINT

The dorsal aspect of the third metacarpal bone should be palpated carefully in 2- and 3-year-old racehorses because "bucked shins" is a very common cause of lameness.

CARPUS

Most horses with carpal injuries show some degree of distension of the joint capsules of the midcarpal and/or antebrachiocarpal joints. The dorsal aspect of the carpus should be palpated over the appropriate joint to check for signs of synovial effusion and joint capsule swelling (Fig. 4-10), followed by extreme flexion of the carpus to determine whether there is resistance to flexion, reduced flexion, or pain.



Figure 4-8. Palpation of the flexor tendons in the metacarpal region of the left foreleg. Note should be taken of heat, swelling, and pain.



Figure 4-10. Palpation of the dorsal aspect of the left carpus for joint capsule distension and thickening over the proximal (radiocarpal) and middle (intercarpal) carpal joints.

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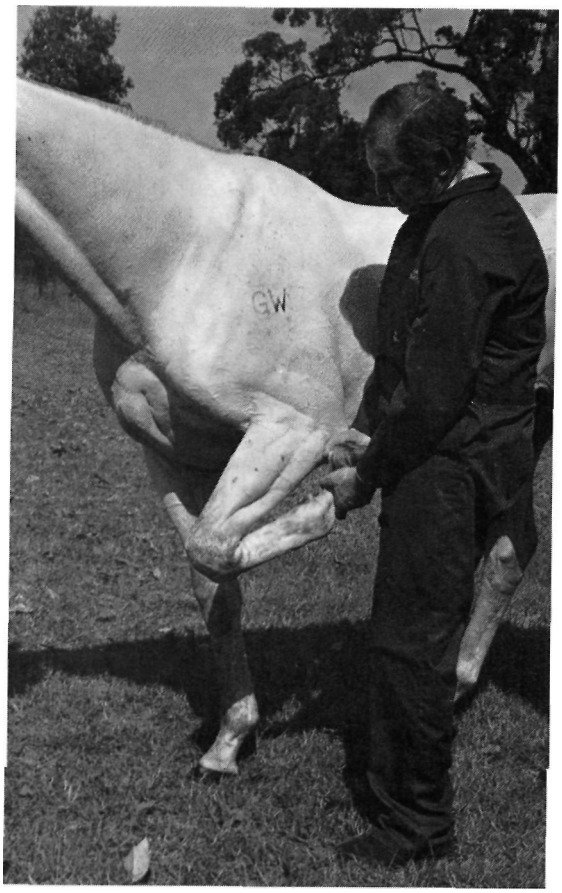


Figure 4-11. Extreme flexion of the left carpus to determine signs of pain localized to the carpus.

KEY POINT

Many horses with carpal problems only show signs of pain during the last few degrees of carpal flexion.

Therefore, the foot should be pulled up past the elbow joint to determine the response to severe flexion (Fig. 4-11). A carpal flexion test should be carried out, where the carpus is held flexed for 1 minute and the horse is trotted off. An increase in the degree of lameness increases the probability of a carpal problem, but this is not a very reliable test.

UPPER FORELIMB (RADIUS, ELBOW, HUMERUS, AND SHOULDER)

KEY POINT

Horses with lameness arising from the upper forelimb will seldom reveal any signs of pain on examination.

However, these areas of the leg are palpated, flexed, abducted, and extended to determine if any localizing signs can be found (Figs. 4-12 to 4-14). Attention also should be paid to careful inspection and palpation of the musculature to determine whether muscle injuries contribute to the lameness.

Hindlimb

The initial part of the examination (foot, pastern, fetlock, and metatarsus) is similar to that for the forelimb. Although lameness associated with the tarsus is common, careful examination of the hindlimb fetlock in performance horses has demonstrated that lameness associated with this joint is more common than was previously reported.

KEY POINT

It is particularly important in chronic hindleg lamenesses to evaluate the symmetry of the left and right gluteal region because atrophy of the gluteal muscles is a feature of such lamenesses.

This is best done by standing the horse square on level ground and examining the appearance of

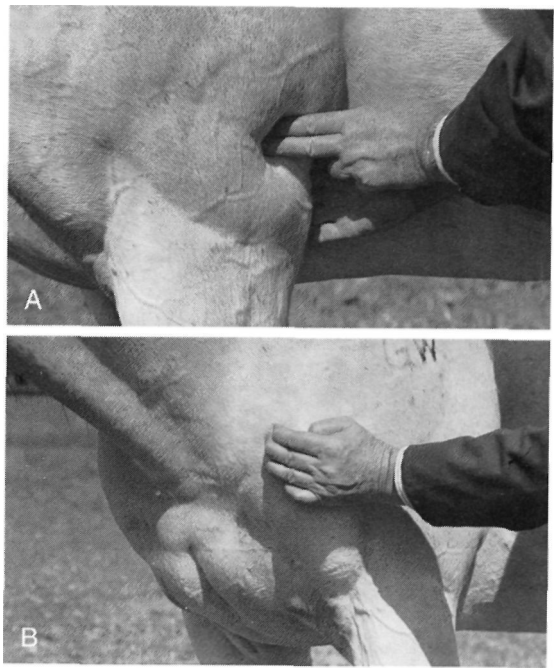


Figure 4-12. Palpation of the musculature in the upper forelimb to localize pain. *A.* Palpation over the triceps muscles. *B.* Palpation over the supraspinatus muscle.

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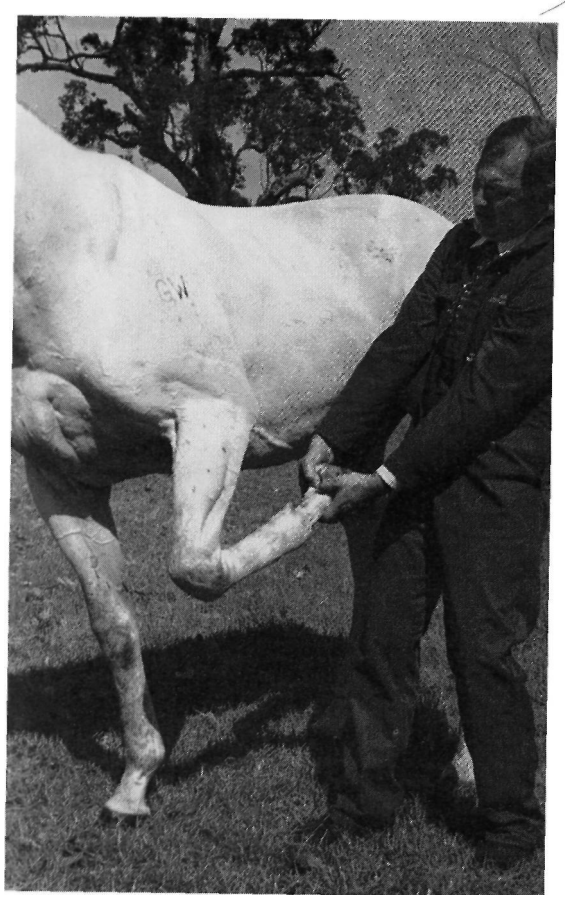


Figure 4-13. Flexion of the upper foreleg to determine whether pain is present.

the hindquarters from behind the horse (but not too close!).

TARSUS (HOCK)

The hock should be inspected for signs of swelling, particularly the tarsocrural joint capsule, which is prominent on the dorsomedial aspect of the joint. The hock should be flexed and extended; however, this seldom produces any signs of pain.

After the initial examination, a spavin test should be carried out (Fig. 4-15).

KEY POINT

The spavin test is a nonspecific test because it results in flexion of the hock, stifle, fetlock, and hip joints to some degree.

The hock is held flexed for 2 minutes (timed by watch) by holding the leg at the midmetatarsus and parallel to the ground. The horse is then trotted off, and if there is an intra-articular problem, the lameness may be aggravated for the first six to eight steps. The spavin test will aggravate lameness due to problems in the hock, stifle, and occasionally the hip. A separate fetlock flexion test should be performed because fetlock problems may give a positive spavin test.

STIFLE

In many stifle problems there is distension of the joint capsule of the femoropatellar pouch just distal to the patella and between the patellar ligaments.

KEY POINT

The femoropatellar joint should be examined by palpating either side of the middle patellar ligament to detect distension (Fig. 4-16).

Because of inconsistent communication between the two joints, conditions in the medial

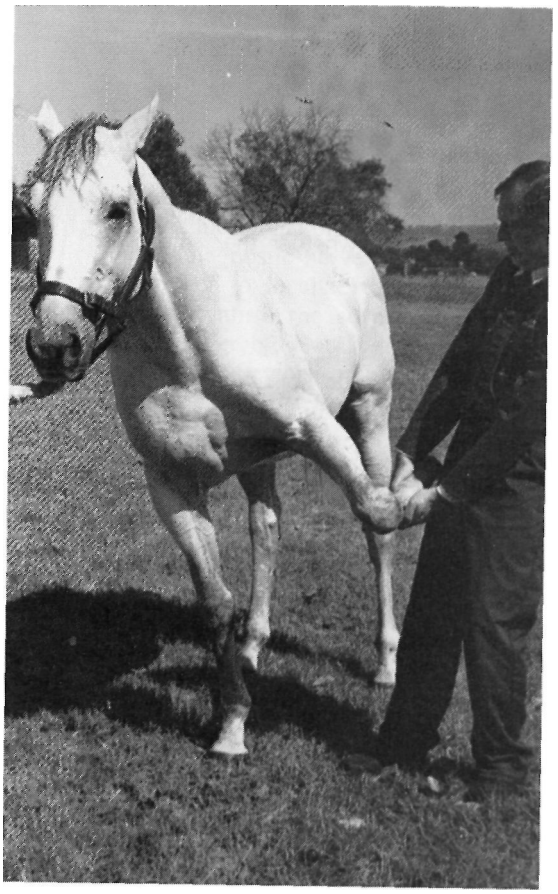


Figure 4-14. Abduction of the upper foreleg to determine whether pain is present.

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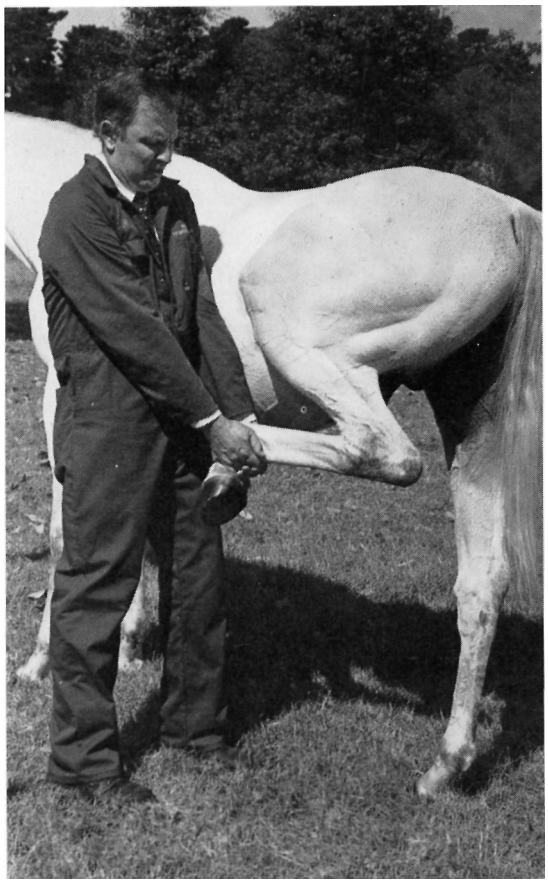


Figure 4-15. Spavin test. The hindleg is held in flexion for 2 minutes, after which the horse is trotted off to determine whether the lameness is aggravated.

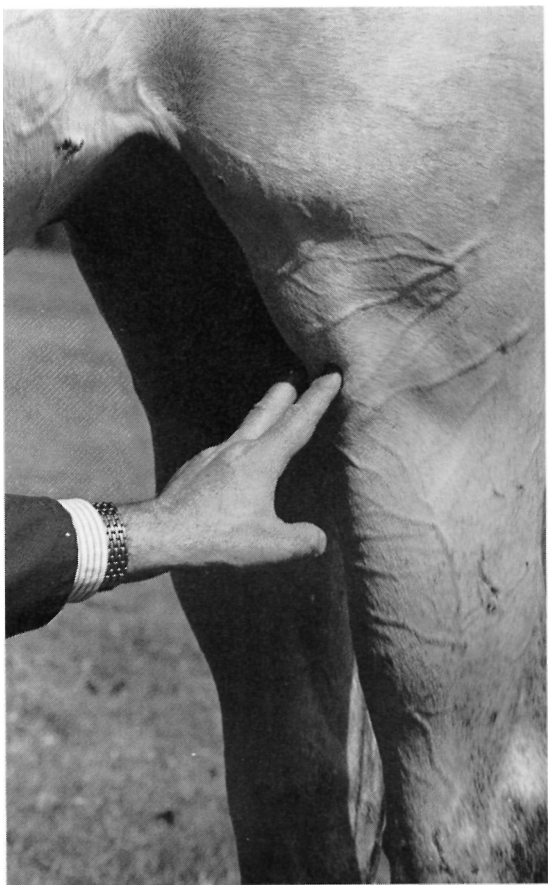


Figure 4-16. Palpation over the medial and lateral femoropatellar pouches to determine synovial effusion.

femorotibial joint do not always cause femoropatellar swelling. Swelling of the medial femoropatellar joint capsule may occasionally be detected cranial to the medial collateral ligament but is not a consistent feature of pathology in the medial femorotibial joint.

HIP

The hip only can be palpated indirectly by placing the palm of the hand over the greater trochanter of the femur (Fig. 4-17). In some cases of damage to the hip joint, crepitus can be detected in this fashion as the horse walks. Soft-tissue injuries around the hip joint are much more common than damage to the hip joint itself. Examination of this region should include palpation of the gluteal muscle mass bilaterally, because pain in this region may contribute to reduced performance.

Back

The clinical signs of back injuries may be quite varied and range from changes in temperament to



Figure 4-17. Palpation over the greater trochanter of the femur to detect signs of pain and crepitus involving the hip joint.

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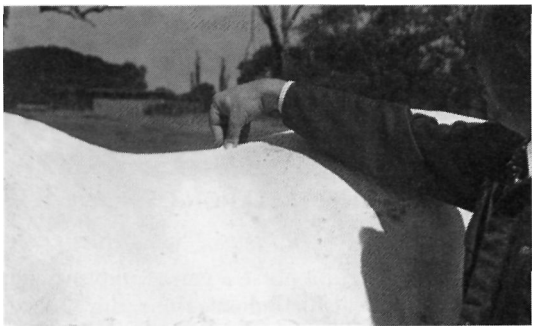


Figure 4-18. Palpation over the dorsal thoracic spinous processes for signs of pain or hypersensitivity.



Figure 4-20. Examination of the caudal sacral region of the back. The horse will normally arch its back on firm stroking.

reduced performance. Temperament problems as a clinical sign are quite common in dressage horses. The majority of problems arise from the thoracolumbar region, although sacroiliac pain is a common problem, particularly in Standardbreds.

The examination should begin with palpation over the dorsal thoracic spinous processes (Fig. 4-18) to detect any pain and should proceed caudally to the sacral region. Normal horses will "dip" their backs on stroking with a blunt object such as a pen over the thoracolumbar region (Fig. 4-19) and arch their backs on stroking over the caudal sacral region (Fig. 4-20). A back problem may be suspected in horses that do not show these responses, because the horse may be attempting to protect itself from back pain by holding its back rigid. Because a number of back problems involve soft-tissue injuries, the longissimus dorsi muscles on either side of the dorsal spinous processes also should be palpated and may be stroked with a firm object such as the blunt end of a ballpoint pen to check lateral flexibility of the back.

If sacroiliac pain exists, palpation over the tuber sacrale (point of the croup; Fig. 4-21) will result in the horse crouching toward the ground. Sacroiliac problems are common in Standardbred horses and may be manifest as gait disorders of a nonspecific nature. Trainers often complain about horses "jumping out of their gear" or "breaking on turns."

DIAGNOSTIC AIDS

Local Anesthesia and Nerve Blocks

In many cases, it may be impossible to localize the site of lameness from the clinical examination or there may be more than one contributing cause. In such cases, diagnostic nerve blocks should be undertaken, either to localize the source of the problem or to confirm the suspected site. This may be done either by blocking sensory nerves innervating specific regions or by injecting local anesthetic intra-articularly. Before all injections, a strict surgical disinfection of the site should be performed. For intra-articular injections, correct

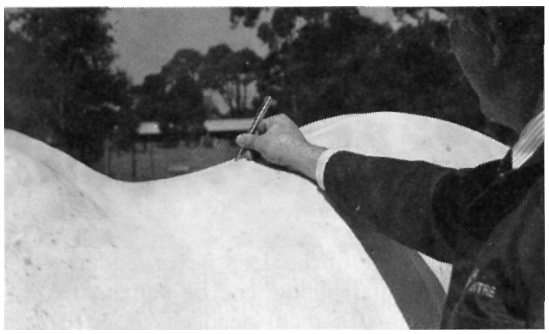


Figure 4-19. Examination of the thoracolumbar region by stroking with a pen. The horse will normally dip its back.

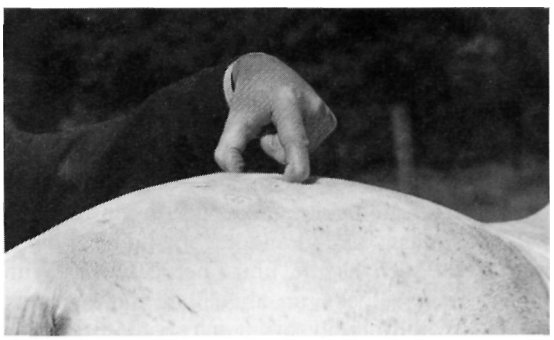


Figure 4-21. Palpation over the tuber sacrale to determine pain in the sacroiliac region.

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placement of the needle in the joint can be confirmed in most joints by the presence of synovial fluid that drips from the needle or may be found on aspiration. It is important to use sterile gloves for intra-articular injections, and clipping or shaving the injection site is preferred.

KEY POINT

In some performance horses, clients object to the hair being removed over the site, and in these cases it is acceptable not to remove the hair.

However, the site should be well prepared by scrubbing for at least 3 minutes with povidone-iodine scrub, followed by application of 70% alcohol. A final skin preparation utilizes an iodine-based product, which can either be tincture of iodine mitis or povidone-iodine (see Disinfectants, Chapter 19). It is important to use a new needle to enter the joint rather than the needle used for aspiration of local anesthetic into the syringe.

CHOICE OF LOCAL ANESTHETIC

While a variety of local anesthetics may be used, lidocaine (Treatment No. 67) is somewhat more irritating to the tissues than such drugs as prilocaine and mepivacaine (Treatment Nos. 72 and 93). For this reason, and because the latter local anesthetics diffuse better and have a more rapid onset of action than lidocaine, prilocaine and mepivacaine are the local anesthetic drugs of choice.

KEY POINT

// the local anesthetic is to be administered intra-articularly, a new bottle should be used rather than one from which samples have already been taken.

ONSET OF ANALGESIA

With the use of prilocaine or mepivacaine, blocking regional nerves distally usually results in analgesia within 3 to 5 minutes. A positive result can be assessed quickly, but if there is no improvement, a period of 20 minutes should elapse before a negative response is assumed. More proximally where nerves are larger and deeper, desensitization will take longer, and 40 minutes should be allowed for blocks such as the tibial nerve block. With intra-articular blocks, an immediate improvement in gait is often found, but it may require as long as 20 to 30 minutes for a complete effect. This is an important point because if a subsequent nerve block is performed too quickly, the improvement in the lameness may be the result of the previous block rather than the current one.

PROBLEMS IN INTERPRETATION OF NERVE BLOCKS

While the failure of a horse's gait to improve after a nerve block usually indicates that the problem is elsewhere in the leg, there are occasions where nerve blocks may provide incorrect information. In cases where the lameness is of neurologic origin or where there are fractures, lameness may not be abolished by the nerve block. Therefore, this possibility should be considered when a horse's gait fails to improve despite considerable certainty that the region of the leg blocked is the site of the problem. A common example of this failure to respond to nerve blocks occurs in a horse with a fracture of the distal phalanx involving the distal interphalangeal joint. An abaxial nerve block, which should abolish the lameness, will result in only partial improvement in the gait.

The following sequence, commencing with the foot and proceeding proximally, is used in investigating a lameness problem where no localizing signs can be found during examination. If a specific area of the limb is thought to be involved, that region can be blocked to determine if the lameness originates from that area.

Forelimb

DESENSITIZING THE PALMAR ASPECT OF THE FOOT: PALMAR DIGITAL NERVE BLOCK

With the affected limb held up, the foot is extended and the ergot on the palmar aspect of the fetlock is pulled proximally. This tenses the ligaments of the ergot, which run palmaromedial and palmarolateral in the pastern (Fig. 4-22). The medial and lateral palmar digital nerves run just deep to these ligaments, and the nerves may be desensitized by inserting a 23- or 25-gauge, 12-mm (0.5-inch) needle to a depth of approximately 6 mm (0.25 inch) as shown in Figure 4-23. Another site, which is more distal and commonly undertaken by many veterinarians, is just above the most proximal part of the lateral cartilages, close to the edge of the deep flexor tendon where the neurovascular bundle can be palpated. A 25-gauge, 12-mm (0.5-inch) needle is inserted deep to the lateral cartilage (Fig. 4-24) or beside the palpable vessels for blocking the palmar digital

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Figure 4-22. With the left front fetlock extended and the ergot pulled proximally, the ligament of the ergot can be seen as the taut structure just distal to the left thumb of the person holding the foot. This is the landmark for insertion of the needle for the palmar digital nerve block.

nerve at this site, medially and laterally. Local anesthetic (1.5-2.0 mL of 2% prilocaine or a similar local anesthetic) is injected into each site, and the block can be assessed by pricking the palmar aspect of the heel with a pen about 5 minutes after injection. The horse is then trotted, and if the lameness is not abolished, the next site more proximal is assessed. However, in some cases, the effect of the block may not be complete for up to 15 minutes. Therefore, this period of time should elapse before a negative response to the nerve block is considered.

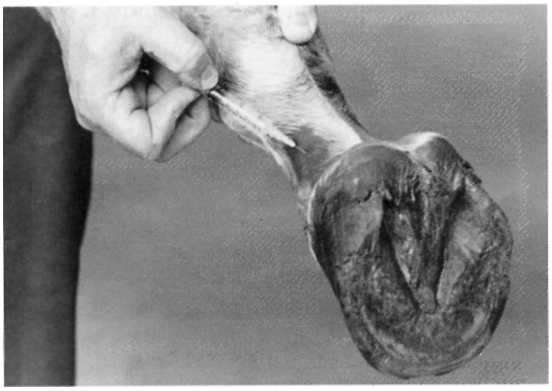


Figure 4-23. Palmar digital nerve block. A 23-gauge, 15-mm (5/8-inch) needle is inserted just underneath the ligament of the ergot. The photograph shows the position for desensitizing the left lateral palmar digital nerve. A volume of 1.5 mL of 2% local anesthetic is injected.

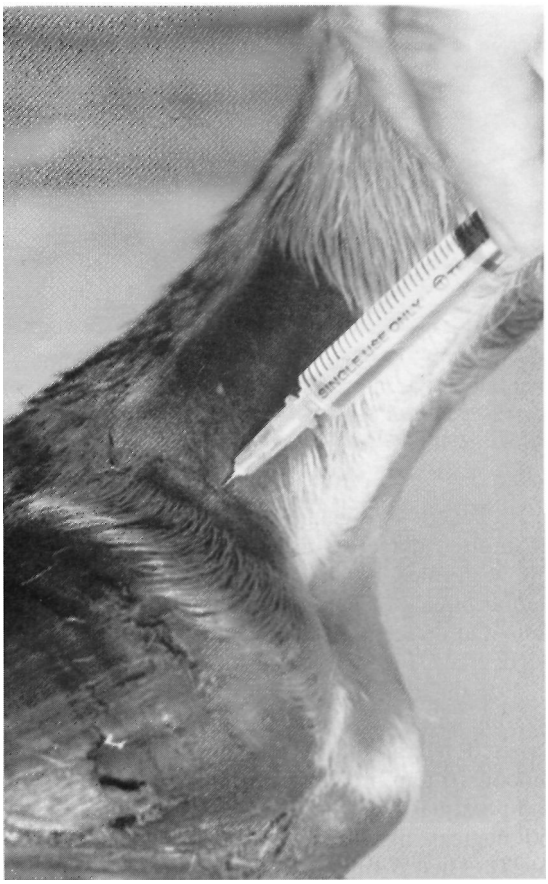


Figure 4-24. Palmar digital nerve block. A 25-gauge, 12-mm (0.5-inch) needle is inserted deep to the lateral cartilage or palmar to the palpable vessels for blocking the palmar digital nerve at this site, medially and laterally. A volume of 1.5 mL of 2% local anesthetic is injected.

DESENSITIZING THE DISTAL INTERPHALANGEAL (COFFIN) JOINT

To localize lameness to the coffin joint, a 21-gauge, 25-mm (1 inch) needle is inserted, about 15 mm (0.6 inch) above the coronary band and 15 mm (0.6 inch) medial or lateral to the dorsal midline and directed distally and toward the midline (Fig. 4-25). Then 5 mL of 2% local anesthetic is injected, and a period of 15 minutes should elapse before the gait is re-evaluated.

DESENSITIZING THE PROXIMAL INTERPHALANGEAL (PASTERN) JOINT

Arthrocentesis of the proximal interphalangeal joint can be performed in both the dorsal and palmar pouches of the joint. The palmar approach is the easier of the two. With the joint held par-

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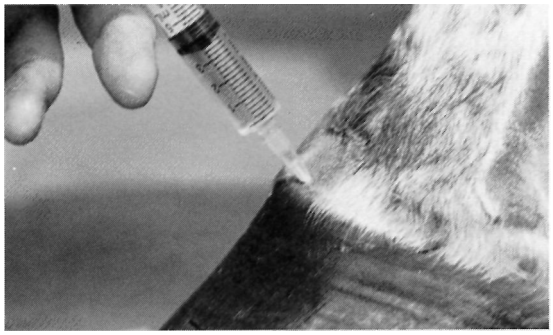


Figure 4-25. Position of a 21-gauge, 25-mm Cl-inch) needle in the coffin (distal interphalangeal) joint to desensitize the joint. A volume of 5 mL of 2% local anesthetic is injected.

tially flexed, a 20-gauge, 30-mm (1.5-inch) needle is inserted dorsal to the neurovascular bundle in the V formed by the proximal phalanx and the lateral branch of the superficial digital flexor tendon, and directed distal and medial at an angle of 45 degrees (Fig. 4-26).

For the dorsal approach, the limb is weight bearing and a 21-gauge, 25-mm (1-inch) needle is inserted lateral to the common digital extensor tendon 10 mm proximal to the distal eminence of the proximal phalanx. The needle is angled 45 to 60 degrees distally and slightly medially (Fig. 4-27). Then 5 mL of 2% local anesthetic is injected into the joint.

DESENSITIZING THE FOOT, PASTERN, AND SESAMOIDS: ABAXIAL BLOCK

The abaxial block desensitizes structures distal to the fetlock joint and varying proportions of the

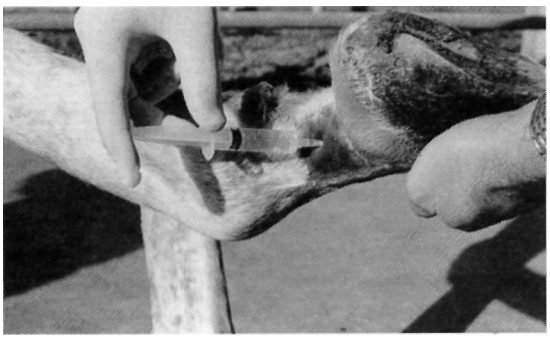


Figure 4-26. Palmar approach to the proximal interphalangeal joint. With the joint held partially flexed, a 20-gauge, 30-mm (1.5-inch) needle is inserted dorsal to the neurovascular bundle in the "V" formed by the proximal phalanx and the lateral branch of the superficial digital flexor tendon and directed distal and medial at an angle of 45 degrees. A volume of 5 mL of 2% local anesthetic is injected.



Figure 4-27. Dorsal approach to the proximal interphalangeal joint. The limb is weight bearing, and a 21-gauge, 25-mm (1-inch) needle is inserted lateral to the common digital extensor tendon 10 mm proximal to the distal eminence of the proximal phalanx. The needle is angled 45 to 60 degrees distally and slightly medially and 5 mL of 2% local anesthetic is injected into the joint.

sesamoid bones. With the limb held up, the palmar vessels can be palpated over the abaxial aspect of the sesamoid bones. The palmar nerves run palmar to these vessels and can be desensitized by inserting a 23- or 25-gauge, 25-mm (1-inch) needle subcutaneously as shown in Figure 4-28. The needle is inserted at the base of the sesamoid bones. Approximately 3 to 4 mL of 2% local anesthetic is injected. This nerve block will not desensitize the dorsal aspect of the fetlock. The block can be assessed by using a ballpoint pen or similar object to prick the dorsal aspect of the pastern. While this block is probably the easiest to perform of all nerve blocks, its nonspecific nature often makes it difficult to localize the problem when there is improvement in gait.

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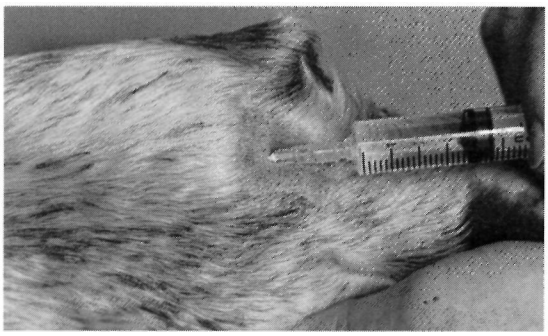


Figure 4-28. Palmar (abaxial) nerve block. The 23-gauge, 25-mm (1-inch) needle is inserted just palmar to the vessels over the abaxial surface of the sesamoid bones. The photograph shows the needle position for the lateral abaxial block in the left foreleg. A volume of 3 mL of 2% local anesthetic is injected.

DESENSITIZING THE FETLOCK: PALMAR METACARPAL AND PALMAR NERVE (LOW FOUR-POINT) BLOCK

This block desensitizes the fetlock joint, sesamoid bones, and all structures distal to this. The palmar nerves are blocked by placing a 23-gauge needle subcutaneously in the groove between the deep digital flexor tendon and the suspensory ligament at the level of the button of the splint bones. Then 2 mL of 2% local anesthetic are injected at this site. The palmar metacarpal nerves emerge from under the distal ends of the second and fourth metacarpal bones (splint bones) and can be desensitized by injecting 1 to 2 mL of 2% local anesthetic under the distal ends or "buttons" of the second and fourth metacarpal bones (Fig. 4-29). If an abaxial nerve block has been performed already, adding the palmar metacarpal nerve block will achieve a similar effect to the "four-point" block.



Figure 4-29. Palmar metacarpal nerve block. A 23-gauge, 15-mm (5/8-inch) needle is positioned underneath the distal end ("button") of the fourth metacarpal bone in the left foreleg. A volume of 2 to 3 mL of 2% local anesthetic is injected.

DESENSITIZING THE FETLOCK JOINT INTRA-ARTICULARLY

If primary fetlock joint pathology is thought to be the cause of lameness, the fetlock joint can be desensitized by injection of 8 to 10 mL of *2%* local anesthetic directly into the joint. A 21-gauge, 25-mm (1-inch) needle is used for this block, which can be performed through one of three sites. The principal site used by most veterinarians is the palmar pouch (Fig. 4-30), particularly if it is distended with synovial fluid. It is easier to gain access to this site if the leg is held off the ground. Alternatively, a more distal site on the lateral aspect of the fetlock joint with the needle inserted through the lateral collateral sesamoidean ligament may be used for fetlock arthrocentesis (Fig. 4-31). With the horse's leg in flexion, a 21-gauge, 25-mm (1-inch) needle is inserted in the groove between the sesamoid bone and the palmar aspect of the third metacarpal bone. Advantages include less hemorrhage into the joint and more definite appearance of synovial fluid after needle insertion. An alternative site is the dorsal aspect of the fetlock joint, between the common and lateral extensor tendons (Fig. 4-32). With injection at this site there is potential for the point of the needle to traumatize the articular cartilage.

DESENSITIZING FROM SUBCARPAL AREA DISTALLY: HIGH PALMAR NERVE BLOCK

The palmar nerves are blocked in the proximal metacarpal area by placing a 23-gauge, 25-mm (1-inch) needle on the dorsal aspect of the deep digital flexor tendon medially and laterally and



Figure 4-30. Position of a 21-gauge, 25-mm Cl-inch) needle in the palmar pouch, between the third metacarpal bone and suspensory ligament of the left foreleg, to desensitize the fetlock joint. It is sometimes easier to obtain synovial fluid when the needle is inserted with the joint flexed. To desensitize the joint, 10 mL of 2% local anesthetic is injected.

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Figure 4-31. Position of a 21-gauge, 25-mm Cl-inch) needle through the lateral collateral sesamoidean ligament to desensitize the left fetlock joint. This site is the most reliable for obtaining joint fluid and causes the least hemorrhage. The landmarks are most easily located with the joint held in slight flexion. The needle has to be placed more deeply than the other two sites for fetlock joint arthrocentesis. To desensitize the joint, 10 mL of 2% local anesthetic is injected.

through the flexor retinaculum. The palmar metacarpal nerves run on the palmar aspect of the third metacarpal bone axial to the second and fourth metacarpal bones. A 21-gauge, 25-mm (1-inch) needle is inserted through the skin and directed dorsally to strike the palmar cortex of the third metacarpal bone. Then 2 mL of local anesthetic is injected (Fig. 4-33). It is important to understand the risk of entering the distal palmar pouches of the distal intercarpal (carpometacarpal) joint with this technique and therefore desensitizing the distal carpal joints. When properly performed, this



Figure 4-32. Position of a 21-gauge, 25-mm (1-inch) needle in the dorsal aspect of the left fetlock joint to desensitize the joint. To desensitize the joint, 10 mL of 2% local anesthetic is injected.

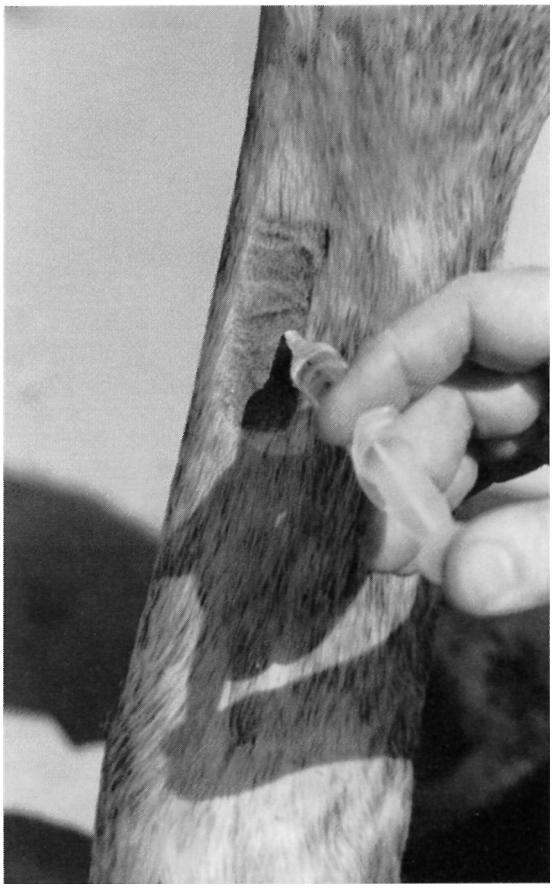


Figure 4-33. High palmar nerve block showing the position of a 21-gauge, 36-mm (1.5-inch) needle underneath the deep flexor tendon to block the lateral palmar nerve of the right foreleg. Note that the needle is located just beneath the skin over the dorsal edge of the deep flexor tendon and, to block the medial nerve, the needle is advanced until the tip can be felt subcutaneously on the medial side and then withdrawn slightly. Care should be taken not to enter the medial palmar artery. A volume of 3 mL of 2% local anesthetic is injected at each site.

block will desensitize the interosseous (suspensory ligament), flexor tendons, and splint bones.

To avoid entering the joint, an alternative method exists whereby the nerves are blocked by injection of local anesthetic dorsal to the heads of the second and fourth metacarpal bones. Anesthesia of the lateral branch is achieved using a 21-gauge, 37.5-mm (1.5-inch) needle, inserted midway between the distal edge of the accessory carpal bone and the most proximal portion of the fourth metacarpal bone. With the needle directed both proximally and distally, 5 mL of 2% local anesthetic is injected in several directions (Fig. 4-34). For the medial nerve, the nerve is blocked

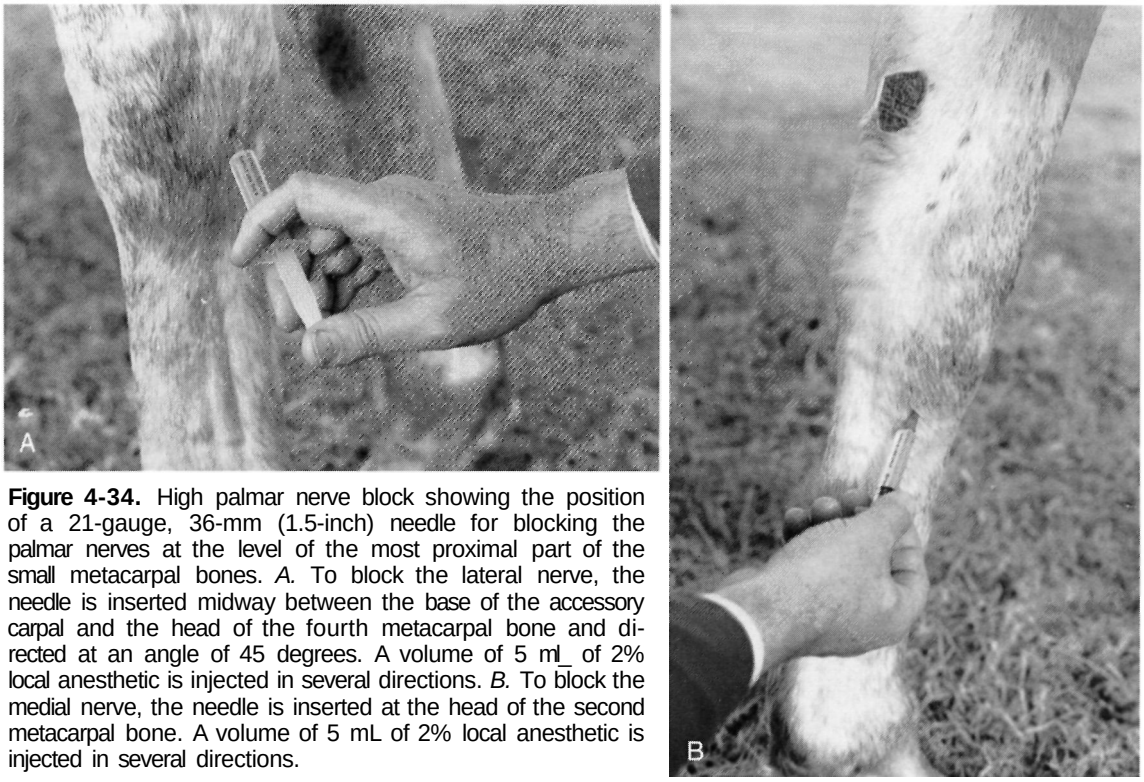


Figure 4-34. High palmar nerve block showing the position of a 21-gauge, 36-mm (1.5-inch) needle for blocking the palmar nerves at the level of the most proximal part of the small metacarpal bones. *A.* To block the lateral nerve, the needle is inserted midway between the base of the accessory carpal and the head of the fourth metacarpal bone and directed at an angle of 45 degrees. A volume of 5 mL of 2% local anesthetic is injected in several directions. *B.* To block the medial nerve, the needle is inserted at the head of the second metacarpal bone. A volume of 5 mL of 2% local anesthetic is injected in several directions.

at the head of the second metacarpal bone, with 5 mL of 2% local anesthetic injected.

DESENSITIZATION OF THE CARPUS

The most common carpal joint involved in injury is the midcarpal joint. This does not communicate with the antebrachiocarpal (radiocarpal) joint, which must be desensitized separately. However, the middle carpal joint does communicate with the distal carpal (carpometacarpal) joint, and this should be taken into account when evaluating a positive response to a nerve block. There is also the potential for local anesthetic to diffuse into the origin of the suspensory ligament due to the close apposition of the palmar distal pouches of the carpometacarpal joint. Both the proximal and middle carpal joints are easily blocked by holding the affected leg off the ground so that the carpus is well flexed. A 21-gauge, 25-mm (1-inch) needle is placed into the middle or proximal carpal joints, either medial or lateral to the extensor carpi radialis tendon (Figs. 4-35 and 4-36) in the depressions formed by the openings of the joints. Then 10 mL of 2% local anesthetic is injected into the affected joint. At least 10 minutes should elapse before re-evaluating the horse's gait.

DESENSITIZING FROM THE DISTAL RADIUS TO THE FOOT

To desensitize the distal radius (and all structures distally), it is possible to block the median, ulnar, and musculocutaneous nerves. While these nerve blocks are seldom necessary in practice, they are sometimes useful if the veterinarian wants to ensure that the problem is proximal to the carpus



Figure 4-35. Position of a 21-gauge, 25-mm (1-inch) needle in the left middle carpal (intercarpal) joint to desensitize the joint. A volume of 10 mL of 2% local anesthetic is injected.



Figure 4-36. Position of a 21-gauge, 25-mm (1-inch) needle in the left proximal carpal (radiocarpal) joint to desensitize the joint. A volume of 10 mL of 2% local anesthetic is injected.

by allowing complete desensitization of the distal limb. The nerve blocks are illustrated in Figures 4-37 to 4-39. Between 10 and 20 mL of local anesthetic is injected at each site.

DESENSITIZING THE ELBOW JOINT

Lameness arising from the elbow joint is uncommon, but when it does occur, it is almost impossible to diagnose by physical examination because there is seldom pain on palpation and synovial effusion may not be detectable. Arthrocentesis of the elbow joint is achieved by palpating the lateral collateral ligament and determining its point of insertion on the lateral radius. A notch can then be felt immediately caudal to the ligament, and a 21-gauge, 25-mm (1-inch) needle is placed into this notch to a depth of approximately 12 mm (0.5 inch) (Fig. 4-40). A total of 15 to 20 mL of 2% local anesthetic is injected.

A proximocaudal approach has also been described. A line is drawn between the lateral epicondyle of the humerus and the most proximal palpable point of the ulnar, and an 18-gauge, 3.5-inch needle inserted one third of the way along and 10 mm proximal, angling down at 45 degrees.

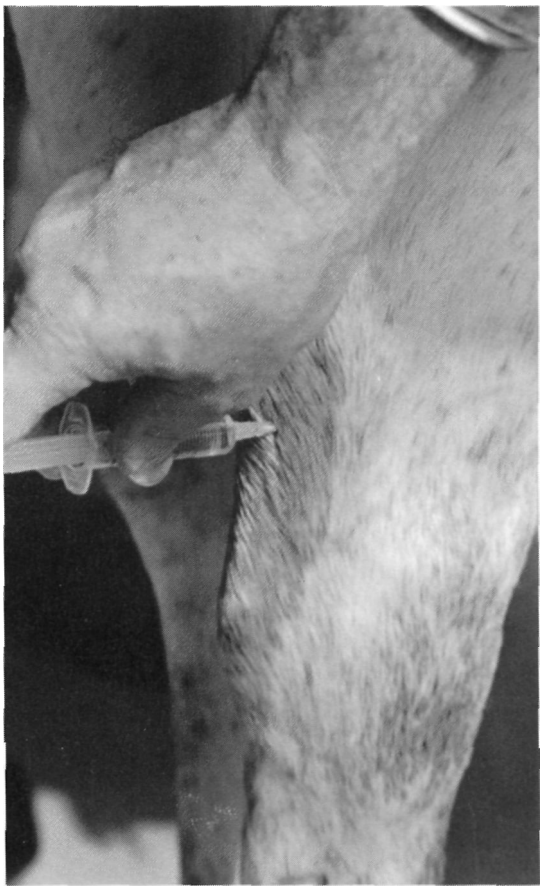


Figure 4-38. Ulnar nerve block. A 21-gauge, 25-mm (1-inch) needle is inserted 8 to 10 cm above the accessory carpal bone in the groove between the ulnaris lateralis and the flexor carpi ulnaris muscles. A volume of 10 mL of 2% local anesthetic is injected.



Figure 4-37. Median nerve block. A 19-gauge, 36-mm (1.5-inch) needle is inserted underneath the belly of the pectoralis descendens muscle in a proximal direction and just caudal to the radius. Because the median artery lies close to this site, it is possible to penetrate the vessel. Therefore, if blood drips from the needle, it should be directed more caudally. A volume of 10 mL of 2% local anesthetic is injected

DESENSITIZING THE SHOULDER JOINT

As with the elbow joint, horses with lameness arising from the shoulder joint seldom show pain on palpation or flexion. The site for needle insertion for intra-articular local anesthesia is determined by palpating the lateral tuberosities of the

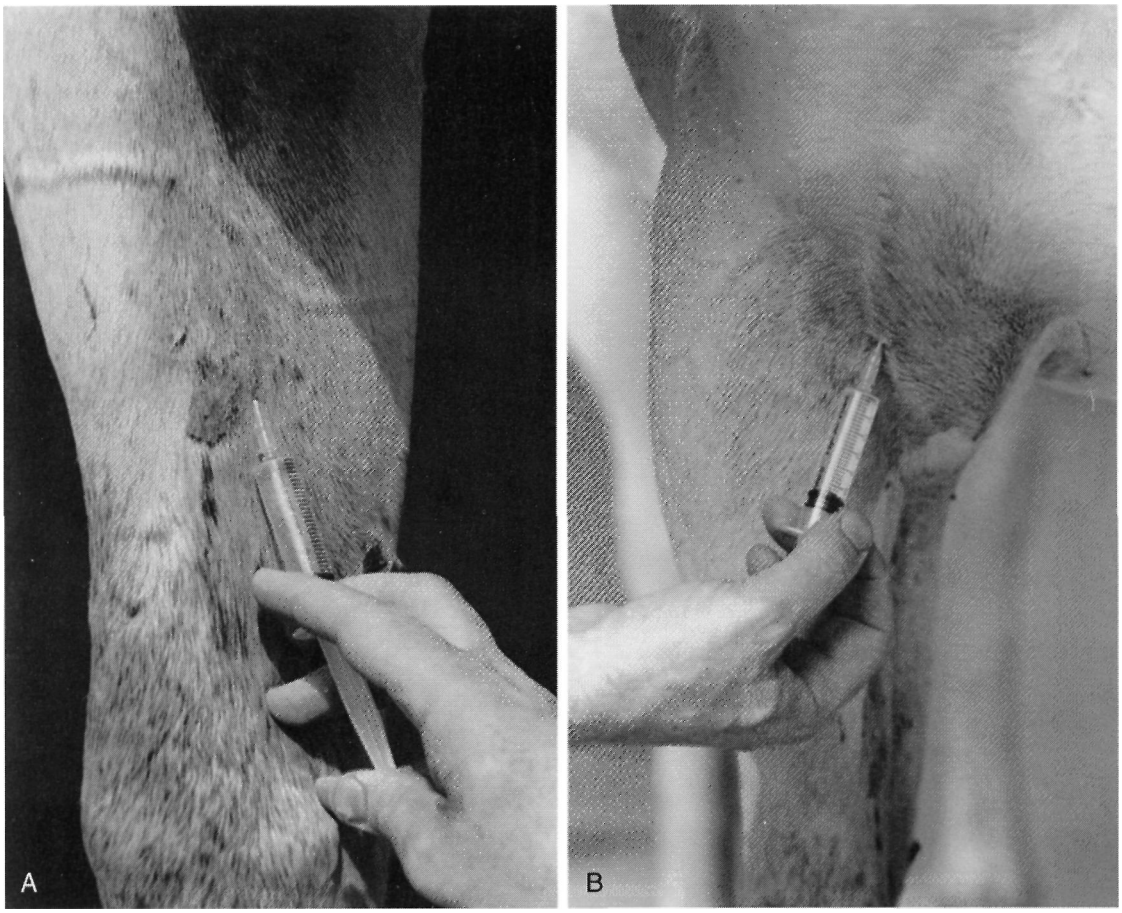


Figure 4-39. Musculocutaneous nerve block. Two injection sites are necessary to desensitize the musculocutaneous nerve. *A.* One site is on the medial aspect of the midradius, close to the cephalic vein. The nerve can be palpated and a 21-gauge, 25-mm needle used to inject 5 mL of local anesthetic. *B.* The more proximal site is over the lacertus fibrosus. A 21-gauge, 25-mm needle is inserted where the nerve can be palpated, and 5 mL of 2% local anesthetic is injected.

humerus with the thumb and forefinger (Fig. 4-41). After desensitizing the skin with a bleb of local anesthetic, an 18-gauge, 7.5-cm (3-inch) spinal needle is passed in the groove between the tuberosities, along the humeral head (Fig. 4—42). When the point of the needle enters the joint, a slight sucking sound is usually heard, and aspiration with a 10-mL syringe produces synovial fluid. Approximately 20 mL of 2% local anesthetic is injected to desensitize the joint.

Hindlimb

The regimen for local anesthesia in the hindlimb is similar to that for the forelimb, from the foot proximally to below the hock. However, the nerve distribution is slightly more variable than that of the forelimb. When blocking the fetlock region, the dorsal metatarsal nerve should also be blocked, along with the plantar metatarsal nerves and planter nerves. After depositing local anesthetic at the base of the button of the splint bone, the needle is partially withdrawn and then passed dorsally and a further 2 to 3 mL of local anesthetic is deposited on the lateral aspect of the third metatarsal bone. Distal limb nerve blocks are also more difficult in the hindlimb because the leg must be held off the ground by the veterinarian while carrying out the blocks and the risk of injury to the operator is potentially greater.

TARSUS (HOCK)

The hock is a complex joint, with four possible sites for intra-articular pathology: tarsocrural (tibiotarsal), talocalcaneocentroquatral (proximal intertarsal), centrodistal (distal intertarsal), and tarsometatarsal joints. The tarsocrural and talocalca-

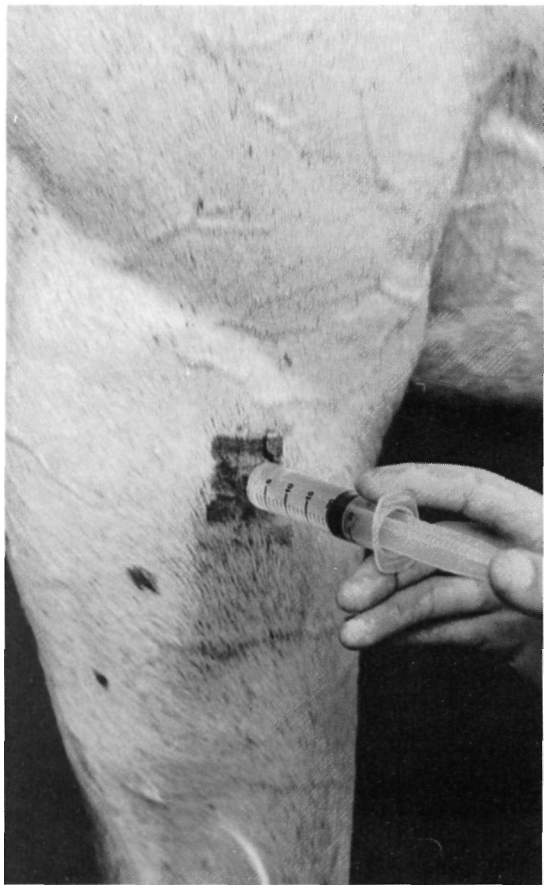


Figure 4-40. Position of a 19-gauge, 36-mm (1.5-inch) needle in the left elbow joint to desensitize the joint.

neocentroquatral joints communicate, whereas the centrodistal and tarsometatarsal joints have been reported to communicate in 8 to 38% of cases. Arthritis of the hock (bone spavin) usually is confined to the tarsometatarsal and centrodistal joints, and therefore, these joints are blocked first.

DESENSITIZING THE TARSOMETATARSAL JOINT

Arthrocentesis of the tarsometatarsal joint is undertaken over the head of the fourth metatarsal bone on the plantarolateral surface of the distal hock. A 21-gauge, 25-mm (1-inch) needle is inserted to a depth of 12 to 18 mm (0.5-0.75 inch) (Fig. 4-43). In a difficult horse, it may be helpful to insert a bleb of local anesthetic in the skin before the needle is inserted into the joint. If synovial fluid does not appear in the hub of the needle, it should be rotated, after which synovial fluid should emerge.

DESENSITIZING THE CENTRODISTAL (DISTAL INTERTARSAL) JOINT

The centrodistal joint can be desensitized on the medial aspect of the hock, 10 to 12 mm (0.5 inch) above the head of the second metatarsal bone and slightly farther dorsally on the distal border of the cunean tendon. Usually, it is difficult to palpate the joint space, whereas the space of the talocalcaneocentroquatral (proximal intertarsal) joint above it is more readily discernible. A 23-gauge, 25-mm (1-inch) needle is introduced at a right angle to the long axis of the leg and directed slightly plantar (Fig. 4-44), and 2 mL of 2% local anesthetic is injected.

DESENSITIZING THE TARSOCRURAL (TIBIOTARSAL) AND TALOCALCANEOCENTROQUATRAL (PROXIMAL INTERTARSAL) JOINTS

The tarsocrural joint and talocalcaneocentroquatral (proximal intertarsal) joint can both be desensi-



Figure 4-41. Desensitization of the shoulder joint. Deposition of local anesthetic to desensitize the skin before insertion of the spinal needle.

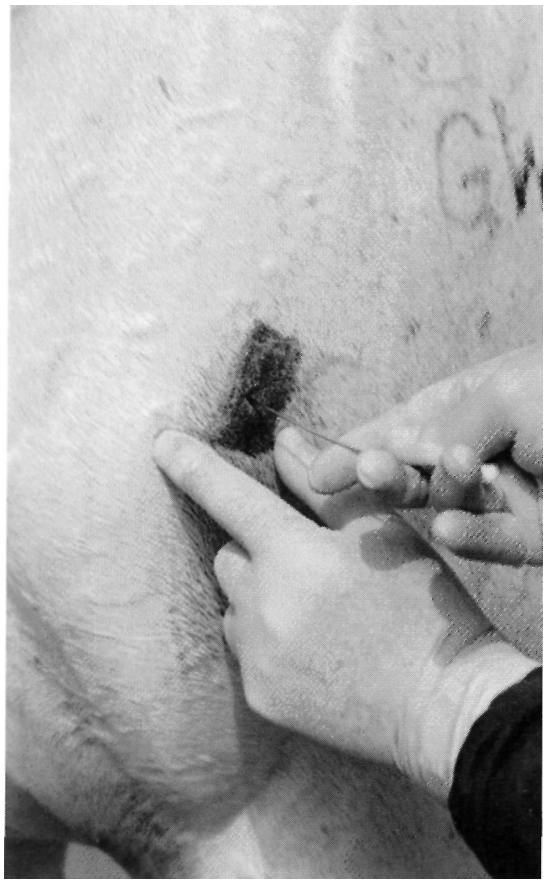


Figure 4-42. An 18-gauge, 125 mm (5-inch) spinal needle inserted along the humeral head into the left shoulder joint.

tized by introducing local anesthetic into the talocrural joint on the medial aspect of the hock. After palpation of the medial malleolus of the distal tibia, a 21-gauge, 25-mm (1-inch) needle is inserted just distal to it and either dorsal or plantar to the saphenous vein to a depth of 12 mm (0.5 inch) (Fig. 4-45). A total of 8 to 10 mL of 2% local anesthetic is injected to achieve desensitization.

DESENSITIZING FROM THE DISTAL TIBIA TO THE FOOT

Desensitization of the peroneal and tibial nerves is a useful technique for excluding lower limb lamenesses. The tibial nerve is blocked on the medial aspect of the hindleg, approximately 8 cm (3 inches) above the point of the hock, on the caudal border of the deep flexor tendon (Fig. 4—46). To block the left tibial nerve, the operator should stand on the lateral side of the right hindleg and have an assistant hold up the left foreleg. After a bleb of local anesthetic has been placed in the skin, a 19-gauge, 25-mm (1-inch) needle is inserted under and along the edge of the deep flexor tendon. A total of 20 mL of 2% local anesthetic is deposited in several locations by moving the needle.

The peroneal nerves are located in the groove between the lateral and long digital extensor muscles on the lateral aspect of the tibia approximately 10 to 12 cm (4-5 inches) above the point of the hock (Fig. 4-47). A 19-gauge, 1.5-inch needle is passed 20 to 30 mm deep, and 15 mL of local anesthetic is injected. As the needle is removed, a further 5 mL is injected to desensitize the superficial peroneal nerve.

DESENSITIZING THE STIFLE

There are three compartments of the stifle joint: the femoropatellar joint and the lateral and medial

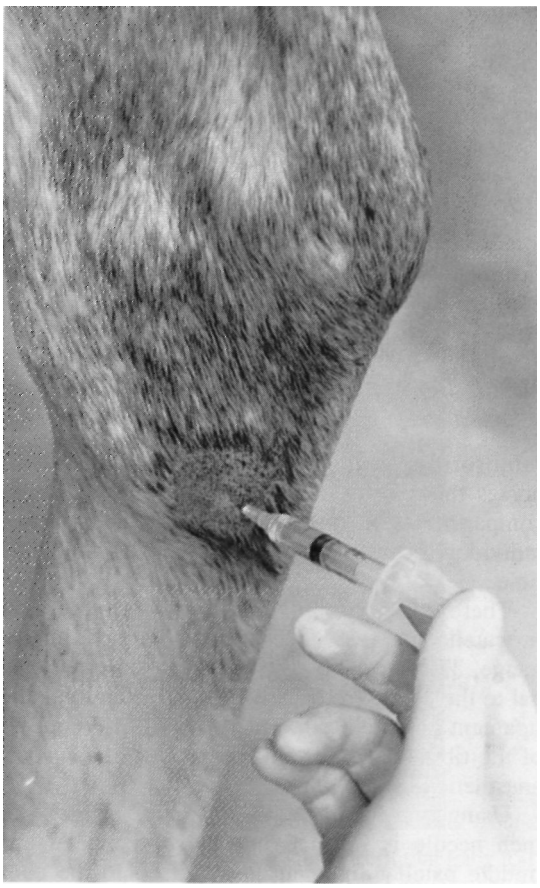


Figure 4-43. Position of a 21-gauge, 25-mm Cl-inch) needle in the left tarsometatarsal joint to desensitize the joint. The needle is inserted at an angle over the head of the fourth metatarsal bone. A volume of 5 mL of 2% local anesthetic is injected.

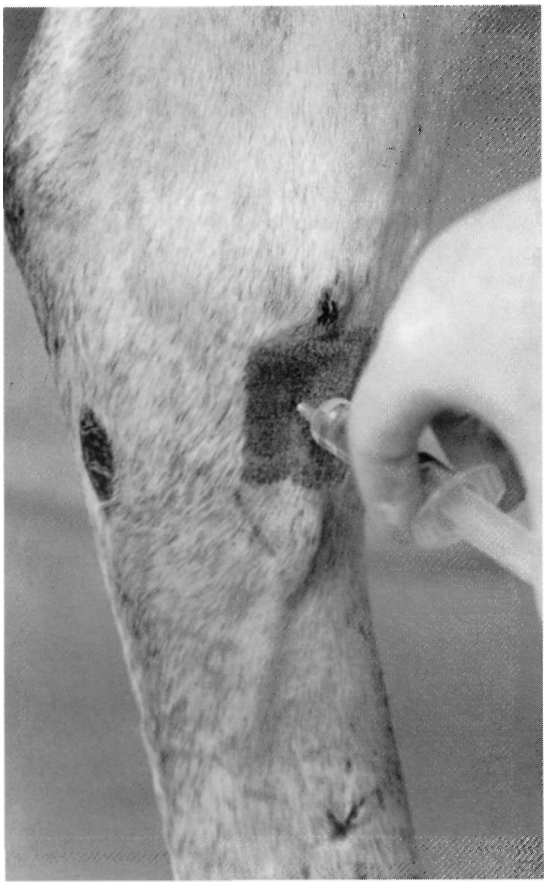


Figure 4-44. Position of a 23-gauge, 15-mm (5/8-inch) needle on the medial aspect of the left distal centrodistal (intertarsal) joint on the medial aspect of the hock. A volume of 2 to 3 mL of 2% local anesthetic is injected.

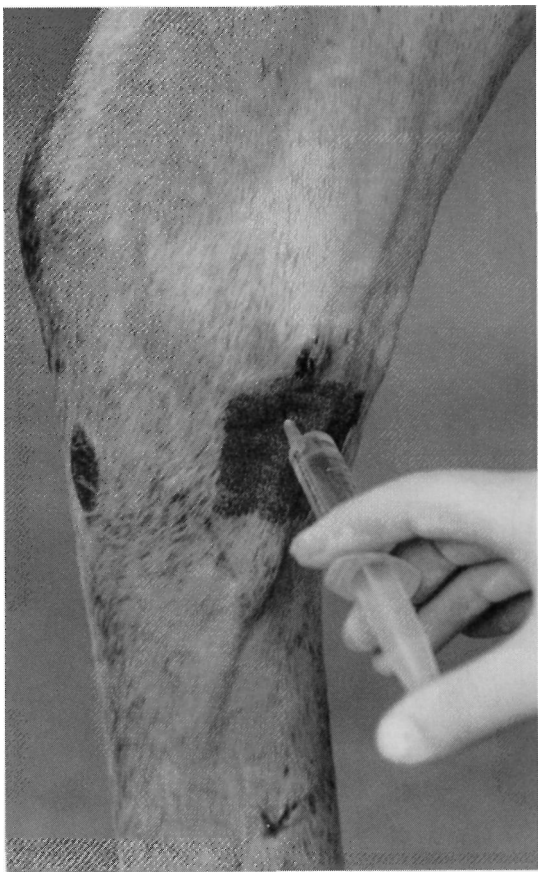


Figure 4-45. Position of a 21-gauge, 25-mm Cl-inch) needle in the left tarsocrural (tibiotarsal) joint just distal to the medial malleolus of the tibia. Note the position of the needle on the medial aspect of the leg, just plantar to the saphenous vein.

femorotibial joints. In approximately 20% of horses there is no communication between these compartments; therefore, all should be blocked individually but for speed are done at the same time.

There are two common approaches to the femoropatellar joint. For the lateral approach, an 18-gauge, 37.5-mm (1.5-inch) needle is inserted caudal to the lateral trochlear ridge and lateral patellar ligament and 5 cm proximal to the lateral condyle of the tibia (Fig. 4-48). Then 30 mL of 2% local anesthetic is injected.

Using the dorsal approach, an 18-gauge, 1.5-inch needle is inserted between the medial and middle patellar ligament 2 cm distal to the apex of the patella and passed proximally into the intertrochlear groove (Fig. 4-49); 30 mL of 2% local anesthetic is injected. Synovial fluid may not be obtained.

For the medial femorotibial joint, an 18-gauge,

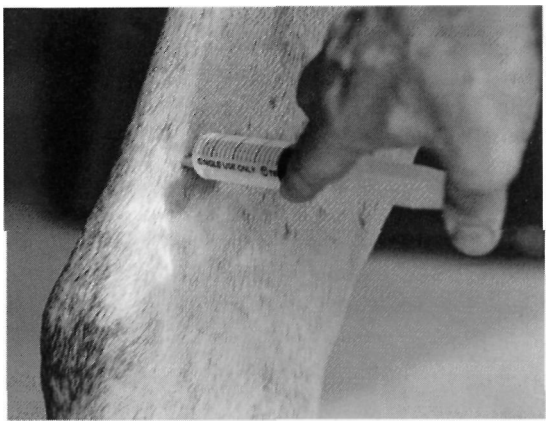


Figure 4-46. Position of 19-gauge, 25-mm needle on the medial aspect of the hock to block the tibial nerve, just caudal to the deep flexor tendon. Approximately 20 mL of 2% local anesthetic is deposited in several sites.

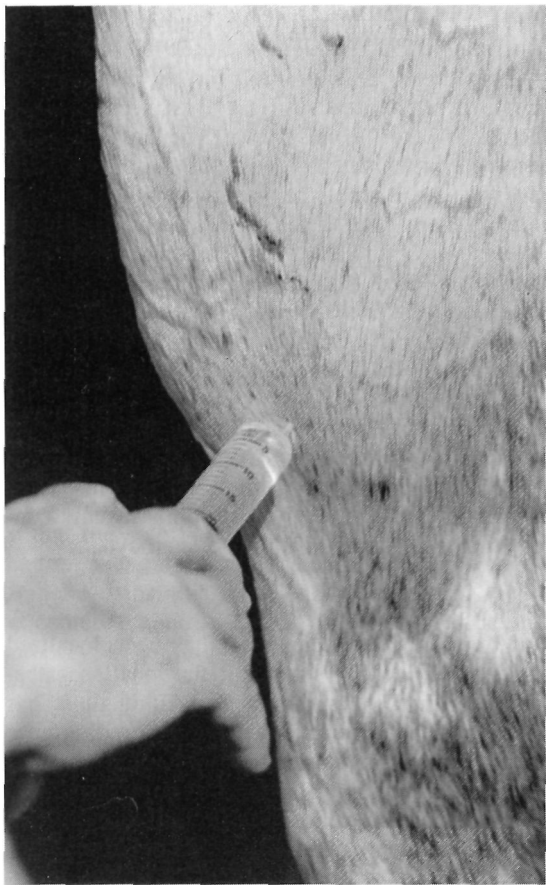


Figure 4-47. Position of 19-gauge, 37.5-mm needle on the lateral aspect of the distal tibia to block the peroneal nerves. The needle is located about 10 cm above the point of the hock in the groove formed between the lateral and long extensor muscles. Local anesthetic is injected at a depth of 3 cm to block the deep peroneal insertions and withdrawn superficially to block the superficial peroneal insertions.

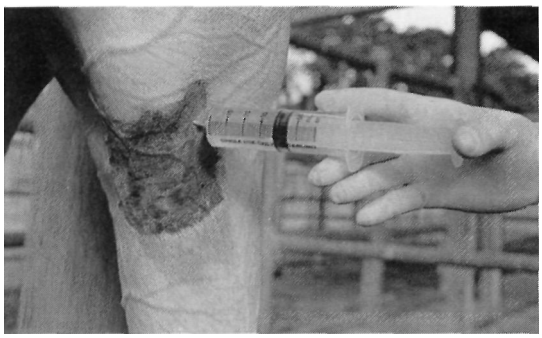


Figure 4-48. To desensitize the lateral femorotibial joint, an 18-gauge, 37.5-mm (1.5-inch) needle is inserted caudal to the lateral trochlear ridge and lateral patellar ligament, 5 cm proximal to the lateral condyle of the tibia.

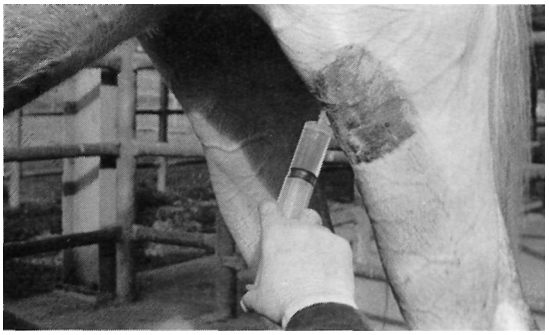


Figure 4-49. To enter the femoropatellar pouch, an 18-gauge, 37.5-mm (1.5-inch) needle is inserted between the medial and middle patellar ligaments, 2 cm distal to the apex of the patella and passed proximally into the intertrochlear groove.

37.5-mm (1.5-inch) needle is inserted between the medial patellar ligament and the medial collateral ligament 10 mm above the tibial plateau. The needle is directed proximocaudally to contact the articular surface of the medial femoral condyle (Fig. 4-50). Then 20 mL of 2% local anesthetic is injected.

Two approaches are possible for the lateral femorotibial joint. A 19-gauge, 37.5-mm (1.5-inch) needle can be inserted just cranial to the lateral collateral ligament as for the medial femo-

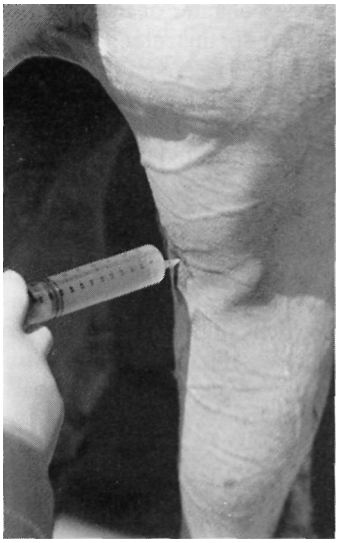


Figure 4-50. For the medial femorotibial joint to be entered, an 18-gauge, 37.5-mm (1.5-inch) needle is inserted between the medial patellar ligament and the medial collateral ligament 10 mm above the tibial plateau. The needle is directed proximocaudally to contact the articular surface of the medial femoral condyle.

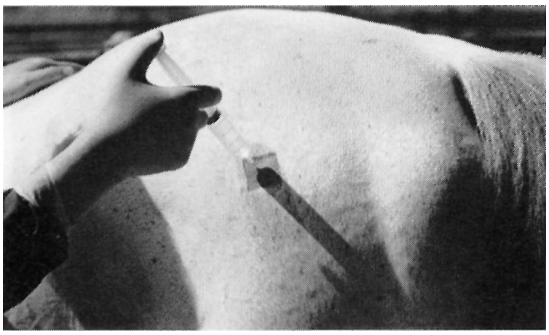


Figure 4-51. Desensitization of the hip joint. Position of an 18-gauge, 150-mm (6-inch) spinal needle in the hip joint.

rotibial joint. Alternatively, an 18-gauge, 90-mm (3.5-inch) needle is placed through the tendon of origin of the long digital extensor tendon 30 mm distal to the tibial plateau to reach the distal pouch of the lateral femorotibial joint. Local anesthetic is injected when the needle contacts the tibia.

DESENSITIZING THE HIP

Because of its depth and muscle covering, the hip is probably the most difficult joint to access. Fortunately, hip problems in horses are extremely rare, and the hip joint seldom requires desensitization. The joint is desensitized by using an 18-gauge, 175-mm (7-inch) spinal needle. After locating the greater trochanter of the femur, the needle is passed cranial to this along the femoral neck and head (Fig. 4-51). Aspiration with a 10-mL syringe will indicate entry to the joint, which is 10 to 13 cm (4-5 inches) deep, and 20 mL of 2% local anesthetic is injected.

Synovial Fluid Analysis

Synovial fluid analysis is useful in distinguishing septic from nonseptic forms of joint disease. Although cytologic analyses have been advocated by some veterinarians to assist in determining the stage of degenerative joint disease, this is rarely useful.

KEY POINT

There is no correlation between synovial fluid cytologic findings and the severity of degenerative joint disease.

Rather, clinical and radiographic findings are of more use in establishing prognosis and treatment of joint disorders. If synovial fluid is to be collected, it should be obtained as previously described, and portions should be placed in a tube containing EDTA and in a sterile plain tube. If samples are to be submitted for bacteriology because of suspected septic arthritis, great care should be taken to ensure that the fluid does not become contaminated during collection. It may be necessary to collect the samples with the horse under general anesthesia, particularly if there is severe pain associated with manipulation of the joint. The fluid sample in the sterile tube can be submitted for bacteriology (see Chapter 17). Worthwhile analyses include total and differential white cell count and total protein. Normal values for most joints are as shown in Table 4-1.

In septic arthritis, total protein is increased to more than 30 g/L (3 g/dL), and the total white cell count increases to a value usually in excess of 10,000 X 10^6/L, with neutrophils usually contributing more than 90% of the leukocytes.

Radiography

Once the site of the lameness has been localized with nerve blocks or intra-articular local anaesthesia, radiographs can be taken. Although it has become common for clients to request radiographs of all four limbs, from the foot to the carpus or tarsus, interpretation of the changes found is often difficult. For this reason, it is best to perform a thorough clinical examination followed by radiography only of areas that are of concern. Several radiographic views are usually required, with lateral to medial and oblique views being the most rewarding. Although there are a number of excellent books indicating radiographic positioning and interpretation, we provide some perspective and practical tips for each of the areas that are commonly radiographed.

FOOT

Many veterinarians attempt to examine the navicular bone and distal phalanx (pedal bone) on the

TABLE 4-1. Normal Values for Synovial Fluid Analyses

Measurement	Value
Total protein (g/L)	10-20 (1-2 g/dL)
White cell count ($x10^6$/L)	100-800 (100-800/μL)
Differential count	
Neutrophils	<20%
Mononuclear cells	80-95%
Alkaline phosphatase (U/L)	20-40

same radiograph, but the exposure necessary to properly examine the navicular bone will result in overexposure of the pedal bone on the dorsopalmar view.

KEY POINT

Separate exposures must be made of the distal phalanx (pedal) and navicular bones depending on the suspected site of the problem.

The sulci of the frog should be cleaned and packed with Play-Doh to eliminate the possibility of radiolucent lines over the distal phalanx and navicular bone. The dorsopalmar view can be taken in either of two ways. The first is the *upright pedal view,* where the foot is positioned in a block so that the sole is at 30 degrees to the plate and the foot rests on the toe. To radiograph the navicular bone using this view, the x-ray beam should be centered about 12 mm (0.5 inch) above the coronary band and coned down to reduce the scatter. The second is the *high coronary view,* where the foot is positioned in a normal weight-bearing stance on a reinforced cassette. This view provides a satisfactory view of the pedal bone but causes magnification of the navicular bone, making assessment of minor changes difficult. *Oblique views* are of particular use when evaluating horses with fractures of the pedal bone. The normal lines of the frog sulci, together with the direction of the fracture line, often result in a fracture being unapparent. The appropriate oblique view enables a fracture line to be seen more easily and potential involvement of the articular surface to be evaluated. The *special navicular or palmar (skyline) view* enables the wings of the pedal bone and the flexor surface of the navicular bone to be assessed. This view is particularly useful in cases of suspected fracture of the navicular bone. The *lateral view* of the foot enables the orientation of the pedal bone in the foot to be evaluated, which is essential when assessing possible pedal bone rotation in cases of laminitis. It is useful to stick a short piece of wire longitudinally on the middorsal hoof wall to aid this evaluation. This view should be taken with the foot on a wooden block around 25 to 50 mm (1-2 inches) high so that the solar aspect of the pedal bone can be assessed.

PASTERN AND FETLOCK

The four standard views (dorsopalmar, lateral, dorsomedial to palmarolateral, dorsolateral to palmaromedial) should be used when assessing problems involving the pastern or fetlock.

KEY POINT

A lateral view of the fetlock is particularly important to evaluate the dorsal aspect of the distal third metacarpal bone.

This site is important when determining changes to the dorsal contour of the distal metacarpus, which occur when there is chronic proliferative synovitis (villonodular synovitis). If this condition is suspected, ultrasound of the dorsal aspect of the fetlock may be useful to confirm it or contrast radiography using 30 mL of air injected into the joint.

METACARPUS AND METATARSUS

Oblique views are very important to evaluate the second and fourth metacarpal bones if fractures are suspected. Lateral views of the metacarpus or metatarsus with good detail are important if fissure fractures of the dorsal cortex are suspected.

CARPUS

Minimum radiographic views include a flexed lateral, dorsolateral palmaromedial, and dorsomedial palmarolateral obliques and a skyline of the third carpal bone. Because most midcarpal joint problems occur on the dorsomedial side of the joint, and most antebrachiocarpal joint problems occur on the dorsolateral aspect, both oblique views are necessary. On the flexed lateral view, the dorsal aspect of the intermediate carpal bone sits more proximal than that of the radial carpal bone.

KEY POINT

Some undisplaced chip and slab fractures and sagittal fractures of the third carpal bone will only be visible on a skyline view.

The *flexed lateral view* is useful for assessment of the articular surfaces of the distal radial and proximal third carpal bones.

ELBOW AND SHOULDER

For adequate radiographs of both of these areas, the horse is best anesthetized, although standing shots are possible. Although x-ray machines with capacities for high (about 300 mA) output are optimal for radiographing the elbow and shoulder, it is possible to obtain adequate exposures with portable x-ray machines. This is achieved by using repeated exposures with the same plate in position.

HOCK

Four views, dorsoplantar, lateral, and two obliques, are necessary for full evaluation of hock disorders.

KEY POINT

Because most hock problems involve the distal joints, the primary beam should be centered at the level of the chestnut.

Although many horses with bone spavin have lesions on the dorsomedial aspects of the tarsometatarsal and intertarsal joints, the dorsomedial to plantarolateral oblique view, which highlights lesions on the dorsolateral aspect of the hock, often demonstrates significant changes that may not be apparent on the other views. Therefore, this view should always be included when radiographing the hock for suspected bone spavin. Note that radiographic changes involving the hock can be present without signs of lameness.

STIFLE

Radiographs of the stifle are difficult to take with the portable x-ray machines that are used to radiograph the distal limbs. This is particularly so for the craniocaudal view of the stifle. However, because one of the most common lesions involving the stifle is osteochondritis dissecans of the lateral trochlear ridge of the femur, a lateral radiographic view, often possible with a portable machine, may be useful. Also, cystic lesions of the medial condyle of the femur can often be observed on a flexed lateral view of the stifle.

HIP

By positioning the x-ray machine under the horse's hindleg, standing radiographs of the hip and pelvis are possible. The x-ray tube is positioned underneath the abdomen and just cranial to the hindleg. The head of the x-ray machine is angled (35 degrees from the vertical) so that the beam is directed slightly caudally and dorsally. The cassette and grid are positioned above the gluteal muscle mass and angled to the natural slope of the croup. To obtain a radiograph of the ilium, the same technique is used, with a less acute angle of the tube.

Ultrasound

Ultrasound is an important aid for assessing the prognosis of tendon and ligament injuries and imaging superficial soft tissue structures of joints. Monitoring the progress of tendon and ligament lesions via repeated ultrasound examinations allows determination of the rate of healing of the injury and the time at which the horse can be returned to training. Although ultrasound waves cannot penetrate bone, the surface contour can be imaged, and this has proved useful for detecting fractures in bones in which radiography without anesthesia is difficult, such as the pelvis. Periosteal new bone and enthesiophytes in soft tissue attachments can also be detected.

Both linear array and sector scanners have been used, with the 7.5-MHz probe being the most commonly used. An in-built or separate standoff is used for superficial structures. A 5-MHz probe will give deeper penetration and is often used to evaluate the pelvis.

For best detail, the hair is clipped with a number 40 blade. The area should be cleaned and alcohol applied to defat the skin. Ultrasound coupling gel is then liberally applied.

The technique requires practice because artifacts easily can be created. Comparison with the opposite limb is often helpful. Lesions in dense collagenous studies such as tendon will appear as focal hypoechoic (black) areas or a more diffuse loss of echogenicity. On longitudinal scans, loss of fiber alignment will be evident.

Nuclear Scintigraphy

Nuclear scintigraphy has become an indispensable tool in equine orthopedics. The image is produced by detection of gamma rays emitted by a radiopharmaceutical administered to the subject. The most commonly used radiopharmaceutical is technetium-99m-labeled methylene diphosphonate (MDP). There are three imaging phases: the flow phase, the pool phase (soft-tissue phase), and the bone phase. The bone phase is most often used in equine orthopedics, and increased uptake indicates increased blood flow and bone turnover. Because of this, it is more sensitive than radiography to bony pathology, which requires structural changes.

Indications for nuclear scintigraphy include horses with acute lameness where there are no radiographic changes, where local anesthesia fails to localize the lameness, evaluation of areas that are difficult to radiograph in the standing horse, horses that respond to intra-articular anesthesia but have negative radiographic findings, and horses where multifocal lameness is suspected. If local anesthesia has been performed, scintigraphy should be delayed at least 1 week because in-

creased uptake often will occur in the desensitized limb.

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Forelimb Abnormalities

FOOT PROBLEMS

"Corns" (Subsoiar Bruising)

Subsolar bruising is a common cause of lameness in horses exercising on hard ground. Trauma to the sole results in hemorrhage between the sensitive and insensitive layers of the hoof. A common site is at the heel, between the bars and the wall, and at this site it is called "corns." Many horses are predisposed to subsolar bruising due to poor hoof conformation such as thin or flat soles and low heels. Horses that have pedal bone rotation due to laminitis also are commonly affected.

In horses with flat soles, it is often very difficult to apply shoes without pressure being applied to the sole. This compromises subsolar blood flow and predisposes to bruising. Some horses have collapse of the heel so that the horn tubules are growing parallel to the ground. This is often associated with bruising at the heel.

Horses occasionally present with acute onset of severe lameness, but most show chronic low-grade lameness due to repetitive injury.

HISTORY AND PRESENTING SIGNS

- Chronic low-grade forelimb lameness
- Reluctance to stretch out during exercise
- Occasionally acute lameness

CLINICAL FINDINGS AND DIAGNOSIS

- Most commonly horses are bilaterally lame, although the lameness may be more severe in one limb.
- Examination of the foot may reveal poor foot conformation and weight bearing of the shoe inside the white line. Application of hoof testers produces varying degrees of pain.

KEY POINT

Bruising may be observed when trimming the sole, especially in unpigmented feet, but can also be present in sound horses.

DIFFERENTIAL DIAGNOSIS

- Foot abscesses
- Navicular disease
- Nonarticular fractures of the pedal bone
- Pedal osteitis

TREATMENT

KEY POINT

Shoes should be applied so that all the weight is borne on the hoof wall and none on the sole. Using wide web shoes and grinding out the inside upper surface will protect the sole without impairing blood supply.

A bar shoe may be useful in more severe cases, particularly when bruising is at the heel. This will distribute weight evenly over the foot and prevent the concentration of force and pressure over the angle of the bars of the foot. Horses with collapsed heels may benefit from heel reconstruction with synthetic polymers.

Anti-inflammatory drugs such as phenylbutazone (Treatment No. 88) can be administered for 5 to 7 days to hasten recovery. A dose rate of 4.4 mg/kg should be used for 24 hours, followed by 2.2 mg/kg q12h thereafter.

Coronitis

Coronitis is an inflammation of the coronary band and usually results from systemic disease. It is a rare condition in clinical practice but may be seen in febrile illnesses or in horses with laminitis.

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HISTORY AND PRESENTING SIGNS

- Sudden onset of lameness with reluctance to bear weight
- Swelling around the dorsal coronary band of the foot
- Systemic illness

CLINICAL FINDINGS AND DIAGNOSIS

- Two or more feet are usually involved, and there is initial swelling of the coronary band, followed by exudate development and severe pain.
- Over a period of 7 to 14 days, there may be complete separation of the hoof wall from the coronary band.
- The result may be complete loss of the protective hoof horn from the affected foot.

DIFFERENTIAL DIAGNOSIS

- Foot abscesses
- Laminitis
- Fractures of the pedal bone
- Septic arthritis of the coffin joint

TREATMENT

There appears to be little effective treatment for this condition. The nonsteroidal anti-inflammatory drugs such as phenylbutazone (Treatment No. 88) or flunixin meglumine (Treatment No. 52) should be used, but the prognosis is very poor.

In many cases that do resolve, the coronary band is permanently affected so that abnormal hoof growth results.

Foot Abscesses (Subsolar Abscesses, Nail Prick)

KEY POINT

Foot abscesses are probably the most common cause of acute lameness encountered in equine practice.

When bacteria gain access to the sensitive structures of the foot, usually via cracks in the sole or hoof wall or from penetrating foreign bodies such as nails, an abscess usually results. Horses with hoof wall separation due to "seedy toe" or laminitis are particularly at risk. Due to the anaerobic conditions, bacteria such as *Clostridium* species may be found, and therefore, tetanus prophylaxis should always be given.

HISTORY AND PRESENTING SIGNS

- Horse recently shod with suspicion that the sensitive structures were "pricked"
- Horse kept at pasture in wet conditions after a dry period
- Chronic laminitis with secondary abscessation
- Known history of foreign-body penetration of the sole

CLINICAL FINDINGS AND DIAGNOSIS

- Acute, severe lameness unassociated with exercise, with the horse reluctant to bear weight on the affected leg.
- In the early stages of infection there is increased heat in the affected foot and coronary band, together with increased rate and amplitude of the pulse in the palmar or plantar digital arteries. After several days there may be little or no heat in the affected foot.
- After 24 to 48 hours there may be swelling in the pastern and fetlock.
- If untreated, the infection may underrun the hoof wall and ultimately form a sinus with a discharging point at the coronary band.
- The use of hoof testers demonstrates severe generalized pain (acute cases) or localized pain (chronic cases) over the sole. With care, even in acute, severe cases, the use of hoof testers around the sole should localize one site where there is more pain. Then this site can be investigated with a hoof knife.
- Paring of the sole with a hoof knife may indicate the site of penetration of the sole.
- Abaxial nerve block should abolish or substantially relieve the pain and lameness.
- In cases where the history of a penetrating injury is not clear, radiographs of the foot may be necessary to eliminate the possibility of a fracture of the distal phalanx.

DIFFERENTIAL DIAGNOSIS

- Fractures of the distal phalanx (pedal bone)
- Infection of the navicular bursa or deep flexor tendon sheath
- Laminitis
- Severe subsolar hematoma (corn)
- Fractures of the navicular bone

TREATMENT

- Determine the most painful site over the sole, because this is the point to begin paring with the hoof knife. It may be necessary to perform an abaxial block (see Fig. 4-26) to desensitize the foot in cases where there is severe pain. Once pus is located, the paring should continue until all underrun sole is removed.
- Establish effective drainage of the infected area

and apply a poultice dressing to the foot to encourage further drainage.
- In most cases, antibiotic therapy is not necessary and will be ineffective because the infection is localized. In a more generalized infection, the antibiotic of choice will be procaine penicillin (Treatment No. 83) (see Chapter 19) administered for 4 to 5 days at a dose rate of 15 mg/kg ql2h.
- Effective drainage is the key to successful treatment. The poultice is changed daily for 3 to 5 days and further trimming may be required.
- A protective boot can be applied until provisional keratinization protects the sensitive structures of the foot (5-7 days).
- Where there is extensive infection with underrunning of the sole and/or wall of the hoof, stripping of the sole and hoof wall may be necessary.

KEY POINT

It is essential to administer tetanus prophylaxis, and the usual regimen is to give 3000 IU of tetanus antitoxin (Treatment No. 115) subcutaneously, plus intramuscular tetanus toxoid (Treatment No. 116) administered in a site remote from the antitoxin injection. The tetanus toxoid injection should be repeated 4 weeks later in a horse with no previous vaccination history.

Foot Cracks (Sand Cracks)

Foot cracks usually result from the hoof becoming dehydrated and brittle or overlong. Some cracks, especially those at the toe, are secondary to hoof wall separation from the sensitive laminae. Most cracks are longitudinal and extend proximally up the hoof wall from the solar aspect. However, some hoof cracks arise from previous trauma to the coronary band with subsequent failure of horn growth at the injured site. In these cases, the crack will extend from proximal to distal as the hoof grows.

HISTORY AND PRESENTING SIGNS

- Previous trauma to the coronary band
- Poor hoof care
- Laminitis or "seedy toe"
- Wet followed by prolonged dry pasture conditions

CLINICAL FINDINGS AND DIAGNOSIS

- In most cases, the cracks, which are self-evident, will not cause any lameness. If, however, they extend into the deeper sensitive structures of the foot, infection may ensue, with signs of a foot abscess.
- It is important to determine the depth of the cracks to determine whether infection is likely.

KEY POINT

Foot conformation should be evaluated carefully together with assessment of shoe placement. Some cracks, particularly those toward the quarters and heels, may be due to abnormal conformation or poor shoe placement.

DIFFERENTIAL DIAGNOSIS

- Foot abscess
- Subsolar bruising

TREATMENT

- Appropriate trimming and rehydration of the hoof with a hoof dressing are important. A number of commercial hoof dressings are available.
- A number of techniques have been described for stabilizing hoof wall cracks with sutures, wires, or fiberglass. With the availability of hoof wall acrylics, resection of the crack and hoof wall reconstruction is now the preferred technique. The hoof wall is resected either side of the crack down to the white line, and provided no infection is present, the cavity is filled with acrylic usually reinforced with mesh and then an appropriate shoe applied.
- Bar shoes may be useful for reducing excessive hoof wall movement. If the crack is the result of hoof wall separation or a coronary band abnormality, permanent resolution is usually not possible.

Fracture of the Distal Phalanx (Pedal Bone, Third Phalanx)

Fracture of the pedal bone is a condition that occurs in all athletic horses and is more common in Standardbred trotters and pacers than Thoroughbred racehorses. Most fractures occur through the lateral wing of the pedal bone (type 1 fracture), and horses will show the most pain if the fracture extends intra-articularly (type 2 fracture).

HISTORY AND PRESENTING SIGNS

- The most common history is that a horse shows acute lameness immediately after a race and

within 30 to 60 minutes will be reluctant to bear weight on the affected leg.
- In cases where there is an intra-articular fracture, the lameness will gradually improve over 2 to 3 days with box rest, whereas in the case of nonarticular fractures, the degree of improvement is usually more pronounced.

CLINICAL FINDINGS AND DIAGNOSIS

KEY POINT

An acute non-weightbearing lameness is a feature of the presenting signs in horses with pedal bone fractures.

- Severe pain is evident on application of the hoof testers. This is usually concentrated on the lateral side of the sole and wall of the affected foot.
- Occasionally, horses will not show marked pain with hoof testers but will react to tapping around the affected side of the hoof wall with a hoof hammer or closed hoof testers.
- An abaxial block (see Fig. 4-26) will resolve the lameness, but most horses do not become completely sound.
- In many cases, horses will show considerable improvement after 48 hours' rest in a stall and, if the fracture does not extend into the joint, will walk almost sound.
- Radiography should include dorsopalmar, lateral to medial, dorsolateral to palmaromedial, and dorsomedial to palmarolateral oblique views. It is essential to establish whether or not the fracture extends into the coffin joint. In some cases, the fracture may be difficult to visualize on the dorsopalmar view. However, one of the oblique views will usually show the fracture more clearly. Also, undisplaced fractures may not be visible initially. Radiographs should be repeated at 10 to 14 days when lysis around the fracture will allow visualization.

DIFFERENTIAL DIAGNOSIS

- Fractures of the other phalanges
- Foot abscess
- Infection of the navicular bursa
- Fractures of the navicular bone

TREATMENT

- If the fracture does not extend into the coffin joint, conservative treatment with the application of a bar shoe (i.e., a bar of steel welded across the heels of an ordinary shoe) with quarter clips to limit hoof expansion will help to immobilize the fracture. Good results also have been reported recently with the use of a continuous-rim bar shoe.
- The bar shoe requires changing every 6 weeks or so, and the fracture will usually heal after 6 to 7 months of rest. In some cases, healing may be delayed for up to 12 months. During the first 2 to 3 months of rest, the horse should be confined to a box stall, after which a small yard or pasture is adequate.
- If the fracture is intra-articular, the application of a bar or rim shoe for 6 months together with rest for 9 to 12 months may result in the fracture healing if the horse is less than 3 years of age.

KEY POINT

Because of inconsistent healing of intra-articular fractures in horses over 3 years of age, internal fixation has been recommended for these cases.

This involves the insertion of a cortical bone screw in the distal phalanx, using the lag screw technique. Most fractures will heal in 6 to 9 months. A bar or rim shoe also should be applied. Some horses subsequently require screw removal because the screw loosens and irritates the sensitive laminae.

A disadvantage of internal fixation of pedal bone fractures is the possibility of osteomyelitis because of the difficulty in disinfecting the hoof before surgery.

Canker

Canker is a rare condition resulting in chronic hypertrophy of the horn-producing tissues of the foot. Although the hindfeet are more commonly affected, any foot or all feet may be involved. It is usually found in hot humid conditions, such as those found in the southern part of the United States.

HISTORY AND PRESENTING SIGNS

- Usually no lameness until well advanced
- Often there is neglect of the feet or poor hoof care
- Persistent foul-smelling exudate from the frog region, with little tendency to resolve spontaneously

CLINICAL FINDINGS AND DIAGNOSIS

- The foot has a distinct odor, and the frog, which may have a frayed appearance, loosens easily to

reveal a foul-smelling, localized necrotic horn covered with a caseous cream-colored exudate.
- The condition often goes unrecognized until well advanced, when the corium shows chronic proliferation and the sole and wall may be involved.
- The appearance of the foot together with the characteristic odor is diagnostic of canker.

DIFFERENTIAL DIAGNOSIS

- Thrush
- Foot abscess

TREATMENT

- Thorough debridement with removal of all affected tissue is essential. The foot should then be soaked in a bucket of hot water containing a suitable disinfectant (see Chapter 19) and a bandage applied. Astringent agents such as a mixture of 30 g zinc sulfate and 20 g lead acetate in 500 mL water (White lotion) may be used.
- Topical chloramphenicol has been found useful when applied by soaking a pad of cotton wool and bandaging this to the foot, repeating the treatment daily for 5 to 7 days. However, the use of chloramphenicol is banned in some countries because of classification of the horse as a food-producing animal.
- Systemic penicillin therapy (see Chapter 19) has been advocated for the treatment of canker but in our experience is not useful.
- The foot should be dressed until the condition has resolved, and the horse should be kept in a clean dry environment. The prognosis is guarded, and improvement, when it occurs, is slow.
- It is common for the hoof to require extensive debridement on a number of occasions.

Fracture of the Extensor Process of the Distal Phalanx (Pedal Bone)

Fractures of the extensor process of the pedal bone, also called type 4 pedal bone fractures, may or may not involve the insertion of the common extensor tendon.

KEY POINT

Although this type of fracture can cause a significant lameness, small fractures can occur that are not responsible for lameness.

Therefore, care needs to be taken in ascribing lameness (particularly chronic lameness) to extensor process fractures. Many older horses without lameness can be found to have small extensor fractures when radiographs are taken as part of a prepurchase examination. In these cases it may be difficult to provide clear advice to the client.

HISTORY AND PRESENTING SIGNS

- Acute onset of a moderate forelimb lameness after exercise
- Low-grade, chronic forelimb lameness

CLINICAL FINDINGS AND DIAGNOSIS

- Some horses with small fractures will show no clinical signs, and the extensor process fracture only may be found on a routine radiograph of the foot.
- In other cases, where larger portions of the extensor process have been fractured, there is lameness with swelling of the coronary band in the dorsal midline.
- Diagnosis should be made by a confirmatory nerve block to localize the lameness to the dorsal region of the foot (abaxial block or coffin joint block), together with a lateral radiograph of the foot to demonstrate the presence of a fracture.

DIFFERENTIAL DIAGNOSIS

- Trauma to soft tissues of the dorsal midline
- Low ringbone

TREATMENT

- Long-standing fractures that may not be causing lameness should be left untreated.
- Small fragments can be removed arthroscopically, which will also allow inspection of the dorsal aspect of the joint for evidence of associated degenerative changes.
- Larger fragments may have to be reaffixed using a lag screw to achieve stability. However, secondary arthritis is common, and a change in shape of the hoof wall (see Pyramidal Disease) may occur.

Grease Heel (Pastern Dermatitis, Scratches)

- Grease heel is a seborrheic dermatitis involving the skin at the palmar aspect of the pastern and occasionally the fetlock. Like most skin infections, it requires a predisposing factor such as moisture or repetitive trauma to become established. Horses with white legs appear to be especially susceptible. Organisms involved are

opportunistic pathogens such as dermatophilus, *Staphylococcus* species, and fungi. *Chorioptes equi* can cause a similar condition in horses with feathering.

HISTORY AND PRESENTING SIGNS

- Constant or repeated exposure to moisture (e.g., swimming, wet bedding, muddy paddocks, mud around water troughs)
- Low heel conformation
- White legs

CLINICAL FINDINGS AND DIAGNOSIS

- In the early stages there may be a mild dermatitis with swelling in the palmar region of the pastern.
- This is usually followed by secretion and exudate formation. In the chronic stages, the skin is thickened, and there may be excoriation.

DIFFERENTIAL DIAGNOSIS

- Other skin disorders, including contact dermatitis and allergic dermatitis, should be considered.

TREATMENT

- The most important part of therapy is to identify and remove the inciting cause. It is essential that the affected area is kept dry and to remove sources of moisture. Cleaning the area is important, and it may be necessary to clip the hair.
- In severe cases, a systemic course of an antibiotic such as procaine penicillin (Treatment No. 83) at a dose rate of 15 mg/kg (15,000 IU/kg) q12h may be necessary. If chorioptes is found in skin scrapings, an oral dose of ivermectin should be given.

Infection of the Navicular Bursa

Penetrating injuries of the sole or the frog may lead to penetration of the navicular bursa, which lies between the deep flexor tendon and the navicular bone on the palmar aspect of the pedal bone. Infection in this location leads to a severe non-weightbearing lameness of the affected leg that is very difficult to treat successfully. Penetrating wounds of the navicular bursa may also involve the distal interphalangeal joint and/or the digital tendon sheath due to their close proximity.

HISTORY AND PRESENTING SIGNS

- Nail or wire penetration to the foot
- Sudden onset of an acute foreleg lameness with the horse reluctant to take weight on the affected leg

CLINICAL FINDINGS AND DIAGNOSIS

- Severe non-weightbearing unilateral foreleg lameness.
- If any weight is taken on the leg, it is usually only on the toe, with heel held off the ground.
- Increased heat in the foot and increased digital pulse are found.
- Pain is evident on application of hoof testers, particularly over the frog.
- Improvement in gait is found after palmar digital nerve block (see Fig. 4-23), and further improvement usually occurs after abaxial nerve block (see Fig. 4-26). However, usually horses will not become sound.
- Radiographs may not show any abnormalities in the early stages of the condition. If possible the nail should be left in place and a lateromedial radiograph taken to determine its depth of penetration.
- If the lameness has been present for several days, radiographs may show radiolucency on the palmar aspect of the navicular bone, indicating the presence of infection. These changes are often most obvious on the lateral and skyline views of the navicular bone.
- In some cases it is possible to confirm the presence of infection by inserting a needle into the navicular bursa to aspirate purulent material. This is done following an abaxial nerve block to desensitize the distal limb. An 18-gauge, 8.5-cm (3.5-inch) needle is inserted in the axial plane in the palmar midline between the bulbs of the heels. A lateromedial radiograph is required for confirmation of correct positioning. Synovial fluid should also be collected from the distal interphalangeal joint and the digital tendon sheath to determine if they are also involved.

DIFFERENTIAL DIAGNOSIS

- Foot abscess
- Fracture of pedal bone
- Septic tenosynovitis
- Fracture of the navicular bone

TREATMENT

KEY POINT

It is important to make an early diagnosis of navicular bursa infection because response to treatment depends on early intervention.

- Treatment should include debridement of the navicular bone. Surgical exposure of the navicular bursa can be performed using the "street nail operation," which involves resecting the middle third of the frog down to the depth of the deep flexor tendon with an oscillating saw. A window is then cut in the deep flexor tendon to allow debridement of necrotic bone and establishment of drainage. It is important to determine if other synovial structures such as the distal interphalangeal joint and digital tendon sheath are involved. Injection of sterile saline under pressure into the joint or tendon sheath will result in fluid entering the surgical wound if there is direct communication. Lavage of these structures should be performed if they are involved.
- Alternatively, an arthroscopic approach to the navicular bursa has recently been described that is a less invasive method of debridement, and results in a small number of cases have been encouraging. A prolonged course of antibiotics, based on the results of culture, is essential.
- The prognosis is guarded and in cases where the infection has been present for more than 1 week, the degree of improvement may be limited. Healing of the surgical wound is often delayed when the "street nail" approach is used. Many horses will be left with a chronic lameness even if there is initial improvement in gait.

Laminitis

Laminitis is an avascular necrosis involving the sensitive laminae, which intermesh with the hoof wall. The exact cause is unknown, but blood bypasses the dermal laminae via arteriovenous shunts at the base of the laminae, either due to increased postcapillary resistance and edema within the laminae or as a direct effect of opening of the shunts due to vasoactive compounds. The unique housing of the blood supply to the foot in a nonexpandable structure (the hoof) may accentuate the impact of blood flow changes. Laminitis occurs in association with conditions that cause endotoxemia such as acute gastrointestinal diseases and metritis. However, endotoxemia is not consistently found in cases of experimentally induced laminitis, and laminitis has not been induced by administration of endotoxin. Therefore, there must be other mechanisms involved.

The outcome is a loss of blood supply to the laminae, despite an increase in blood flow to the foot. This leads to separation of the dermal and epidermal laminae and an unstable pedal bone within the foot. The pedal bone may rotate due to the pull of the deep digital flexor tendon or, with more extensive laminae involvement, displace distally.

The most common causes are grain engorgement, grazing in lush pastures, postfoaling metritis, and systemic gram-negative bacterial infections. In fat ponies, it is common to find laminitis during the spring months, when the soluble carbohydrate content increases in grasses and clovers. Laminitis can also occur in a single limb due to excessive weight bearing for a prolonged period.

HISTORY AND PRESENTING SIGNS

- Overfeeding of grain
- Gastrointestinal problems, particularly after colic surgery
- Mare recently foaled having had retained fetal membranes
- Fat pony grazing lush pasture
- Recent pleuritis or pneumonia due to gram-negative infection
- Non-weight-bearing lameness in opposite limb

CLINICAL FINDINGS AND DIAGNOSIS

- Although all four feet can be involved in laminitis, the forelimbs are more frequently affected than the hindlimbs.
- Affected horses show reluctance to move, and a typical "sawhorse" stance is seen with the forelimbs placed well out in front of the body. It will be difficult to pick up one of the forelegs.
- Examination usually will reveal increased heat in the feet and around the coronary band, with intense pain on application of the hoof testers.
- Palpation of the palmar arteries over the abaxial surface of the sesamoid bones will reveal an increase in both rate and amplitude of the pulse.
- An abaxial nerve block (see Fig. 4-26) will improve the signs of lameness, but in acute cases, it is contraindicated due to instability of the pedal bone.
- Signs of pedal bone rotation or sinking include a palpable indentation at the dorsal aspect of the coronary band and protrusion of the sole, dorsal to the apex of the frog. In chronic cases, abnormal hoof wall growth with a greater distance between growth rings at the heels than at the toe, and a concave dorsal hoof wall, are noted.

KEY POINT

Lateral radiographs should be taken of the feet to reveal the extent of rotation or distal displacement of the pedal bone. A radiopaque marker, such as a strip of metal, on the dorsal midline of the foot will allow accurate assessment of the degree of rotation.

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DIFFERENTIAL DIAGNOSIS

- Pedal osteitis
- Foot abscess
- Fractures of the pedal bone

TREATMENT

KEY POINT

In acute cases of laminitis, it is important to establish the cause so that treatment can be directed at eliminating the sources of vasoactive substances.

- In cases of grain engorgement, 3 to 4 L of paraffin should be administered by stomach tube to help eliminate the grain from the gastrointestinal tract.
- Where metritis is the initiating cause, aggressive irrigation of the uterus to remove accumulated exudates should be combined with systemic broad-spectrum antibiotics (see Endometritis, Chapter 8).
- Early in the course of disease, application of cold to the feet will be helpful in decreasing the severity of the problem. This can be done most simply by standing the horse in cold water.
- If there are signs of shock (prolonged capillary refill time, injected mucous membranes, etc.), intravenous fluid therapy may be required (20-40 mL/kg of a polyionic, isotonic fluid). For further details, see Chapter 19 (Fluid Therapy).

KEY POINT

In acute cases the horse should be confined to a stall, preferably with a deep sand bed as even walking has the potential to exacerbate displacement and rotation of an unstable pedal bone.

- If cases are identified within the first 48 hours, peripheral vasodilators such as acepromazine will improve digital blood flow and have been shown to prevent the development of chronic laminitis in a high proportion of cases. Acepromazine also has the advantage of sedating the horse, thus reducing movement. Therapy should be continued for 48 hours. Chronic cases of laminitis rarely respond to this type of treatment.
- Nonsteroidal anti-inflammatory drugs such as phenylbutazone (Treatment No. 88), flunixin meglumine (Treatment No. 52), or ketoprofen are essential. Maximal dose rates are used for the first 2 to 4 days, and continuation of therapy for between 7 and 21 days may be necessary depending on the response. Of the nonsteroidal anti-inflammatory drugs, phenylbutazone appears to be the drug of choice for effective pain relief. It is important to realize that nonsteroidal anti-inflammatory drugs have the potential to cause toxic effects, particularly on the gastrointestinal system, and potential toxicity should be monitored by intermittent measurement of plasma total protein concentration.
- Many different methods have been suggested to provide support for an unstable pedal bone. Deep sand bedding conforms well to the sole and also has the advantage of allowing the horse to stand with its feet at the most comfortable angle. Padded bandages can be applied to the feet if there is any risk of sole penetration. In acute cases shoeing should be avoided, because the concussion will cause pain, and prolonged weight bearing on the opposite limb is detrimental.
- Hoof wall resection is indicated where there is marked separation and the dorsal lamina is providing no support to the pedal bone and/or is applying excessive pressure to the coronary band, preventing normal growth. Sectioning of the deep digital flexor tendon, as a salvage procedure, can be used in cases that are not responding to therapy. Although often providing relief from signs, there is evidence that it does not improve the long-term prognosis.
- In chronic laminitis where some pedal bone stability has returned, corrective trimming of the feet is essential. Because the blood supply to the coronary band at the heels is greater than at the dorsal midline, it is important to trim back the excessively long heels to maintain normal heel height and to square the toe to facilitate breakover. Progress can be assessed from lateral radiographs.

Navicular Syndrome

Navicular syndrome is a degenerative condition involving the subchondral bone and fibrocartilage of the flexor surface of the navicular bone, and the navicular bursa and deep digital flexor tendon. A range of pathologies have been observed including subchondral bone sclerosis, subchondral lytic lesions, cartilage erosions of the flexor surface of the navicular bone, enthesiophyte formation, and adhesions between the deep digital flexor tendon and the navicular bone. Increased subchondral bone pressure has also been demonstrated in horses with navicular disease. The cause is unknown, but the most widely held view is that it is a response to abnormal or excessive forces on the bone with subsequent remodeling and subchondral bone failure. There is good clinical evidence that foot imbalance predisposes horses to navicular

disease. Diagnosis is complicated by the difficulty in specifically blocking the navicular bone and bursa and the poor correlation between radiographic changes and clinical signs.

KEY POINT

It is important to note that navicular disease is a clinical rather than a radiologic diagnosis.

HISTORY AND PRESENTING SIGNS

- An older (6- to 8-year-old) horse is the most common candidate with a chronic low-grade forelimb lameness.
- Intermittent, progressive lameness with exacerbations and remissions is typical in the early stages of the disease.
- Some horses will stand with an affected limb in front of the contralateral limb ("pointing").
- Thoroughbreds, Quarter horses, and warmbloods are affected more commonly than Standardbreds and other riding breeds.

CLINICAL FINDINGS AND DIAGNOSIS

- The most common presentation is a unilateral or bilateral chronic forelimb lameness that is gradually progressive in a horse with no obvious swelling in the leg(s) or abnormal findings on clinical examination.

KEY POINT

It is important to note that most horses with navicular disease will not show a positive response to hoof testers.

- A broken back hoof pastern axis is found in a high percentage of cases, and medial to lateral hoof imbalance is also common.

KEY POINT

A phalangeal and fetlock flexion test is a useful diagnostic test, with a high percentage of cases showing worsening of lameness, but the test is not specific for navicular disease.

- The test should be performed by flexion of the fetlock and phalanges, pulling up on the toe of the foot. After 1 to 2 minutes the horse is trotted off, and lameness is aggravated in approximately 60% of cases. This test is not diagnostic because a range of other distal limb disorders will show worsening of lameness after fetlock flexion. However, most navicular disease cases do not show marked pain during flexion, unlike problems involving the phalangeal or fetlock joints.
- A palmar digital nerve block should be performed to localize lameness to the palmar aspect of the foot. It is important to remember that many other structures are blocked, including the caudal sole and heels. Definitive diagnosis is by a navicular bursa block that requires the use of fluoroscopy to confirm accurate placement. Cases with pain arising from the navicular bone and bursa may also become sound with a distal interphalangeal joint block, but this will not differentiate navicular disease from pain arising from the joint itself.
- Radiography is also an important part of the diagnosis of navicular disease, but care must be taken in interpretation of findings. A complete set of views includes dorsopalmar, lateromedial, and palmar skyline views. Diagnosis in the past has been based on the size and shape of synovial fossa on the dorsopalmar view, although a number of studies have demonstrated no correlation between these findings and the degree of lameness. Enthesiophytes on the wings of the navicular bone are also very difficult to interpret. The most important view is the palmar skyline view. Cystic lesions or defects in the flexor surface subchondral bone are definitive signs of navicular disease. Other changes that may be observed are loss of corticomedullary definition on both the skyline and lateral views, osseous fragments on the distal border, and, in severe cases, calcification in the deep digital flexor tendon.

DIFFERENTIAL DIAGNOSIS

- Pedal osteitis
- Corns
- Low ringbone
- Shoulder problems
- Degenerative joint disease involving the distal joints of the forelimbs
- Fetlock degenerative joint disease

TREATMENT

KEY POINT

There is no treatment that is invariably successful for navicular disease.

- The first step is to address hoof balance. The most common problem is the overlong toe and low heels and/or lateromedial imbalance. The foot should be balanced and many cases will benefit from the application of a squared-toe,

egg-bar shoe. Heel reconstruction may be considered in horses with severely collapsed heels.

- Analgesic drugs such as phenylbutazone (treatment no. 88) may give temporary relief of lameness, and this combined with foot balancing may be successful in early cases. Other drug therapies that have been advocated include polysulfated glycosaminoglycans (PSGAGs) and isoxsuprine (Treatment No. 61). These drugs probably act by improving poor subchondral bone blood flow, and horses that respond best are those with minimal radiographic changes or that have been lame for a period less than 6 months.
- With isoxsuprine, an initial dose rate of 1.0 mg/kg q12h for 3 weeks is used. If there is complete relief of lameness, then an additional 3 weeks at the same dose rate is given, followed by 3 weeks of treatment once daily. If complete relief of lameness does not occur in the first 3-week period, the dose of isoxsuprine is increased by 50% and therapy is maintained for a further 6 weeks. If the horse is sound at the end of this period, the treatment can be decreased to once daily. We have found that there is a better long-term response to therapy if the drug dosage is not acutely discontinued but rather goes to an every-other-day schedule after the once-daily treatment. Many horses will have up to 6 to 12 months free of lameness after withdrawal of isoxsuprine therapy, particularly in milder cases of navicular disease.
- Treatment is difficult in more advanced cases or those that do not respond to drug therapy and hoof care.
- Desmotomy of the suspensory ligament of the navicular bone has been described as a method of altering the loading on the navicular bone. It is unclear how this works, but success rates of 75% at 6 months postsurgery and 43% at 3 years postsurgery have been reported. These results are very similar to those reported for palmar digital neurectomy, but there appear to be fewer side effects.
- Palmar digital neurectomy tends to be used as a last resort due to the possibility of side effects. Neuroma at the cut ends is often blamed for a poor clinical response, but true neuromas are rare. More commonly, a poor result is due to failure to section all the branches of the nerves or reinnervation postsurgery. Rupture of the deep digital flexor tendon is a more serious complication after palmar digital neurectomy but is uncommon and generally seen only where there is calcification of the deep digital flexor tendon.

Navicular Bone Fracture

Fracture of the navicular bone is a rare condition that can cause a forelimb or hindlimb lameness. Although most cases cause an acute lameness, the fracture may not be diagnosed initially, so it is possible to have horses presented with a chronic lameness, particularly if the fracture is undisplaced.

KEY POINT

A misdiagnosis of fracture of the navicular bone can be made because radiolucent lines associated with the sulci of the frog can appear to overlie the navicular bone in radiographs taken of the foot. A true fracture line will not extend above or below the respective borders of the navicular bone.

HISTORY AND PRESENTING SIGNS

- The most common presentation is that of an acute unilateral forelimb lameness after exercise, with reluctance of the horse to bear weight on the foot.
- If the fracture is displaced, the toe of the foot is rested on the ground, with hesitation of the horse to bear weight on the heel of the foot.
- The condition is found more commonly in pleasure horses than in horses used for athletic pursuits.

CLINICAL FINDINGS AND DIAGNOSIS

- Acute severe non-weightbearing unilateral foreleg lameness is most common.
- Occasionally, a more chronic unilateral forelimb lameness may be found.
- Pain on application of hoof testers across both heels and also over the middle third of the frog typically is found.

KEY POINT

Improvement of the lameness occurs after a palmar digital nerve block. However, many horses will require a low four-point block to completely block the lameness.

- Good-quality radiographs are essential for an accurate diagnosis. Because the lines of the sulci of the frog appear radiolucent on radiographs, it is easy to mistake these lines for fractures. Packing of the foot with a material such as Play-Doh will eliminate lines of the frog sulci.

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KEY POINT

The radiographic views that are most useful to enable a diagnosis are the dorsopalmar (high coronary or upright pedal) and skyline (special) navicular views.

- The skyline navicular view is taken with the foot on an x-ray plate and the direction of the beam coming down at an angle of 60 to 75 degrees vertical and from behind the leg. This demonstrates the flexor surface of the navicular bone and often permits a definitive diagnosis that may not be possible using the other views. If the fracture has been present for more than 2 to 3 weeks, considerable rarefaction will be found.

DIFFERENTIAL DIAGNOSIS

- Fracture of the pedal bone
- Infection of the navicular bursa
- Subsolar (foot) abscess
- Navicular disease

TREATMENT

- The prognosis is poor for athletic soundness in horses that have sustained fractures of the navicular bone.
- Rest will sometimes result in healing of the fracture, particularly if it is undisplaced. However, in many cases a fibrous union develops, and the fracture line can often be demonstrated several years after the fracture was sustained. Six months' stall rest is generally required before clinical signs are alleviated. This is often combined with a bar shoe with quarter clips and raised heels to reduce deep digital flexor tendon pressure on the bone and limit heel expansion.
- Palmar digital neurectomy may be used in horses that fail to improve with stall rest, but reported results are very poor.

Pedal Osteitis

- *Pedal osteitis* is a term used for bone lysis and new bone production on the solar margin of the distal phalanx. It is a common condition in athletic horses, often associated with exercise on hard tracks, and is usually bilateral. The changes observed in the pedal bone appear to be a response to chronic subsolar bruising.

KEY POINT

In many cases the problem arises from bad hoof conformation and excessive weight bearing on the sole.

HISTORY AND PRESENTING SIGNS

- Horse not stretching out during exercise
- Decreased performance
- The gait may be described as "looking like the horse is stepping on hot bricks"

CLINICAL FINDINGS AND DIAGNOSIS

- The horse may have a shuffling forelimb gait and may be reluctant to stretch forward when trotting so that there is a reduced anterior phase of the stride.

KEY POINT

Examination reveals pain over the entire sole on application of hoof testers. It is common for there to be more sensitivity at the toe and the heels of the foot.

- Pedal osteitis usually occurs bilaterally in the front feet. However, one foot may be affected more severely than the other.
- There is a great improvement in gait after an abaxial nerve block. There also may be some improvement after a palmar digital nerve block.
- Radiographs of the feet may show areas of roughening around the margins of the pedal bone, indicating bone lysis and periosteal new bone formation. Additionally, there is often rarefaction around the wings and toe of the pedal bone. Changes usually are visible on dorsopalmar (upright pedal or high coronary) and lateral to medial views and may be missed on overexposed radiographs. Solar margin fractures of the pedal bone also may be observed, as they have a similar pathogenesis.

DIFFERENTIAL DIAGNOSIS

- Subsolar bruising
- Navicular disease
- Fracture of the wing of the pedal bone (nonarticular)
- Solar margin fracture of the pedal bone
- Chronic low-grade laminitis

TREATMENT

- The shoes should be checked to ensure that they are not contacting the sole inside the white line.

KEY POINT

If there is excessive contact of the shoe with the sole, the inside margin of the solar aspect of the shoe should be ground down using an

electric grinder to relieve sole pressure so that the shoe only maintains contact with the wall up to the region of the white line.

- An egg-bar shoe may help by increasing the weight-bearing surface area.
- Various types of pads are available for use under the shoe, and some farriers favor rubber under the rim of the shoe to reduce the concussion. However, pads have only a small effect on the amount of concussion due to their limited thickness and full pads degrade the sole by trapping moisture.
- A course of phenylbutazone (Treatment No. 88) for 10 to 14 days (4.4 mg/kg q24h for 1 day, 2.2 mg/kg ql2h for 5 days, followed by 2.2 mg/kg q24h) in conjunction with stall rest may assist in resolution of the condition.

Club Foot

Club foot is the term given to a condition where one or more of the feet is abnormally shaped, with upright conformation and a foot axis of 60 degrees or more.

KEY POINT

The condition results apparently from initial flexure deformity of the deep digital flexor tendon.

The superficial digital flexor tendon and suspensory ligament also may be involved. In young horses it may be congenital or a result of nutritional deficiencies during growth. However, club foot may develop in horses of any age as a result of prolonged disuse of the foot or chronic painful conditions in the same limb.

HISTORY AND PRESENTING SIGNS

- May be an incidental finding on clinical examination
- May have developed acutely or present as a chronic condition that has been neglected
- Abnormal foot conformation usually concerns breeders at the time of weaning or before the sale of yearlings

CLINICAL FINDINGS AND DIAGNOSIS

- In acute cases, horses will present with normal-shaped hooves, but the heel is unable to touch the ground. These horses are generally lame in the affected limb.
- In chronic cases the heels are elongated and contracted and the dorsal hoof wall is concave. The appearance of the foot is diagnostic and the gait may or may not be affected.

KEY POINT

Radiographs are essential to discern whether the pedal bone has moved relative to the dorsal aspect of the hoof wall. In many cases, the pedal bone will appear misshapen, and there is periosteal new bone formation at the tip of the bone on a lateral-projection radiograph.

DIFFERENTIAL DIAGNOSIS

- Contracted heels
- Chronic laminitis

TREATMENT

- In acute cases, judicious use of phenylbutazone may result in the relaxation of the tendons and normal weight bearing. A careful clinical examination of the limb should be performed to identify sources of pain. If the limb fails to respond within 48 hours, an inferior check ligament desmotomy is indicated. Prolonged use of phenylbutazone in foals should be avoided due to the potential for toxic side effects.
- Chronic cases may be managed conservatively if there is no associated lameness. Trimming to improve the hoof angle is contraindicated because it will result in lameness. The hoof should be kept balanced but in its natural shape. Where the condition is causing lameness, an inferior check ligament desmotomy should be performed and combined with aggressive trimming and/or toe extension shoes to correct the hoof wall angle before the desmotomy heals.

KEY POINT

Care should be taken when inspecting the feet during clinical examinations because corrective trimming, as described above, may disguise a club foot.

Pyramidal Disease

Pyramidal disease is a condition that results in abnormal growth of the hoof wall in the dorsal midline so that the hoof wall at this point projects forward from the surrounding horn. It is usually the result of a chronic injury such as low ringbone or fracture of a large part of the extensor process of the pedal bone.

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HISTORY AND PRESENTING SIGNS

- Long-standing injury to the distal limb
- Gradual change in the shape of the dorsal hoof wall
- Low-grade lameness

CLINICAL FINDINGS AND DIAGNOSIS

- The change in shape of the hoof wall is diagnostic, with the appearance of a ridge in the hoof wall gradually extending from the coronary band to the sole.
- This change probably takes place from the local increase in blood supply. The horse usually will show lameness due to coffin joint degenerative joint disease.
- Radiographs should be taken of the foot. A lateral radiograph will demonstrate the extent of any bony changes involving the coffin joint.
- If lameness is present, a coffin joint block should be performed to determine if this joint is the site of the problem.

DIFFERENTIAL DIAGNOSIS

- Chronic laminitis
- Extensor process fracture
- Trauma to coronary band

TREATMENT

- The change in shape of the hoof wall is only a clinical sign of underlying pathology, which may or may not cause lameness.
- If radiographs indicate arthritis of the coffin joint, intra-articular medication may be useful. The best response is from long-acting corticosteroids.

Quittor

Quittor is infection of the lateral cartilage, usually following trauma around the heels. This is an uncommon injury these days, but it can be difficult to treat because of the avascular nature of cartilage and the failure of antibiotics to penetrate the area. The condition was most commonly found in draught horses after local cuts and trauma to the heel region of the foot.

HISTORY AND PRESENTING SIGNS

- Lacerations around the heel of the foot
- Chronic purulent discharge in the palmar area of the distal pastern

CLINICAL FINDINGS AND DIAGNOSIS

- Purulent discharge from the palmar/plantar lateral or medial aspect of the foot usually is found.
- Lameness is not usually a feature of the condition, particularly when the problem has been present for some period of time.
- Radiographs should be taken to determine whether there is any involvement of the pedal bone. If there is infection, there may be a radiolucency in one of the wings of the pedal bone.

DIFFERENTIAL DIAGNOSIS

- Osteomyelitis of the pedal bone
- Foot abscess
- Trauma to the heel
- Foreign-body (wood, wire) localization near the lateral cartilages

TREATMENT

- Infection in the lateral cartilage rarely responds to conservative treatment (antibiotics, irrigations).

KEY POINT

Radical surgery is necessary to resect any infected cartilage.

- Because the cartilage is distal to the coronary band, an incision should be made in the skin above the coronary band. Any discolored cartilage should be removed by sharp dissection, and the area should be packed with gauze soaked in povidone-iodine (Treatment No. 91). For extensive infections, a hole may be drilled in the hoof wall to allow more distal drainage.

Seedy Toe

Seedy toe is a condition that results from separation between the hoof wall and the sensitive laminae at the toe of the foot. The resulting space usually is filled with crumbly horn. It is often a sequel to laminitis, particularly in ponies, and it also occurs in horses with club foot.

KEY POINT

Horses with seedy toe are predisposed to recurrent foot abscesses.

HISTORY AND PRESENTING SIGNS

- Previous history of laminitis
- Lameness, which may be low grade or acute and severe depending on the presence of infection

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CLINICAL FINDINGS AND DIAGNOSIS

- In many cases there is no associated lameness. However, when the separation becomes filled with dirt or small stones so that there is pressure on the sensitive laminae, lameness ensues. Severe lameness will be present if an abscess develops under the separated horn.

DIFFERENTIAL DIAGNOSIS

- Foot abscess
- Chronic laminitis
- Club foot

TREATMENT

KEY POINT

Seedy toe is an unrewarding condition to treat, with recurrence quite common. Once separation has occurred between the laminae and the hoof wall, it is difficult to re-establish the connection.

A partial wall strip will allow access to the area, which is then cleaned and any necrotic tissue debrided. Radiographs will assist with monitoring correct foot balance and help determine how much toe can be removed. Regular trimming and keeping the feet as dry as possible will reduce the risk of recurrence.

Sidebone (Calcification of the Lateral Cartilages)

Sidebone is a calcification of the lateral cartilages that usually occurs in the front feet of older horses. Calcification of the cartilages is not a direct cause of lameness, unless there is fracture of the calcified cartilage from direct trauma.

HISTORY AND PRESENTING SIGNS

- Older horses are usually affected.
- Lameness is seldom found, except if there is a fracture of the calcified cartilage.

CLINICAL FINDINGS AND DIAGNOSIS

- Horses with fracture of the lateral cartilage present with acute lameness and evidence of external trauma.
- Diagnosis is made on a lateromedial radiograph. Occasionally, a separate center of ossification of the cartilage can be confused with a fracture.

DIFFERENTIAL DIAGNOSIS

- Trauma to the heel of the foot
- Fracture (nonarticular) of the wing of pedal bone

TREATMENT

- If the calcified cartilage is fractured, rest for 6 months with the horse shod with a bar shoe will usually result in healing.

Thrush

Thrush is a bacterial infection of the sulci of the frog resulting from prolonged exposure to excessively moist conditions where the horse is stabled. It results from improper foot care, where the sulci of the frog are not cleaned out daily.

KEY POINT

Thrush is seldom the cause of lameness unless the condition has been present for a prolonged period, with deep erosion of the sulci.

HISTORY AND PRESENTING SIGNS

- Usually found in stabled horses
- No lameness unless the condition is severe
- Owners may notice a foul smell associated with the feet

CLINICAL FINDINGS AND DIAGNOSIS

KEY POINT

There is a characteristic foul-smelling discharge associated with the affected frog sulci.

- The discharge is usually black in color, and there is a deep erosion of the medial and lateral sulci in severely affected cases.
- Most horses will not show any signs of lameness. However, when deep erosion and extension of infection occurs, the sensitive structures of the palmar/plantar foot may be involved, and the horse will become lame.
- Diagnosis is made by observation of the black discharge associated with the sulci of the frog.

DIFFERENTIAL DIAGNOSIS

- Foot abscess
- Canker
- Greasy heel

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TREATMENT

- It is important that the sulci of the frog are cleaned out and all necrotic horn is removed with a hoof knife. Various disinfectants (see Chapter 19) may then be used.
- Antibiotics are not usually necessary. However, if there is chronic infection with deep erosion of the sulci and lameness, it may be necessary to give a 5- to 7-day course of procaine penicillin (Treatment No. 83) at a dose rate of 15 mg/kg (15,000 IU/kg) ql2h.
- The client must be asked to clean the horse's feet out on a daily or twice-daily basis, and if the horse is being stabled in moist conditions, an attempt should be made to relocate it to a dry surface until the condition is controlled.

Keratoma

Keratomas are keratinized masses in the hoof wall or sole. Histologically, they consist of squamous epithelial cells, keratin, and granulation tissue. They are generally associated with chronic sepsis. Keratomas of the hoof wall often cause pressure necrosis of the underlying pedal bone.

HISTORY AND PRESENTING SIGNS

- Chronic lameness
- Recurring foot abscess

CLINICAL FINDINGS AND DIAGNOSIS

- Keratinized mass in hoof wall or sole
- Pain on application of hoof testers
- Distortion of white line adjacent to mass
- Well-circumscribed lytic area in pedal bone observed on upright pedal bone view

DIFFERENTIAL DIAGNOSIS

- Pedal bone osteomyelitis
- Other neoplasms of the foot
- Foot abscess
- Canker

TREATMENT

- The whole mass should be resected, and this may require a partial wall strip to improve access to the mass. This is best done under local anesthesia.
- A tourniquet applied at the fetlock will minimize bleeding.
- Depending on the size of the defect, a bar shoe with clips may be needed to prevent excessive instability in the hoof wall. The foot should be bandaged until adequate keratinization has developed.

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PASTERN PROBLEMS

Fracture of the Proximal (First) Phalanx

Fracture of the proximal phalanx is a common injury in performance horses. Fractures are usually longitudinal and incomplete, extending varying distances distally from the sagittal groove. Occasionally, comminuted fractures occur. Most cases present with an acute onset of lameness; however, some short undisplaced fractures may present as a chronic lameness.

KEY POINT

Fractures of the proximal phalanx are frequently undisplaced and may not be easily visible on radiographs.

HISTORY AND PRESENTING SIGNS

- Acute lameness after work or a race
- Lameness usually worsens dramatically in the first 30 minutes after stopping exercise
- The horse is usually presented unable to bear weight on the leg

CLINICAL FINDINGS AND DIAGNOSIS

- Reluctance to place any weight on the affected leg.
- If examined immediately after the injury, there will be little or no swelling.
- Pain is found on palpation, particularly over the dorsal aspect of the pastern and fetlock. There will also be pain on fetlock flexion.
- In an acute case, nerve blocks should be avoided and the area radiographed so that excessive

weight bearing does not occur on an unstable limb. There is minimal risk in blocking a horse with a history of chronic lameness. Chronic fractures may improve with an abaxial nerve block and become sound with a low four-point block. Response to an intra-articular block is variable.

- Radiography of the pastern region may demonstrate a fracture line(s), but in some cases the fractures may be difficult to detect. The most useful radiographic view is the dorsopalmar projection. If a fracture is suspected but cannot be seen on the radiographs, oblique views (10 degrees from the dorsal midline) should be taken. Scintigraphy may be used to localize the fracture if radiographs are negative. Repeat radiographs at 7 to 10 days will generally demonstrate a fracture line.
- Larger fractures may extend from the sagittal groove to the proximal interphalangeal (pastern) joint but more commonly turn toward the lateral cortex at the midpoint of the bone.

DIFFERENTIAL DIAGNOSIS

- Foot abscess
- Fractures of other phalanges
- Condylar fractures of the third metacarpal bone
- Acute joint sprain
- Fractures of other long bones
- Tenosynovitis
- Fracture of the navicular bone

TREATMENT

- Short sagittal fractures extending 1 to 2 cm can often be treated with box rest alone for 4 to 6 weeks. Radiographs should be taken to confirm healing, before letting the horse out into a yard. If the fracture line persists, placement of a single 4.5-mm lag screw across the fracture line may be required.
- Larger nondisplaced sagittal fractures should be repaired with internal fixation using two or more 4.5-mm cortical bone screws with the lag screw technique. For transportation before surgery, a dorsal splint bandage should be applied (see Chapter 19). Screws can be inserted using stab incisions after careful measurements are made on a dorsopalmar radiographic view to assess the degree of overdrilling necessary for the proximal drill hole. This is confirmed with intraoperative radiographs. If the fracture is displaced, an incision over the dorsal aspect is made to confirm accurate reduction. A short limb cast is applied for recovery only. In most cases the screws do not have to be removed subsequently. Horses should be box rested for 4 weeks, given walking exercise only for another 4 weeks, and then 4 weeks in a small yard. The horse should be reradiographed before returning to training.
- Comminuted fractures of the proximal phalanx can be repaired by internal fixation if there is an intact strut of bone running from the metacarpophalangeal joint to the proximal interphalangeal joint. Radical exposure of the bone is required to allow accurate reduction. Multiple 4.5-mm cortical screws are then placed with the lag screw technique. Smaller fragments may require the use of 3.5-mm screws.
- If there is no intact strut, a transfixation cast or external fixator are required to prevent collapse of the bone that will occur in a simple cast. Two or three 4- to 6-mm pins are placed transversely in the distal metacarpus and included in a fiberglass cast extending to immediately below the carpus. The cast is changed at 6 weeks and replaced with a standard half limb cast for another 6 weeks.

Fracture of the Middle (Second) Phalanx

Fractures of the middle phalanx are most common in pleasure horses performing cutting, roping, and barrel racing. Fractures are usually comminuted, although simple palmar or plantar eminence fractures also occur. These fractures are more common in the hindlimbs than the forelimbs.

HISTORY AND PRESENTING SIGNS

- Acute lameness after exercise or a race
- Inability to bear weight on the affected leg

CLINICAL FINDINGS AND DIAGNOSIS

- As with proximal phalanx fractures, horses with fractures of the middle phalanx will show acute lameness immediately after work.
- There will be severe pain on flexion or palpation of the distal pastern region, and the horse will be reluctant to bear weight. Nerve blocks should not be performed because of the possibility of worsening the fracture displacement.

KEY POINT

At least four radiographic views (dorsopalmar, lateral, and obliques) should be taken to ensure that the complete extent of the fracture lines can be seen, which may not be apparent if only dorsopalmar and lateral views are taken.

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DIFFERENTIAL DIAGNOSIS

- Foot abscess
- Fractures of other phalanges
- Acute joint sprain
- Fractures of other long bones

TREATMENT

- If the horse requires transport to a referral center, immobilization of the leg to below the carpus should be undertaken using a dorsal splint bandage (see Chapter 19) or a temporary cast.

KEY POINT

Because accurate reduction of these fractures at the proximal interphalangeal joint is extremely difficult, best results have been achieved when arthrodesis of the joint is performed.

- Horses treated by immobilization in a standard half limb cast (see Casting, Chapter 19) usually suffer severe pain for a prolonged period. Transfixation casts may be used where horses are being salvaged for breeding purposes. Unilateral palmar and plantar eminence fractures are best treated by arthrodesis of the proximal interphalangeal joint with three parallel 5.5-mm cortical screws placed across the joint. Bilateral or large unilateral plantar eminence fractures and comminuted fractures can be treated by internal fixation using two narrow 4.5-mm four hole plates, placed dorsolaterally and dorsomedially. Cortical screws of 5.5 mm are placed through the distal holes of the plates into each of the plantar/palmar eminence fragments as lag screws. A third screw may be placed between the plates. A short limb cast is kept in place for 4 to 6 weeks and the horse confined to a stall for 3 to 4 months. Walking exercise can begin at 2 to 3 months.

Ringbone (Phalangeal Exostosis)

Ringbone is the name given to a periostitis and osteitis with new bone growth involving the pastern region. Although it can affect both forelimbs and hindlimbs, it is most common in the forelimbs. It is classified in two ways:

Articular or nonarticular—This depends on whether or not the proximal (pastern) or distal (coffin) interphalangeal joints are involved or whether the abnormality is extra-articular.

High or low—*High ringbone* is that which involves the distal aspect of the proximal phalanx and the proximal part of the middle phalanx. *Low ringbone* is the term used to describe an osteitis and periostitis of the distal part of the middle phalanx and the proximal part of the distal phalanx (pedal bone). Ringbone is usually the result of direct or indirect trauma to the pastern and may be associated with an upright pastern conformation.

HISTORY AND PRESENTING SIGNS

- Chronic unilateral or bilateral forelimb lameness
- Swelling around the phalanges
- Usually older horses are affected

CLINICAL FINDINGS AND DIAGNOSIS

- There is a variable degree of swelling involving the dorsal and medial and/or lateral aspects of the pastern, usually around the middle to distal pastern region.

KEY POINT

If the ringbone is nonarticular, the horse may not be lame. However, if the ringbone is articular, then there will usually be a chronic lameness that varies in severity with the extent of the joint involvement.

- There may be no pain on palpation or flexion of the pastern, particularly if the problem is chronic. Degenerative joint disease of the distal interphalangeal joint often is associated with poor hoof balance.
- Some improvement will be observed after an abaxial nerve block. A low four-point block often is required to completely block the lameness. Intra-articular blocks of the proximal and distal interphalangeal joints will determine if there is joint involvement. However, it is important to remember that a distal interphalangeal joint block will also block pain associated with the navicular bone.
- Radiographs (a minimum of dorsopalmar and lateral views) should be taken to assess the extent of joint involvement. Changes usually will be most apparent on the lateral view. Typical findings include variable periosteal new bone production and narrowing of the joint space.

DIFFERENTIAL DIAGNOSIS

- Osteochondritis dissecans (OCD) of the distal first phalanx

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- Phalangeal fractures
- Trauma (e.g., wire cuts to the phalangeal region), resulting in periosteal new bone growth
- Collateral ligament damage

TREATMENT

- Once the problem is chronic, with extensive new bone growth and articular involvement, there is little that can be done apart from surgical arthrodesis of the proximal interphalangeal joint. Arthrodesis of the distal interphalangeal joint has been described but is difficult and results in residual lameness.
- It may be useful to inject a long-acting corticosteroid such as 80 mg of methylprednisolone acetate (Treatment No. 74) into the affected joint. This should be combined with correcting any hoof imbalance.
- Arthrodesis of the pastern joint can be accomplished using three 5.5-mm cortical screws inserted through the distal dorsal aspect of the proximal phalanx into the palmar/plantar aspect of the middle phalanx. The prognosis for athletic function is good when the surgery is performed in hindlimbs but more guarded for forelimbs.
- In the early stages of the condition, before new bone growth has occurred, it may be possible to limit the extent of the new bone growth. This can be done by giving a 2- to 3-week course of phenylbutazone (Treatment No. 88) at a dose rate of 4.4 mg/kg for the first 24 hours, 2.2 mg/kg q24h for 5 days, and 2.2 mg/kg q24h for 5 to 10 days. This medication is combined with restriction of movement and perhaps immobilization using a Robert Jones bandage (see Chapter 19).

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FETLOCK PROBLEMS

Osteoarthritis of the Fetlock Joint (Osselets, Fetlock Joint Sprain)

Osteoarthritis of the fetlock is a relatively common condition in athletic horses in both forelimbs and hindlimbs and is thought to be a wear-and-tear injury.

HISTORY AND PRESENTING SIGNS

- Variable degrees of forelimb lameness
- Swelling around the fetlock joint
- Usually found in horses used for racing or other high-speed athletic events

CLINICAL FINDINGS AND DIAGNOSIS

- In the early stages, there may be only swelling around the dorsal aspect of the fetlock and/or the palmar pouches.

KEY POINT

The most consistent clinical finding in horses with fetlock joint pathology is distension of the palmar pouches of the fetlock joint. The palmar pouch is located between the suspensory ligament and the palmar aspect of the distal third metacarpal bone and can be visualized by examining the fetlock joint from the lateral aspect.

- Even in the chronic stages of the condition there is pain on flexion of the joint, with more severe pain in the acute stage.

KEY POINT

The lameness can be aggravated by holding the fetlock flexed for 1 minute, after which the horse is trotted off.

- Lameness localized to the fetlock joint with a low four/six-point block (see Figs. 4-26 and 4—27), or intra-articular anesthesia (see Figs. 4-28, 4-29, and 4-30).
- Radiographs are essential to establish the severity of the condition and to rule out intra-articular fractures. Four views (dorsopalmar, lateral to medial, and two oblique views) are necessary.

DIFFERENTIAL DIAGNOSIS

- Chip fracture of the dorsal aspect of proximal articular margin of the proximal phalanx

- Chronic proliferative synovitis (villonodular synovitis)
- Tearing of the insertion of the common and/or lateral extensor tendons on the proximal dorsal area of the proximal phalanx
- Sesamoiditis

TREATMENT

- There are a variety of treatments for osteoarthritis of the fetlock (see section on treatment of osteoarthritis, below). Cases with synovitis only will respond well to anti-inflammatory therapy such as nonsteroidal anti-inflammatory drugs either systemically or topically combined with dimethyl sulfoxide. Intra-articular hyaluronate also has anti-inflammatory properties, and best results appear to be in cases with signs of early osteoarthritis. Inconsistent results have been achieved with PSGAGs and pentosan polysulfate given systemically, with some cases responding very well and other cases showing no response to treatment. In advanced cases of osteoarthritis, intra-articular corticosteroids may be the only treatment that resolves the clinical signs.

Chip Fracture of the Proximal Phalanx

Chip fractures in the fetlock joint usually occur at the dorsal aspect of the proximal articular margin of the proximal phalanx and appear to arise from repetitive loading due to overextension of the fetlock joint during fast exercise. The chips occur on either the medial or lateral dorsal eminence of the proximal phalanx. These fractures commonly are associated with some enlargement of the dorsal synovial pad of the third metacarpal bone, which can be seen best on observing the dorsal profile of the fetlock joint from a lateral view.

HISTORY AND PRESENTING SIGNS

- Standardbred, Thoroughbred, and Quarter horse racehorses most commonly affected
- Low-grade forelimb lameness after an initial acute episode
- Swelling around fetlock joint, particularly the dorsal aspect

CLINICAL FINDINGS AND DIAGNOSIS

KEY POINT

In some cases, there will be little or no lameness resulting from the fracture, and the presence of a chip fracture on a radiograph may be an incidental finding.

- In cases where the fragment is the cause of lameness, there will generally be swelling over the dorsal aspect of the fetlock joint, distension of the palmar pouches of the joint capsule, and perhaps slight pain on flexion of the fetlock.
- If there is doubt whether the fragment is the cause of lameness, an intra-articular block should be performed (see Figs. 4—28, 4-29, and 4—30) to demonstrate an improvement in gait.

KEY POINT

The chip fracture is most easily seen on a lateral radiograph of the fetlock.

Oblique views (dorsolateral to palmaromedial and dorsomedial to palmarolateral) should be used to determine whether the fracture involves the lateral or medial dorsal eminence of the proximal phalanx.

DIFFERENTIAL DIAGNOSIS

- Arthritis of the fetlock joint
- Chronic proliferative synovitis (villonodular synovitis)
- Tearing of the insertion of the common and/or lateral extensor tendons on the proximal dorsal area of the proximal phalanx
- Synovitis of the dorsal fetlock joint capsule

TREATMENT

- If these chip fractures are causing lameness, they can be removed. Arthroscopic surgery will allow assessment of the degree of degenerative changes associated with the lesion and the size of the dorsal synovial pad.
- Most horses with small fractures can resume training 4 to 6 weeks after surgery. However, this is dependent on the status of the articular cartilage of the dorsal fetlock as visualized during arthroscopy. If there are extensive areas of cartilage erosion, a 3- to 6-month rest period should be given together with the possible use of intra-articular medication.

Palmar/Plantar Condylar Necrosis (Traumatic Osteochondrosis)

This is an acquired condition of performance horses that results in sclerosis of the subchondral bone of the palmar/plantar condyles of the third metacarpal/metatarsal bone and superficial necrosis of the bone and overlying articular cartilage. In advanced cases, subchondral cystic lesions may develop. Diagnosis is difficult due to the insensi-

tivity of radiographs to detect changes in subchondral bone density. However, scintigraphy appears to be very sensitive to bone remodeling in this area.

HISTORY AND PRESENTING SIGNS

- Forelimb or hindlimb lameness in a racehorse
- Usually minimal swelling

CLINICAL FINDINGS AND DIAGNOSIS

- Forelimb or hindlimb lameness localized to fetlock joint with regional or intra-articular anesthesia.
- Effusion of fetlock joint may or may not be present.
- Minimal radiographic changes in early cases. In more advanced cases, subchondral lytic lesions observed in the subchondral bone of the palmar/plantar aspect of the condyle.
- A flexed dorsopalmar/plantar view of the fetlock with the limb pulled forward will highlight the palmar/plantar surface of the condyle and show subchondral bone lesions.
- Increased uptake on scintigraphy scans in the palmar/plantar condyle.

DIFFERENTIAL DIAGNOSIS

- Fetlock osteoarthritis
- Intra-articular fractures
- Sesamoiditis
- Sesamoid fractures

TREATMENT

- Because many cases are advanced when the diagnosis is made, there is little that can be done due to the extensive nature of the subchondral bone necrosis and the inaccessibility of this site. Earlier cases identified with scintigraphy may respond to rest and modification in training regimens. However, the prognosis remains guarded.

Palmar Annular Ligament Constriction

The palmar annular ligament is a fibrous band that wraps around the flexor tendons as they pass over the sesamoid bones at the palmar aspect of the fetlock joint in the digital sheath. This forms an inelastic canal, constriction of which occurs when there is shortening of the annular ligament itself or swelling of any of the contents of the canal.

The most common cause in forelimbs is tendonitis of the superficial digital flexor tendon, but constriction may also occur due to deep digital flexor tendonitis, chronic tenosynovitis of the digital sheath, or desmitis of the annular ligament. In the hindlimb, trauma to the annular ligament with subsequent desmitis is the most common cause. Some cases are complicated by adhesion formation within the digital sheath.

KEY POINT

The most consistent aspect of palmar annular ligament constriction is the characteristic notch in the palmar contour of the limb at the proximal margin of the annular ligament.

HISTORY AND PRESENTING SIGNS

- Superficial flexor tendonitis in the distal metacarpal area (low bow)
- Chronic, progressive, but low-grade forelimb lameness
- Swelling noted above the palmar aspect of the fetlock joint, with a characteristic "notched" appearance

CLINICAL FINDINGS AND DIAGNOSIS

- Because of the constriction, there is usually notching at the palmar aspect of the fetlock, with distension of the flexor tendon sheaths above the annular ligament. The notching is a result of the inability of the inelastic ligament to swell in response to inflammation, and therefore, the swelling occurs above the ligament.
- The horse will show various degrees of lameness depending on the initiating cause of the swelling and its severity.
- A palmar nerve block above the proximal extent of the digital sheath usually will result in improvement of the lameness, but due to the physical constriction, complete resolution of the lameness may not occur.

KEY POINT

Ultrasound examination will determine which structures are affected and the severity of tendon involvement. Adhesions also may be apparent.

DIFFERENTIAL DIAGNOSIS

- Tenosynovitis
- Sesamoid bone injuries
- Flexor tendon injuries
- Sesamoidean ligament injuries

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TREATMENT

- Sectioning of the palmar annular ligament (desmotomy), to relieve the constriction, is generally effective, provided adhesions are not present. This surgical procedure can be undertaken via a small stab incision just above the annular ligament, followed by subcutaneous sectioning of the ligament with scissors. Although most horses show improvement after surgery, the underlying cause of the problem should be established and in particular whether there is any tendon damage. If adhesions are suspected, a larger incision will allow inspection of the proximal digital sheath to confirm their presence. Alternatively endoscopy will allow the whole sheath to be inspected. A technique for sectioning the annular ligament via endoscopy has been described. Adhesions can be broken down, but this must be combined with a postoperative exercise program to minimize recurrence, which often occurs.
- If surgery is not possible because of economic considerations, some horses may show short-term (1-3 months) response after injection of 80 to 120 mg methylprednisolone (Treatment No. 74) into the distended area of the flexor tendon sheaths.

Proximal Sesamoid Fracture

The sesamoid bones are part of the suspensory apparatus and receive reflections to their abaxial surfaces from the suspensory ligament. With repetitive overextension of the fetlock joint, which occurs during fast exercise, fatigue failure of the sesamoid bone(s) may occur.

HISTORY AND PRESENTING SIGNS

- Acute forelimb lameness with variable degrees of swelling
- Athletic horses (racehorse, polo horse, eventing horse, etc.)

CLINICAL FINDINGS AND DIAGNOSIS

- The forelimbs are more frequently involved than the hindlimbs. The degree of lameness depends on the size of the fracture fragment and the degree of damage to the suspensory ligament.
- Examination reveals pain on flexion of the fetlock joint or on palpation over the affected sesamoid bone(s). Within 12 to 24 hours there is swelling around the palmar fetlock that will vary in degree depending on the extent of the sesamoid fracture(s). If both sesamoid bones in the one leg are fractured, there will be complete loss of support for the fetlock joint, with the result that the joint becomes overextended and the fetlock sinks toward the ground.
- Radiographs will establish the extent of the fracture(s), and it is important that the relevant oblique view be included in addition to the dorsopalmar and lateral views. With some basilar fractures, it may not be clear whether the fracture arises from the distal articular margin of the sesamoid bone or the proximal palmar aspect of the proximal phalanx. In such cases, a flexed lateral radiographic view may be helpful.
- Ultrasound examination of the suspensory apparatus is important to determine the degree of damage to soft tissue support structures. It is also useful for establishing the position of small sesamoid fragments.

DIFFERENTIAL DIAGNOSIS

- Sesamoiditis
- Injury to the sesamoidean ligaments
- Palmar annular ligament constriction
- Flexor tendon and suspensory ligament injuries

TREATMENT

KEY POINT

The time that the fracture has been present is vital to the prognosis. Fractures that have been present for less than 1 month have a favorable prognosis with surgery, whereas fractures present for greater than 1 month have a more guarded prognosis.

- The course of treatment will depend on the extent and type of fracture found on radiographs. With apical fractures, where less than one-third of the bone is involved, surgical removal is the treatment of choice, and the prognosis is quite good provided suspensory ligament damage is minimal. If more than one third of the bone is involved, removal of the fractured piece of bone should not be undertaken because there will be too much damage and loss of support for the suspensory apparatus. Surgery is performed via a vertical incision in the lateral or medial palmar pouch of the fetlock joint. Smaller fragments can be removed arthroscopically.
- In fractures involving more than one third of the bone or basilar fractures, initial immobilization in a cast (see Casting, Chapter 19) followed by a rest of 9 to 12 months may result in a fibrous union. Internal fixation techniques, using cortical bone screws or cerclage wire, can be

performed. Success rates reported for this surgery range from 30 to 60%. When midbody fractures occur in both medial and lateral sesamoid bones, arthrodesis of the fetlock joint is the only option.
- Although removal of small basilar chip fractures is difficult, there is a better prognosis when the chips are removed than when such fractures are left undisturbed and treated by rest.

Sesamoiditis

Sesamoiditis is a term used to describe radiographic changes in the proximal sesamoid bones, including enthesiophyte production and widening of vascular channels. The cause of these changes is probably high-strain repetitive loading of the suspensory apparatus resulting in tearing of the suspensory ligament attachments and bone resorption within the sesamoid bones. A high incidence of suspensory desmitis has been associated with sesamoiditis.

HISTORY AND PRESENTING SIGNS

- Low-grade, unilateral or bilateral foreleg lameness
- Usually found in racehorses (Thoroughbred or quarter horse)
- History of gait restriction during exercise

CLINICAL FINDINGS AND DIAGNOSIS

- The clinical signs of sesamoiditis are similar to but less severe than those resulting from sesamoid fractures. Depending on the extent of the damage, there will be varying degrees of lameness and swelling. In most cases lameness will be substantially improved following a low four-point block (see Fig. 4-29).
- Pain on the flexion of the fetlock and on palpation of the sesamoid bones usually is found. Some cases of severe sesamoiditis may be difficult to distinguish from sesamoid bone fracture, and therefore good radiographs are required.

KEY POINT
The most useful views for examining the sesamoids are dorsolateral to palmaromedial oblique (shows the lateral sesamoid bone), dorsomedial to palmarolateral oblique (shows the medial sesamoid bone), and lateral to medial views.

- The radiographic features of sesamoiditis include changes in contour of the abaxial surface of the affected sesamoid, calcification within the suspensory ligament, and radiolucent lines (looking similar to fracture lines except that there is no fragment distraction) running obliquely lateromedially across the bone. These changes are most easily seen on oblique views.

KEY POINT
Many horses with sesamoiditis also will have suspensory ligament damage.

- It is important to palpate both medial and lateral branches of the suspensory ligament and to compare the findings with those of the opposite foreleg. Ultrasound examination should be undertaken to fully assess the suspensory ligament.

DIFFERENTIAL DIAGNOSIS

- Sesamoid bone fracture
- Navicular disease
- Pedal osteitis
- Suspensory ligament injury
- Palmar annular ligament constriction

TREATMENT

- Despite a variety of treatment regimens, the prognosis for horses with sesamoiditis is not good. A high percentage of horses, even those that have had prolonged rest, will become lame 6 to 8 weeks after recommencing training.
- In cases where there is local swelling and evidence of inflammation, a 7- to 10-day course of oral phenylbutazone (Treatment No. 88) may be useful. The dosage regimen is 4.4 mg/kg for the first 24 hours, followed by 2.2 mg/kg ql2h for 2 to 3 days, and then 2.2 mg/kg q24h.
- Depending on the severity of the problem and the response to initial therapy, a rest period may be necessary. In mild cases, a 3- to 4-week rest period is required, but in more severe sesamoiditis, 3 to 6 months of rest may be needed.
- Oral isoxsuprine therapy (Treatment No. 61) may help some cases, but the mechanism for this is unknown.

Chronic Proliferative Synovitis (Villonodular Synovitis)

Chronic proliferative synovitis is a condition that causes a low-grade foreleg lameness in racehorses and other athletic horses. Hypertrophy of the synovial pad on the dorsal aspect of the third metacarpal bone, just distal to the attachment of the joint capsule, appears to occur in response to repetitive

trauma from the dorsal aspect of the proximal phalanx. Osteochondral damage on the proximal phalanx commonly is observed in association with this condition.

HISTORY AND PRESENTING SIGNS

- Low-grade chronic forelimb lameness
- Swelling of dorsal fetlock region

CLINICAL FINDINGS AND DIAGNOSIS

- Unilateral forelimb lameness
- Distension of fetlock joint capsule, including the palmar pouches
- Pain on flexion and marked worsening of lameness after 1 minute of flexion
- Improvement in gait after intra-articular anesthesia of the fetlock (see Figs. 4-28 to 4-30)
- Radiography of the fetlock should be done, using four radiographic views (dorsopalmar, lateral, dorsolateral to palmaromedial oblique, and dorsomedial to palmarolateral oblique)

KEY POINT

On the lateral radiographic view, there is a characteristic indentation on the dorsal distal aspect of the third metacarpal bone, just above the articular surface.

- Ultrasound of the dorsal aspect of the fetlock joint will demonstrate thickening of the synovial pad. This should be differentiated from joint capsule thickening. The lesion also can be demonstrated by an air arthrogram, which is simpler, cheaper, and causes less joint reaction than iodine-based contrast agents.

DIFFERENTIAL DIAGNOSIS

- Fetlock arthritis
- Chip fracture of dorsal aspect of proximal articular surface of the proximal phalanx
- Fetlock joint sprain

TREATMENT

- The synovial pad can be removed arthroscopically and any osteochondral damage on the proximal phalanx debrided.
- The prognosis for return to full athletic function after surgery is good. Rest time depends on the degree of associated osteochondral damage and usually ranges from 3 to 6 months.

Plantar/palmar Proximal Phalangeal Osteochondral Fragments

Osteochondral fragments of the plantar/palmar aspect of the proximal phalanx are a developmental lesion that are particularly common in Standardbreds and occasionally identified in other breeds. The most common site is the medial aspect of a hindlimb. These lesions only cause lameness at high speeds and occasionally are associated with osteoarthritis of the fetlock joint.

HISTORY

- Poor performance
- Inability to run straight at high speed

CLINICAL FINDINGS AND DIAGNOSIS

- Fetlock joint effusion may or may not be present
- Most cases will have a positive fetlock flexion test

KEY POINT

Lameness should be localized to the fetlock joint by local anesthesia. However, in cases where lameness only is present at high speed, this may need to be done at the racetrack, or intra-articular corticosteroids can be injected and the horse's performance monitored. Radiographs of the fetlock should include elevated oblique views that separate the sesamoid bone and plantar/palmar aspect of the proximal phalanx to allow better visualization.

TREATMENT

- Most horses will respond temporarily to intra-articular corticosteroids.
- Arthroscopic removal of the fragments is the most successful treatment, with approximately 75% of horses improving their performance postoperatively.

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METACARPAL PROBLEMS

Bucked Shins (Dorsal Metacarpal Disease)

"Bucked shins" is the lay term for pain and new bone production associated with the dorsomedial aspect of the middle third of the third metacarpal bone.

KEY POINT

Bucked shins is by far the most common lameness affecting 2-year-old racehorses in their first training season.

It appears that this syndrome is a response to decreased bone stiffness due to cyclic fatigue. The bone responds by laying down new bone on the dorsal aspect. Rapid subperiosteal bone deposition causes pain and results in the typical radiographic appearance. In some horses, stress fractures of the dorsal cortex occur.

HISTORY AND PRESENTING SIGNS

- Two-year-old racehorse
- Bilateral forelimb lameness (more common than unilateral)
- Shuffling forelimb gait with reluctance of horse to stretch out

CLINICAL FINDINGS AND DIAGNOSIS

- Both forelegs usually are affected, and the horse may show a range of signs from reluctance to stretch out to severe lameness.
- There is usually some swelling in the dorsal midmetacarpus, with severe pain on mild palpation of this region.
- In cases that have resolved, there may be periosteal new bone production with obvious swelling in the dorsal midmetacarpal region, but there will be no pain on palpation.
- Radiographs may be necessary in cases with recurrent or severe lameness because some horses have fissure or saucer fractures of the dorsal cortex of the third metacarpal bone. Such fractures cause a more severe lameness that tends not to respond to the usual treatments of anti-inflammatory drugs and rest. If a horse fails to respond to rest and has a recurrence of lameness, fissure fractures should be suspected.

DIFFERENTIAL DIAGNOSIS

- Fissure fracture of the dorsal cortex of the third metacarpal bone
- Soft-tissue swelling of tissues over the dorsal metacarpus
- Condylar fracture of the third metacarpal bone

TREATMENT

The type of treatment depends on the extent of new bone production. Large amounts of new bone suggest significant weakening of the dorsal cortex. These horses should be rested to allow remodeling. Where there is minimal new bone production the horse can be kept in training provided the training load is substantially modified.

- There is evidence that the incidence of bucked shins can be substantially reduced by modifying training regimens. Instead of slowly increasing distance and speed, it is recommended that when speed is increased, distance is reduced. This allows the bone to adapt to high-speed loading without causing significant loss of bone stiffness.
- Horses that develop early signs of bucked shins should be treated with nonsteroidal anti-inflammatory drugs and have the distance of their high-speed training halved. The distances over which they are trained at high speed are then slowly increased as clinical signs improve.
- Studies have shown that the type of track surface on which the horse trains has a bearing on the incidence of bucked shins. Horses trained on dirt have a higher incidence of this problem than those trained on a wood-fiber track. One would also anticipate that the incidence of this problem would be lower in horses trained on turf tracks. This is borne out to some extent by

the lower incidence of bucked shins reported in the United Kingdom, where horses are trained on grass, than in the United States, where training on turf is less common.

Fractures of the Third Metacarpal (Cannon) Bone

Fractures of the third metacarpal bone (McIII) may be the result of direct trauma (e.g., kicks), in which case they are usually *transverse fractures* in the midmetacarpus, or a racing injury, in which fractures are usually longitudinal oblique fractures *{condylar fractures)* extending proximally from the distal articular surface of the McIII. The other types of fractures are *stress fractures* involving the middle third of the dorsal cortex of the McIII.

HISTORY AND PRESENTING SIGNS

- Direct trauma, usually a kick from another horse, particularly in foals
- Acute lameness after fast exercise (racehorses)

CLINICAL FINDINGS AND DIAGNOSIS

- *Transverse midmetacarpal fractures* provide no difficulty in diagnosis because the leg is obviously fractured. It is important to establish whether the fracture is open or not, because compound fractures have a very poor prognosis. Radiographs (a minimum of lateral to medial and dorsopalmar views) are necessary to determine the extent of the fractures. *Condylar fractures* usually occur after racing or fast work and result in a severe acute lameness. Examination reveals some pain on flexion of the fetlock joint together with pain on palpation over either the medial or lateral surface of the dorsal metacarpus.

KEY POINT

Most commonly, the fractures are of the lateral condyle, and of the athletic breeds, Thoroughbred racehorses are most frequently affected.

- A dorsopalmar view is usually the best radiographic view to demonstrate the presence of a condylar fracture line(s). However, in some cases there may only be a fissure fracture, and radiographs may fail to reveal any fracture lines. If the clinical signs indicate the likelihood of a condylar fracture despite negative radiographic findings, a number of oblique radiographic views should be taken at 10-degree intervals from the vertical midline. Such views may show small fissure fractures that may not be apparent on routine views.
- *Stress fractures of the dorsal cortex* are sometimes found in Thoroughbred racehorses. Clinical signs include variable lameness that is mild to moderate. Usually, horses will show some lameness at the trot, and there is pain over the middorsal metacarpal region on palpation. Good-detail lateral radiographs show the presence of a very fine fracture line, usually in the middle of the dorsal cortex and/or a saucer-shaped fragment. These fractures sometimes can be difficult to see, and fine-detail film may be necessary. Scintigraphy or xeroradiography, if available, will be useful in these situations.

DIFFERENTIAL DIAGNOSIS

- Fracture of the proximal phalanx (condylar fracture)
- Fracture of the sesamoid bones (condylar fracture)
- Fracture of the pedal bone
- "Bucked shins" (dorsal metacarpal periostitis and cortical fractures)

TREATMENT

Transverse Midmetacarpal Fractures

- With transverse fractures of the midmetacarpus, the most important aspect of treatment is the effective immobilization of the leg before transport to a surgical facility. The type of splint depends on the level of the fracture (see Chapter 19).
- Some midmetacarpal fractures may heal with a cast (see Chapter 19) applied from the foot up to just below the elbow. However, prolonged immobilization (3-6 months) often is required, and nonunion or delayed union often occurs.

KEY POINT

The treatment of choice is internal fixation using compression plates and screws, and this is most successful in foals.

- In very young foals, one plate is sufficient, but in weanlings, yearlings, and older horses, two plates placed at right angles usually are necessary. Surgery needs to be combined with external fixation using a cast for 4 to 6 weeks. After the cast is removed, a Robert-Jones supporting bandage is necessary for a further period, until there is some radiographic evidence of fracture healing. The plates and screws may require re-

moval if a discharging sinus or lameness develops due to screw movement.

Condylar Fractures

- Condylar fractures that are undisplaced can be treated with a cast applied to just below the carpus (see Chapter 19). However, if there is some displacement of the fractured piece, arthritis may result because of the difficulty in achieving perfect anatomic reduction, with external coaptation alone. Comminution or osteochondral defects often occur at the palmar aspect of the condyle of the third metacarpal bone.

KEY POINT
The treatment of choice is the transverse insertion of two or three cortical bone screws, starting just above the fetlock joint, using the lag screw technique.

- If there is no displacement, internal fixation can be performed using 4.5-mm cortical bone screws inserted using the lag screw technique via stab incisions. Displaced fractures require arthroscopy or arthrotomy to check accurate reduction at the joint surface. A cast (see Chapter 19) or a heavy Robert-Jones bandage (see Chapter 19) is applied for the immediate postoperative period, but after 7 to 10 days, a lighter bandage is all that is required. The horse should be kept in a box stall for 4 to 6 weeks, and healing should be complete in 4 to 6 months. In most cases, the screws need not be removed. However, if lameness develops as a result of the screws loosening (radiography shows lysis around screw heads), then the screws should be removed.

Fissure and Saucer Fractures of the Dorsal Cortex

- Dorsal cortical fractures often do not heal satisfactorily with rest alone. Treatment methods include forage and/or unicortical screw fixation. More consistent results have been achieved with a combination of the two methods rather than forage alone. Most fractures have healed at 2 months, and the screw can be removed.

Fracture of the Second or Fourth Metacarpal/Metatarsal (Splint) Bones

The small metacarpal bones are prone to trauma. Fracture of the fourth metatarsal bone is common in horses that kick fences or are kicked by other horses.

KEY POINT
Radiographs of these bones are essential when assessing traumatic wounds to the metacarpus or metatarsus.

Fracture of the second metacarpal bone may be associated with suspensory ligament desmitis.

HISTORY AND PRESENTING SIGNS

- Low-grade forelimb lameness
- Swelling around medial aspect of midmetacarpus
- Trauma to metacarpus/metatarsus

CLINICAL FINDINGS AND DIAGNOSIS

- There is usually a mild to moderate lameness and a variable degree of swelling, depending on the extent of the fracture.

KEY POINT
In both acute and more long-standing cases, there is pain on palpation over the fracture site.

- When the second metacarpal bone is fractured, it usually occurs in the middle to lower third of the bone. Dorsomedial to palmarolateral oblique radiographs are necessary to evaluate the extent of the fracture. Usually there is little displacement of the fracture line, but, depending on the time since the fracture, there may be periosteal new bone formation around the fracture site.
- It is difficult to eliminate the lameness with standard nerve blocks, but infusion of 2 to 3 mL of 2% prilocaine (Treatment No. 93) subcutaneously around the site of the fracture usually results in improvement in the gait.
- If the fracture has been present for several weeks, there will be new bone production around the fracture site. The main differential diagnosis is "splints."

DIFFERENTIAL DIAGNOSIS

- Splints
- Suspensory ligament sprain
- Condylar fracture of the third metacarpal bone
- Osteomyelitis of the small metacarpal or metatarsal bones

TREATMENT

- Most fracture of the splint bones will heal in 3 to 6 months. However, there is usually quite a

lot of callus formation around the fracture site, which may be unsightly, particularly in a show horse, and in performance horses may interfere with the suspensory ligament.
- Fractures of the distal two-thirds of the bone are best treated with amputation of the bone. Amputation of greater than two-thirds of the bone can result in instability of the remaining splint bone.
- The splint bone and its periosteum is removed just above the "fracture site using an osteotome positioned so that the proximal part of the splint bone is tapered at the removal site. Care must be taken to remove any remaining damaged periosteum. More proximal fractures should be repaired with small bone plates and 3.5-mm screws. If osteomyelitis is established, amputation can be performed, but the remaining proximal splint bone must be stabilized with a bone plate. Simple screw fixation to the proximal metacarpus or metatarsus has proven unsuccessful with screws often breaking. Successful removal of the whole fourth metatarsal bone has been reported and should be considered in severe cases.

KEY POINT

The best cosmetic results are obtained by attempting to reduce the swelling after surgery.

- This is done using pressure bandaging for the first 2 weeks after surgery together with oral phenylbutazone administration (Treatment No. 88). The dose rate of phenylbutazone is 4.4 mg/kg for the first 24 hours, followed by 2.2 mg/kg for 2 to 3 days, and then 2.2 mg/kg q24h for 4 days. A total of 8 weeks of rest usually is required following surgery, before the horse returns to exercise.

Rupture of the Suspensory Apparatus

Traumatic rupture of the suspensory apparatus may occur with or without fractures of both the proximal sesamoid bones and results in a loss of support for the fetlock. It usually occurs as a racing injury and needs immediate and appropriate attention. Treatment should be regarded as a salvage measure and affected horses will not return to athletic competition.

HISTORY AND PRESENTING SIGNS

- Acute, severe lameness, usually of the foreleg
- Lameness usually occurs after racing or after fast training exercise
- Sometimes found in foals as a result of jumping off a ridge in the pasture

CLINICAL FINDINGS AND DIAGNOSIS

- Severe overextension of the fetlock is the characteristic feature of rupture of the suspensory apparatus.
- With a loss of support for the fetlock, the palmar aspect of the fetlock sinks toward the ground when the horse attempts to put weight on the leg.
- Radiographs, a minimum of four views (dorsopalmar, lateral to medial, and two obliques), should be taken to determine if the sesamoid bones have been fractured. In some cases, luxation of the fetlock joint occurs.
- Diagnosis is not difficult because of the extreme nature of the fetlock appearance when the horse attempts to bear weight on the affected leg.

DIFFERENTIAL DIAGNOSIS

- Fetlock luxation
- Weak flexor tendons (after casting or in newborn foals)

TREATMENT

KEY POINT

It is vital to stabilize the limb to prevent further trauma and damage to the blood supply to the affected leg.

- Stabilization is best achieved by splinting or casting the leg (see Chapter 19) with the fetlock in flexion.
- There is no athletic future for a horse with rupture of the suspensory apparatus. However, such horses can be made paddock sound by arthrodesis of the fetlock joint. This surgery is very complex and should only be undertaken at a specialist referral center.

Splints

"Splints" is a lay term used to describe an osteitis and periostitis of the splint bones, notably the second metacarpal bones. This results in localized swelling and new bone production. The cause in many cases is abnormal conformation, with the metacarpal bones being set too far to the lateral side of the carpus. This results in excessive weight being placed on the second metacarpal bone, which causes movement and local periostitis. Other cases may be the result of direct trauma to the metacarpus.

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HISTORY AND PRESENTING SIGNS

- Swelling, usually over the medial aspect of the metacarpus
- Little or no lameness
- May affect any breed of horse
- Most commonly found in younger horses (2 to 5 years old)

CLINICAL FINDINGS AND DIAGNOSIS

KEY POINT

Usually, lameness is only evident in the acute stage of splint formation and lameness often is wrongly attributed to splints.

- If there is marked or persistent lameness associated with the swelling, it is more likely to be a fracture of the splint bone.
- The most obvious clinical sign is swelling over the splint bone. During the acute stages of the reaction, this swelling may be diffuse, whereas later the swelling will be localized and will vary in size from a marble to a golf ball. Care should be taken in excluding other areas of the leg before lameness is ascribed to a splint.
- Radiographs may be necessary to exclude a fracture of the second metacarpal bone. The most important radiographic view is usually the 45-degree oblique view. This view shows the outline of the splint bone, and the extent of soft tissue versus new bone growth can be assessed accurately. Because the junction between the splint bone and third metacarpal or metatarsal bones can appear as an oblique radiolucent line through the body of the second metacarpal bone, a fracture may be diagnosed mistakenly. It may be important to take several oblique views to ensure that a radiolucent line is really a fracture line and not the line of junction between a small and large metacarpal or metatarsal bone.

DIFFERENTIAL DIAGNOSIS

- Fracture of the small metacarpal bones
- Suspensory ligament sprain
- Soft-tissue trauma to the metacarpus

TREATMENT

- During the very early stages, before new bone growth has occurred, local anti-inflammatory measures (cold hosing, bandaging) together with injection of 20 to 40 mg of a long-acting corticosteroid such as methylprednisolone acetate (Treatment No. 74) subperiosteal^ will reduce the swelling and limit the extent of new bone growth.
- Anti-inflammatory drugs such as phenylbutazone (Treatment No. 88) also may be used orally for 10 to 14 days. The initial dose rate is 4.4 mg/kg q24h for 1 to 2 days, followed by 2.2 mg/kg ql2h for 3 to 5 days, and thereafter 2.2 mg/kg q24h for up to 10 days.
- Radiation therapy also has been used with some success. However, because of stimulation of local blood supply, if further trauma occurs, there may be more profuse new periosteal bone growth.
- A variety of iodine-based blistering agents and firing have been used as counterirritants in attempts to resolve the swelling associated with the splint. These procedures do not improve the result and have no place in therapy.

KEY POINT

Once new bone formation has occurred, the splint will seldom cause the horse any problem, apart from being a cosmetic blemish.

- At this stage, only surgical removal of the new bone growth will resolve the problem. However, there is a high chance of recurrence of the bone formation. The best cosmetic result is achieved by removing the periosteum with the new bone. Pressure bandaging for at least 2 weeks after surgery is important, after which a lighter bandage should be applied for a further 2 weeks. During the first 2 weeks after surgery, phenylbutazone (Treatment No. 88) should be given orally at a dose rate of 4.4 mg/kg for the first 24 hours, followed by 2.2 mg/kg ql2h for 5 days, and then 2.2 mg/kg daily for 5 days. This helps to reduce swelling and prevent new periosteal bone formation following surgery.

Suspensory (Interosseous) Ligament Desmitis

The suspensory (interosseous) ligament is part of the suspensory apparatus and has its origin in the proximal palmar metacarpus or metatarsus. It divides approximately 5 to 7.5 cm (3 inches) above the fetlock joint, and the medial and lateral branches attach to the abaxial surfaces of the sesamoid bones. Suspensory desmitis can affect the body, branches, or origin of the ligament.

HISTORY AND PRESENTING SIGNS

- Swelling in the middle to distal metacarpus
- Most common in racehorses, particularly Standardbred pacers and trotters, and other athletic horses
- Mild or no lameness

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CLINICAL FINDINGS AND DIAGNOSIS

- There may or may not be signs of lameness, depending on the severity of the sprain. The lameness is usually low grade, and horses are often presented because of swelling rather than lameness.
- There is usually swelling of the affected area of the suspensory ligament. There is also pain on gentle palpation of the affected area of the suspensory ligament. (Note that firm palpation will result in a painful response in most normal horses.) In long-standing cases, there may be gross enlargement of the ligament due to scar-tissue formation at the site of injury.
- Because the proximal suspensory ligament lies between the splint bones, swelling may be difficult to determine. Horses with proximal suspensory desmitis often present with lameness only. Localizing the cause of the lameness to the proximal suspensory ligament is achieved by local analgesia of the palmar metacarpal nerves at the proximal metacarpus/metatarsus.

KEY POINT

Note that the sesamoid bones are often involved secondarily, owing to tearing of the insertion of the ligament and increased remodeling due to high repetitive loads.

- Additionally, it is relatively common to find fractures of the second metacarpal bone in many Standardbred horses with suspensory ligament desmitis. It is essential, therefore, to obtain radiographs (particularly oblique views of the fetlock and metacarpus) before deciding on the eventual treatment. Radiographic changes associated with proximal suspensory desmitis include sclerosis of the palmar/plantar cortex of the third metacarpal/metatarsal bone, enthesiophytes and avulsion fractures.
- Ultrasound examination is essential to determine the extent of damage to the suspensory ligament. Repeated examinations assist in indicating the course of healing, which helps in deciding when the horse should be returned to training. Ultrasound is also used to confirm a proximal suspensory desmitis. There is a normal area of hypoechogenicity in the proximal suspensory ligament, which makes interpretation difficult. Comparison with the contralateral limb is helpful. Changes in contour of the palmar/plantar surface of the metatarsal/metacarpal bone also may be observed due to enthesiophytes or avulsion fractures.

DIFFERENTIAL DIAGNOSIS

- Fracture of the small metacarpal bones
- Splints
- Deep flexor tendon strain
- Sesamoiditis

TREATMENT

- Initial rest followed by a controlled exercise program for up to 9 to 12 months may be required. Repeated ultrasound examinations at regular intervals will indicate the course of healing of the lesion in the ligament.
- If there is a fracture of the distal part of the second metacarpal bone, this results in continued irritation to the ligament, and therefore the fractured portion of bone should be surgically removed.
- In the acute phase of the injury, local anti-inflammatory measures (cold hosing, cold packs, pressure bandaging) together with anti-inflammatory drugs such as phenylbutazone (Treatment No. 88) for 10 to 14 days, will help reduce the swelling and pain. Oral administration at a dose rate of 4.4 mg/kg for the first 24 hours, followed by 2.2 mg/kg q24h for 3 to 5 days, and then 2.2 mg/kg daily for up to 5 days is usual.
- A number of different surgical treatments have been tried, none of which have proved very successful. Rest for between 6 and 12 months is the key, followed by a graded exercise program to increase the strength of the soft tissues when returning the horse to training.

Tendon Strain ("Bowed Tendon")

The deep and superficial flexor tendons are both prone to injury because of the forces placed on them by galloping horses.

KEY POINT

The superficial flexor tendon is far more commonly injured than the deep flexor tendon, and the most frequent site for injury is the midmetacarpal region.

The reason for the injuries being most common in the midmetacarpal region may be because the cross-sectional area is the smallest in this part of the tendon. Tendon strains are uncommon in the hindlegs. The expression "bowed tendon" is used by owners and trainers because of the characteristic swelling at the palmar aspect of the midmetacarpus.

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HISTORY AND PRESENTING SIGNS

- Variable degrees of swelling in the palmar aspect of the midmetacarpus
- Early lesions show no or mild lameness
- Most commonly found in racehorses (Quarter horses, Standardbreds, and Thoroughbreds) and other horses used for high-intensity exercise

CLINICAL FINDINGS AND DIAGNOSIS

- The first sign of injury is swelling in the affected area of the tendon, together with heat and pain on palpation. Initially, the degree of swelling may be quite mild and may be ignored by the owner or trainer because the horse is not lame.
- In mild injuries there is usually no lameness, but in severe tendon strains there may be a mild to moderate foreleg lameness.
- Examination of the tendons should be performed with the weight off the affected leg, because the horse may show no signs of pain on palpation if examined when bearing weight. With weight off the leg, each of the tendons can be palpated, progressing 1 cm (0.5 inch) at a time, to localize the area where swelling and pain are present.
- In chronic cases, there is usually considerable thickening of the tendon, with an increase of up to four to five times in area, due to scar-tissue formation. Even in long-standing cases, it is possible to detect pain on palpation. A useful guide to tendon healing is gained from palpation of the affected site to determine if pain still is present in the area. If there is pain on palpation, exercise should be restricted to slow training only.
- Ultrasound examination is important to assess the extent of the tendon injury and will distinguish swelling in the peritendinous tissue from that within the tendon fibers. The severity of the lesion is determined by the degree of hypoechogenicity, the proportion of the cross-sectional area affected, and the length of the lesion. Significant changes in ultrasound appearance can occur during the acute stage so repeat ultrasound examinations should be performed at 10 to 14 days after the initial injury. Ultrasound is also essential for monitoring the rate of healing, giving a guide as to when the level of exercise can be increased.

DIFFERENTIAL DIAGNOSIS

- Suspensory ligament desmitis
- Soft-tissue trauma around flexor tendons
- Palmar annular ligament constriction
- Tendon sheath infection

TREATMENT

KEY POINT

The limiting factor to any form of treatment is that when a tendon is injured, healing is very slow, and the normal type I collagen, which is very strong, is replaced by type III collagen, which has poor tensile strength. Additionally, the normal longitudinal orientation of the tendon fibers is lost, so some of the mechanical properties of the tendon are altered.

- Initial treatment is aimed at controlling the inflammatory response, reducing the swelling and preventing further injury. The horse should have box rest, local application of cold packs, systemic anti-inflammatories, and carefully applied pressure bandaging.
- When the initial inflammatory reaction has resolved, the tendon will benefit from controlled exercise. The amount of exercise is difficult to determine and care must be taken because too much exercise too soon is detrimental. The best approach is to begin with short periods of walking exercise, increasing the duration every 2 weeks while monitoring the tendon for heat and swelling. Trotting exercise may begin at 3 months provided that the tendon does not increase in cross-sectional area or its appearance shows signs of deterioration on ultrasound examination. The duration and intensity of exercise gradually is increased, using monitoring via ultrasound examination of the tendon, before increasing the speed of exercise. Using this approach, it is rare for horses to resume full work before 12 months after the tendon injury, but the duration varies considerably depending on the severity of the lesion and the individual horse's ability to heal.

Other treatments that may have benefit include the following:

Tendon Stab or Tendon Splitting. This surgical procedure originated in Sweden and consists of a number of stabs being made into the tendon or the tendon being split longitudinally. The aim of this surgery was to promote vascularity to the damaged area of tendon. Older experimental evidence in normal tendons suggests that the procedure is of limited value and may actually delay tendon healing. More recently, there has been some ultrasound evidence that in horses with severe core tendon lesions, stabs into the area of the damaged tendon can help to release pressure from the local hematoma and may lead to more rapid healing, particularly when combined with superior check ligament desmotomy.

Injection of Agents to Modify Healing. *A* number of local therapeutic agents have been advocated to improve the healing and ultimate strength of the tendon. These include hyaluronate, PSGAGs, and more recently beta-aminoproprionitrile (BAPN). There is little evidence that these agents actually improve the ultimate outcome, but the preliminary results with BAPN, an agent that encourages collagen cross-linking, are encouraging. Some studies have shown that sodium hyaluronate (Treatment No. 60) injected around a damaged tendon may result in fewer adhesions and more rapid return to function, but there is no evidence that this improves long-term outcome.

Superior Check Ligament Desmotomy. The superior check ligament is sectioned adjacent to its insertion on the distal radius. The aim of this surgery is to increase the elasticity of the superficial digital flexor muscle-tendon unit. However, biomechanical tests in cadaver limbs do not support this theory. There is some evidence that the ultrasonic appearance of the lesion improves more rapidly after surgery. Follow-up studies in horses that have undergone superior check ligament desmotomy have shown that between 50 and 80% of horses will race five times without reinjury of the tendon.

Conclusions

Despite the various treatments used, there is a high rate of recurrence of the tendon injuries. The most effective treatment for superficial tendon strain appears to be superior check ligament desmotomy and local tendon stabs when there is a significant core lesion, followed up by a graded exercise program in the convalescent period. There are some differences in prognosis between various classes of performance horses.

Tendon Transection

Complete rupture of tendons is very rare in the horse. However, transection of tendons is quite common as a result of trauma to the metacarpal region. This can occur as a result of having limbs caught in fences, trauma from exposed areas of sharp iron around barns or box stalls, wire cuts, and other similar traumas. The prognosis for extensor tendon transection is better than for flexor tendon transection.

HISTORY AND PRESENTING SIGNS

- Trauma to the limb
- Hyperextension of the fetlock
- Severe non-weightbearing lameness

CLINICAL FINDINGS AND DIAGNOSIS

- Transection of extensor tendons will cause knuckling of the fetlock when the horse attempts to bear weight.
- If both superficial and deep flexor tendons are transected, the fetlock joint will drop as a result of the lack of support, and the toe will rock proximally.
- In some cases, the transection will be obvious, particularly if there is extensive skin laceration. However, sometimes there is transection with minimal skin trauma, particularly if the transection is due to tin on barns. In such cases, the presenting signs may be similar to those in horses with rupture of the suspensory apparatus.

DIFFERENTIAL DIAGNOSIS

- Rupture of the suspensory apparatus
- Fracture of the proximal sesamoid bones
- Fracture of the metacarpus

TREATMENT

- Suturing of transected flexor tendon ends may improve the ultimate outcome, but due to extensive necrosis of tendon ends and contamination of the site, it is not often practical. Initially, the area should be thoroughly debrided and lavaged. If there is minimal contamination and tendon necrosis, the tendon ends can be sutured using heavy-duty PDS, in a compound locking loop pattern for the superficial digital flexor tendon, and a tendon pulley suture for the rounder deep digital flexor tendon. The limb should then be cast, preferably for 12 weeks.
- Where there is marked contamination of the wound, delayed suturing may be considered after casting for 7 to 10 days and broad-spectrum bactericidal antibiotic therapy. This is particularly important for tendons transected within tendon sheaths. If suturing is not possible, healing is generally adequate in extrasynovial locations.
- Casts in the forelimb should extend to just below the carpus, whereas in the hindlimb they should extend to the proximal tibia to prevent hock flexion. When the cast is removed, a heel extension shoe should be applied to reduce tension on the tendons.
- Severed extensor tendons rarely require suturing. A toe extension shoe may be required to prevent knuckling of the fetlock but most horses learn to walk normally in a few days. Less than 50% of horses with flexor tendon transection return to their previous use, whereas 75% ath-

letic soundness has been reported for extensor tendon transection.

Inferior Check Ligament Desmitis

The inferior check ligament is approximately the same width as the deep flexor tendon and runs from its origin on the palmar surface of the distal carpal bones to join the deep flexor tendon in the midmetacarpal region. Inferior check ligament desmitis is an uncommon cause of lameness, and horses used for jumping and Standardbred pacers appear to be predisposed.

HISTORY AND PRESENTING SIGNS

- Localized swelling in the proximal metacarpal area
- Lameness that can be localized to the proximal palmar metacarpus

CLINICAL FINDINGS AND DIAGNOSIS

- Mild foreleg lameness
- Clinical examination of the leg usually reveals pain and swelling in the region of the check ligament.
- The diagnosis is confirmed by ultrasonography. There is generally enlargement of the ligament and a diffuse decrease in echogenicity. Adhesions to the superficial digital flexor tendon at the lateral and medial margins of the ligament may also be present.

DIFFERENTIAL DIAGNOSIS

- Deep flexor tendon strain
- Suspensory ligament desmitis
- Fractures and osteitis of the small metacarpal (splint) bones

TREATMENT

- Treatment involves initial stall rest and anti-inflammatory therapy for 10 days to 2 weeks, followed by a controlled exercise program. Lameness generally resolves before the ultrasonic appearance returns to normal. Six to eight months should elapse before the horse is returned to full exercise.
- Approximately 70% of horses will return to full exercise without recurrence of the desmitis.

Tendon Problems in Young Horses

Two types of tendon problems occur in young horses. The first is *weak flexor tendons* in newborn foals, associated with dysmaturity. The second is *flexure deformity,* which may be a congenital condition or can be acquired in older foals and yearlings. The cause of acquired flexural deformities is not always clear but in many cases is secondary to a painful condition in the affected limb resulting in decreased weight bearing.

HISTORY AND PRESENTING SIGNS

- Most commonly found in newborn foals, although flexure deformities may develop in weanlings and yearlings.
- Obvious change in the contour of the legs. *Weak flexor tendons* are usually found in the hindlegs of newborn foals, with overextension of the limbs. *Flexure deformities* result in the foal or weanling standing very upright, usually in the forelegs, and in severe cases there may be an inability to stand in young foals or knuckling over on the dorsal surface of the hoof wall because of the degree of flexion in older horses.

CLINICAL FINDINGS AND DIAGNOSIS

- *Weak flexor tendons* are easily diagnosed because when the foal stands, the plantar aspect of the fetlock will descend toward the ground as a result of overextension.
- With *flexure deformity,* the pastern and fetlock will be very upright, and in severely affected cases, the foal may actually knuckle over on the dorsal fetlock because of overflexion. The foal will be unable to place the affected feet flat on the ground and usually bears weight on the toe.
- With the foal in lateral recumbency, the distal joints should be flexed and extended to ensure that a normal range of movement exists.

DIFFERENTIAL DIAGNOSIS

- Arthrogryposis
- Neurologic disorders

TREATMENT

KEY POINT

Weak flexor tendons will self-correct if the foal is confined to a box stall with its mother for 3 to 4 days.

- Light bandages may be applied to protect the plantar or palmar aspect of the affected fetlocks. These should not provide support as this will slow correction of the deformity.
- In cases that are slow to respond, a heel exten-

sion shoe should be applied. These can be either a commercially available glue-on shoe or can be made with plywood or something similar. These usually can be fixed in place with elastic adhesive bandage.

- Many cases of newborn foals with flexure deformity also will respond well to stall rest, provided the foals can stand. More severe cases may respond to a single intravenous dose of oxytetracycline. A dose rate of 44 mg/kg, diluted in 1 L polyionic fluids and given slowly IV once, has been suggested and remarkable results are often found.

KEY POINT

All newborn foals with flexural deformities should have their immunoglobulin levels checked because they may not consume sufficient colostrum due to their difficulties in walking.

- If a newborn foal cannot stand and fails to respond to oxytetracycline, surgery may be attempted. The prognosis depends on the severity of the deformity. An inferior check ligament desmotomy is rarely effective in these cases, and deep digital flexor tenotomy may need to be performed. In some cases, sectioning of all the palmar tendons and ligaments will not allow straightening of the limb.
- In older foals with acquired flexural deformities, anti-inflammatory therapy in the acute stage may allow relaxation of the tendon. If a rapid response is not achieved or in chronic cases, surgery should be performed. For flexural deformity of the coffin joint, inferior check ligament desmotomy should be performed with application of a toe extension shoe. For metacarpophalangeal joint deformity, inferior check ligament desmotomy should be combined with splinting of the fetlock. Flexural deformity of the carpus has been successfully treated with sectioning of the tendon of the ulnaris lateralis.

Tendon Sheath Infections

Tendon sheaths provide lubrication to tendons where there are changes in tendon direction as they pass over joints. Infection of a tendon sheath is difficult to treat successfully due to the confined space, the difficulty in establishing drainage, and persistence of infection in the synovial membrane. Even if the infection is resolved, lameness may persist due to adhesion formation. Infection of the digital sheath that extends from the distal third of the metacarpus to the bulbs of the heels is the most serious.

HISTORY AND PRESENTING SIGNS

- An acute traumatic incident is usual due to a cut or penetrating injury in the area of one of the tendon sheaths.

KEY POINT

Acute severe lameness is usual, with the horse reluctant to bear weight on the affected leg, together with gross swelling of the affected

CLINICAL FINDINGS AND DIAGNOSIS

- Heat and swelling around the affected area of the leg, with distention of the tendon sheath.
- In the early stages of a tendon sheath infection, there may be an increase in rectal temperature and an increase in heart and respiratory rates.
- Aspiration of the swollen tendon sheaths should be performed aseptically, after surgical preparation of the area. Fluid should be collected into tubes containing EDTA for cytologic examination and measurement of total protein. In normal tendon sheath fluid, there are few cells (<1000 X 10^6 L), and total protein values are less than 25 g/L or 2.5 g/dL. Fluid also should be collected into sterile tubes for bacteriologic examination using similar techniques to those for joint fluid (see Chapter 17).

DIFFERENTIAL DIAGNOSIS

- Septic arthritis
- Infection of navicular bursa
- Fracture of the phalanges

TREATMENT

- Systemic broad-spectrum bactericidal antibiotic therapy should be commenced as soon as samples have been obtained for culture and sensitivity. Once the results of bacteriology are known, the antibiotic therapy can be amended appropriately.
- Irrigation of the tendon sheath with an isotonic polyionic solution should be performed under general anesthesia. Approximately 2 to 4 L should be flushed through the tendon sheath using large-gauge (12- or 14-gauge) catheters. Tenoscopy allows visualization of the sheath as well, and fibrin clots can be removed. When flushing is complete, local antibiotics can be injected into the sheath. Gentamicin is the most commonly used (150-250 mg). This can be repeated daily.
- Phenylbutazone should be administered initially

at 4.4 mg/kg ql2h to allow walking exercise to limit adhesion formation.
- In many cases it is difficult to culture bacteria, particularly if antibiotic therapy has been instituted before samples are taken for bacteriology. The prognosis is guarded for successful resolution of the problem, generally due to the development of adhesions within the sheath.

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PROBLEMS OF THE CARPUS AND DISTAL RADIUS

Carpitis

Carpitis is an all-encompassing term for lameness associated with the carpus. It includes conditions such as osteoarthritis, synovitis and capsulitis, desmitis of the carpal ligaments, subchondral bone sclerosis, and lysis and periostitis of the bones of the carpus. Most of these conditions are due to fatigue failure of the bone or soft-tissue structures of the joint due to repetitive high loading. Occasionally, direct trauma to the carpus is a cause.

HISTORY AND PRESENTING SIGNS

- Lameness and/or swelling involving the dorsal aspect of the carpus.
- History may be either of acute or chronic lameness depending on the stage of the condition.
- In some cases with trauma to the soft tissues overlying the carpal bones, the horse may be presented with swelling over the dorsal carpus but no significant lameness.

CLINICAL FINDINGS AND DIAGNOSIS

- The most obvious clinical sign is swelling of the dorsal aspect of the carpus and, perhaps, distention of the involved joint capsules.
- Most horses in the acute stage are lame, but in chronic cases there may be localized swelling without any signs of lameness.
- On examination of the carpus, there may be pain on palpation over the affected carpal bones (particularly in the acute stage), and pain on flexion of the carpus is a consistent finding.
- If lameness is present, intra-articular nerve blocks should be performed to determine whether the lameness can be localized to the antebrachiocarpal or midcarpal joints. Although the carpometacarpal joint communicates with the midcarpal joint, primary disease of the carpometacarpal joint is unusual.
- A radiographic survey of the carpus is essential, and at least two oblique views and a flexed lateral view are required. Because the clinical signs are similar to those in horses with chip fractures of the carpal bones, it is important to exclude such fractures.
- If there is any doubt from the initial radiographs, additional oblique and skyline views should be taken.

DIFFERENTIAL DIAGNOSIS

- Chip fractures of the carpal bones
- Soft-tissue trauma to dorsal aspect of the carpus
- Extensor tendon sheath synovitis
- Hygroma of the carpus

TREATMENT

- It is important that an accurate diagnosis be made before treatment and, in particular, that no chip fractures are present. If radiographs reveal only soft-tissue swelling and no periosteal reaction, local anti-inflammatory measures (cold hosing, application of cold packs and pressure bandages) and/or topical application of DMSO (Treatment No. 34) together with a 7- to 10-day course of phenylbutazone (Treatment No. 88) should be used.
- If the lameness fails to respond to topical and systemic therapy, intra-articular therapy is required (see Treatment under Degenerative Joint Disease). Early cases may respond to hyaluronate (Treatment No. 60). In more severe or

chronic cases, intra-articular corticosteroids (Treatment Nos. 12 and 74) can be useful.

Tenosynovitis of the Carpal Sheath

Tenosynovitis of the carpal sheath is an occasional cause of lameness in all types of horses. Tenosynovitis may be secondary to damage to any of the tendinous structures within the sheath. These include the deep digital flexor tendon, superficial digital flexor tendon, and the superior check ligament. Other possible causes are osteochondroma of the caudal distal radius, fractured accessory carpal bone, or direct trauma. As with the annular ligament, the caudal carpal retinaculum forms an inelastic canal with the carpal bones.

HISTORY AND PRESENTING SIGNS

- Low-grade to moderate forelimb lameness
- Swelling on the lateral aspect of the distal antebrachium and lateral and medial aspects of the proximal metacarpal area.

CLINICAL FINDINGS AND DIAGNOSIS

- There is lameness of the affected limb with pain on flexion of the carpus and an absence of lesions on the dorsal aspect of the carpal bones.
- The most consistent finding is fluid distention of the carpal sheath on the lateral aspect immediately proximal to the accessory carpal bone.
- Radiographs should include lateral and flexed lateral and oblique views. An osteochondroma will appear as an osseous density on the distal caudal aspect of the radius.
- An ultrasound examination of the carpal sheath should be performed to assess the tendons and superior check ligament and will also allow assessment of synovial membrane swelling and adhesion formation.

DIFFERENTIAL DIAGNOSIS

- Fracture of the accessory carpal bone
- Synovitis or desmitis of the flexor tendons proximally at the palmar aspect of the carpus

TREATMENT

- Primary tendonitis is best treated with anti-inflammatory therapy and rest, followed by a controlled exercise program (see Tendon Strain). Fractures of the accessory carpal bone usually heal with a fibrous union, and tenosynovitis generally resolves. Osteochondromas are treated by resection of the mass. Small masses can be removed arthroscopically using a lateral approach. Larger masses may require opening of the sheath on the lateral aspect, proximal to the accessory carpal bone. The osteochondroma is removed with an osteotome.
- In cases with a primary tenosynovitis, topical DMSO and systemic phenylbutazone may resolve the inflammation in acute cases. Chronic cases will require intrathecal corticosteroids. In cases that do not respond and where constriction of the carpal canal is suspected, sectioning of the palmar retinaculum is recommended.

Carpal Fractures ("Knee Chips")

Fractures of various carpal bones are common injuries in racehorses and are generally fatigue fractures due to repetitive high loading during fast exercise.

KEY POINT

Most fractures are small chip fractures on the dorsal aspect of the carpus, but slab fractures (particularly of the third carpal bone) also are found.

The most common sites for carpal fractures are the dorsal distal articular margin of the radial carpal bone, the dorsomedial proximal articular margin of the third carpal bone, and the dorsolateral aspect of the distal articular margin of the radius. The intermediate carpal, ulnar carpal, and accessory carpal bones are sometimes fractured. However, fractures of the second and fourth carpal bones are extremely unusual.

HISTORY AND PRESENTING SIGNS

- Most carpal fractures occur in Thoroughbred racehorses.
- Fractures involving the midcarpal joint are mostly on the dorsomedial aspect, whereas those involving the antebrachiocarpal (proximal carpal) joint are usually on the dorsolateral aspect.

CLINICAL FINDINGS AND DIAGNOSIS

- Lameness with most chip fractures is mild. Moderate or severe lameness is suggestive of more extensive fractures.
- Distention of the joint capsule is apparent over the dorsal aspect of the affected carpus within 3 to 6 hours after the injury. This distension often appears like a small bubble that is localized to the dorsal joint capsule over the affected joint. This localized joint capsule swelling is an im-

portant clinical sign of intra-articular pathology of the carpus, and therefore, the joint capsules of the antebrachiocarpal and midcarpal joints should always be palpated carefully when performing a lameness examination.
- If there is a slab fracture of the third carpal bone, the horse will be reluctant to bear any weight on the leg, and the carpus will be considerably swollen. Pain on flexion of the carpus is found, but if the fracture is long-standing, pain may be difficult to demonstrate. In these cases, extreme flexion of the carpus will be necessary to demonstrate a painful response. In long-standing cases, intra-articular local anesthesia may be required to confirm that the carpus is the site of lameness. Good radiographs are essential and should include at least a flexed lateral, two oblique views, and a skyline view of the third carpal bone.

KEY POINT

Incomplete or undisplaced slab fractures and sagittal fractures of the third carpal bone may be seen only on a skyline view.

Radiographs do not always reveal a discrete fracture. Areas of subchondral bone lysis on the dorsal distal aspect of the radiocarpal bone also are suggestive of a chip fracture, in many cases.

DIFFERENTIAL DIAGNOSIS

- Extensor tendon sheath synovitis
- Carpitis
- Soft-tissue trauma to the carpus
- Nonspecific carpal lameness

TREATMENT

- Although surgery to remove the chip fracture is the most logical treatment, this may not always carry with it a good prognosis for further racing. Careful assessment of radiographs to determine if any arthritis is present, the size of the chip, and the extent of articular involvement is important. These factors allow advice to be given to the client regarding the prognosis for future athletic function.
- If only a small chip fracture is present and the fracture is undisplaced, there is the possibility that 6 months of rest will enable successful healing and return to racing. However, because of continued irritation to the joint from the presence of the chip, it is best in most cases to remove the fracture via arthroscopy. Arthroscopy allows a detailed examination of the joint. There will be cases where the subchondral bone damage is far more extensive than the radiographs suggest. This is particularly the case for the distal radiocarpal bone.
- Postoperatively, the horse's carpus remains bandaged for 7 to 10 days. Usually only a light bandage is required, with one bandage change in the week after surgery. The horse should be confined to a box stall for the first 4 weeks after surgery, followed by a larger yard for an additional 4 weeks, before being turned out to pasture. The degree of rest required after surgery will show great variation. Most horses require 4 to 6 months' rest from training. The prognosis depends on the extent of subchondral bone damage.
- If there is a slab fracture of the third carpal bone, internal fixation with a cortical bone screw is required. In many cases, this can be accomplished using a stab incision over the dorsal aspect of the third carpal bone and visualization of the fracture line using the arthroscope. A cortical bone screw, usually 4.5 mm in diameter, is inserted using the principle of lag-screw fixation.
- Fractures of the accessory carpal bone are best treated conservatively with 6 to 9 months of rest.
- Internal fixation of sagittal fractures of the third carpal bone has been described but is rarely necessary as most are quite stable. Arthroscopy should be performed to debride the fracture margins because there is often extensive subchondral bone lysis and fragmentation at the fracture line. The horse should then be stall rested for 6 to 8 weeks followed by 4 to 6 months of rest. The prognosis depends on the extent of subchondral bone damage.

Physitis

Physitis is a condition involving the growth plates of various bones of young horses. It is thought to be caused by excessive forces on the physis, due to excessive exercise and weight, or due to structurally deficient bone that fails under normal loading. It mainly affects the distal radial physis in weanlings and yearlings.

HISTORY AND PRESENTING SIGNS

- Commonly seen in foals and weanlings from 4 to 5 months of age.
- Some cases are found in Thoroughbred yearlings during early phases of training.
- Swellings around the distal radial growth plate

and the distal metacarpal growth plates are the most common presenting sign.
- In some cases, horses may be presented because of lameness. However, this is uncommon.

CLINICAL FINDINGS AND DIAGNOSIS

- Swelling at the site of the growth plate in the distal radius or metacarpus is the most obvious sign. This swelling is usually bilateral.
- There may be pain on palpation over the affected growth plates.
- Radiographs will show a variety of changes in the region of the epiphysis and metaphysis. Most common findings include flaring of the metaphysis, sclerosis of bone immediately adjacent to the physis, and in some cases an irregular and lytic appearance of the physis.

DIFFERENTIAL DIAGNOSIS

- Carpitis
- Carpal chip fractures
- Extensor tendon sheath synovitis
- Trauma to the distal radius
- Carpal valgus deformity (may coexist with epiphysitis)

TREATMENT

- Exercise must be restricted and the weight of the foal reduced to decrease loading on the physis. Analysis of the diet is important and may reveal inadequate calcium or a calcium-phosphorus ratio that is too low. Computer programs such as the *Equine Nutritionist* (Nsquared, Silverton, Oregon) are available and allow comprehensive dietary analyses, comparing results with recommended National Research Council values.
- Correction of this relative calcium deficiency using calcium carbonate or decreasing the amount of grain in the diet may be useful.
- A course of anabolic steroids has been recommended in some horses to hasten closure of the physes.

Hygroma of the Carpus

Hygroma of the carpus is a large fluid-filled subcutaneous swelling on the dorsal aspect of the carpus, usually resulting from direct trauma. Although unsightly, it rarely causes lameness.

KEY POINT

Carpal hygroma rarely causes significant lameness.

HISTORY AND PRESENTING SIGNS

- Usually found in showjumping and eventing horses
- History of trauma to the dorsal carpus and distal radius
- Considerable swelling of the carpal region without significant lameness

CLINICAL FINDINGS AND DIAGNOSIS

- A diffuse fluid-filled swelling localized to the dorsal carpus.
- Usually there is no pain on palpation or flexion of the carpus, although invariably there is some restriction to the degree of flexion.
- Radiographs should be taken to ensure that there is no involvement of the carpal bones. In some cases there may be a coexisting carpitis, with evidence of new periosteal bone formation. Contrast media should be injected to differentiate hygroma from synovial fistula or synovial hernia.

DIFFERENTIAL DIAGNOSIS

- Extensor tendon sheath synovitis
- Synovial hernia
- Synovial fistula
- Carpitis
- Carpal fractures
- Trauma to the medial tuberosity of the distal radius

TREATMENT

KEY POINT

Whatever treatment is used, some residual swelling will remain. In acute cases drainage of fluid, injection of corticosteroids and pressure bandaging may be successful. In chronic cases, surgical drainage of the fluid, insertion of a Penrose drain, and application of a pressure bandage is needed.

- The Penrose drain is inserted via two stab incisions at the most proximal and distal aspects of the fluid swelling. The drains are fixed in place with a single suture in the skin, and a sterile dressing is applied.
- The Penrose drain should remain in place for 5 to 7 days and is removed when there is no evidence of fluid discharge from the distal drainage site. Initially, a daily bandage change is required, but after 2 to 3 days, this can be every other day if the amount of fluid discharge has reduced.

- Synovial fistula and synovial hernia require surgical closure of the defect in the capsule for resolution.

Medial Palmar Intercarpal Ligament Tearing

The dorsal part of the medial palmar intercarpal ligament can be observed during arthroscopy of the midcarpal joint. It runs from the lateral aspect of the radial carpal bone to the second and third carpal bones. Postmortem studies reveal some tearing of the ligament in up to 90% of joints, but horses with greater than 50% of the dorsal part of the ligament torn have significantly worse postoperative performance than those with less tearing.

HISTORY AND PRESENTING SIGNS

- Low-grade forelimb lameness
- May have responded to an intra-articular block of the midcarpal joint on a previous occasion

CLINICAL FINDINGS AND DIAGNOSIS

- Lameness localized to the midcarpal joint. There may or may not be joint effusion.
- Lameness responds to intra-articular anesthesia of the midcarpal joint.
- Radiographic changes vary from mild subchondral bone damage on the dorsal radial carpal bone to occasionally more severe damage.

DIFFERENTIAL DIAGNOSIS

- Occult intra-articular fractures
- Synovitis
- Degenerative joint disease

TREATMENT

- There is a poor prognosis for horses with extensive tears.
- Debridement of the torn fibers has been advocated, but the long-term results are similar whether this is performed or not.
- Prolonged rest (greater than 12 months) may improve the prognosis.

Trauma to the Medial Tuberosity of the Distal Radius

This is a condition most commonly found in Standardbred pacers in which one forefoot traumatizes the skin and soft tissue over the medial tuberosity of the distal radius. Most trainers describe this as the horse "getting on the knee." Horses with this problem will usually have a "toe out" or "penguin-toed" conformation. For this reason, many pacers will wear "knee boots."

HISTORY AND PRESENTING SIGNS

- Swelling noted by trainer or owner over the medial aspect of the distal radius.
- Lameness is unusual in this disorder.
- Horses are occasionally presented with a "big knee."

CLINICAL FINDINGS AND DIAGNOSIS

- Swelling over the medial aspect of the distal radius is quite apparent, and there will be pain on palpation.

KEY POINT

In most cases the horse is not lame. If the condition is chronic, there may be extensive soft-tissue swelling over the medial tuberosity of the distal radius.

- Radiographs are not usually necessary and reveal only soft-tissue swelling. However, if there is any lameness or significant pain on palpation or flexion of the carpus, a full series of radiographs should be taken.

DIFFERENTIAL DIAGNOSIS

- Distal radial fractures
- Hygroma of the carpus
- Chip fractures of the proximal carpal joint

TREATMENT

- Local anti-inflammatory measures are usually all that is required to resolve some of the swelling. These measures include cold hosing, cold packs, and locally applied DMSO (Treatment No. 34).
- If extensive swelling is present, a 7-day course of phenylbutazone (Treatment No. 88) may be necessary.
- To prevent further trauma, the use of a "knee boot" during training should be advised.

KEY POINT

Corrective shoeing, with the application of square-toe shoes on the front feet, will help by enforcing breakover at the toe. Pacers and trotters should be exercised wearing "knee boots."

VALGUS OR VARUS DEFORMITY OF THE CARPUS

KEY POINT

Valgus deviation of the carpus is the most common limb deformity in growing foals and results in deviation of the third metacarpal bone laterally, away from an imaginary line drawn through the midline of the limb viewed from in front of the foal.

The opposite of this (carpus varus) is less common, although it is the most frequent angular limb deformity of the fetlock. The problem can be congenital or acquired. One of the main etiologic factors is uneven forces on the growth plate of the distal radius. Congenital carpal valgus may be due to abnormal limb position in uteri, whereas acquired angular limb deformities are caused by excessive weight bearing.

HISTORY AND PRESENTING SIGNS

- Owner or stud-farm manager notices that the foal's forelimb(s) show progressive deviation with the foal appearing more "knock kneed."
- Usually first noticed when the foal is around 1 month of age, but in some cases (particularly dysmature foals) it may be apparent shortly after birth.
- In cases that are obvious soon after the foal stands, the appearance of the limbs will often improve considerably over the first week of life, as the foal develops better muscle tone.

CLINICAL FINDINGS AND DIAGNOSIS

- The deviation of the limb is apparent, although in some cases it initially may be quite mild. In very young foals, the most important differential diagnoses are carpal bone collapse due to delayed ossification and ligament laxity. Deviations due to abnormal growth will have minimal swelling of the carpus, and it will not be possible to straighten the limb manually, as is often the case with carpal bone collapse and ligament laxity.
- Dorsopalmar radiographs are useful to determine the source of the deviation and to rule out carpal bone collapse. The source of the deviation can be determined by drawing lines down the long axes of the radius and third metacarpal bones. The lines intersect at the major source of the limb deviation.

DIFFERENTIAL DIAGNOSIS

- Carpal bone hypoplasia
- Mild conformation faults

TREATMENT

- Congenital carpal valgus arising from the distal radial physis generally will correct over time, provided the mare and foal have restricted exercise. Mild deformity in foals less than 3 months of age also can be treated conservatively, with reduced exercise and glue-on shoes with lateral extensions.
- In more severe cases or cases with deformity still present after 3 months of age despite conservative therapy, periosteal stripping is used to accelerate growth on the lateral aspect of the distal radius. This technique involves transection of the periosteum just proximal to the distal radial physis and elevation of the resulting periosteal flaps. The remnant of the ulna is also transected to maximize the response.
- In horses over 3 months of age with severe deformities or milder deformities in horses approaching 6 months of age, a transphyseal bridging technique is required on the medial aspect of the distal radius. This can take the form of staples, screws and wires, or a small bone plate. The implants must be removed when the limb is within 5 degrees of being straight to prevent overcorrection.

Carpal Collapse or Carpal Bone Hypoplasia

Carpal collapse is a problem noted most frequently in dysmature foals and results from the foals placing abnormal stresses on the carpal bones before the bones have fully ossified. Similar problems can be encountered in the tarsus. Foals usually present less than 1 week old with a carpus valgus or carpus varus deformity.

HISTORY AND PRESENTING SIGNS

- Foal born prematurely or poorly developed
- Limb deviation apparent shortly after birth
- Other musculoskeletal consequences of dysmaturity such as laxity of the flexor tendons

CLINICAL FINDINGS AND DIAGNOSIS

- Deviation of the forelimbs is common, although in young foals the limb can be straightened with manipulation.
- The most common deviation in foals with carpal bone hypoplasia is carpus valgus.

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- Dorsopalmar radiographs should be taken of both carpi to evaluate the shape of the carpal bones.
- Longitudinal lines drawn through the long axes of the third metacarpal bone and the radius will indicate that the site of the deviation is the carpus.

DIFFERENTIAL DIAGNOSIS

- Angular limb deformities arising primarily from the distal radial growth plate
- Ligament laxity
- Malformation of the second or fourth metacarpal bones

TREATMENT

- If identified early, lateral to medial stability can be provided by a tube cast extending from immediately proximal to the fetlock to the proximal radius. This will allow ossification of the carpal bones to progress. The cast should be changed every 2 weeks.

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FOREARM AND ELBOW PROBLEMS

Fractures of the Radius

Radial fractures are most commonly found in foals and yearlings. Such fractures are due to direct trauma such as kicks and usually occur when horses are run in herds. The long-term prognosis for athletic function in horses with radial fractures is very poor, and owners should be advised of this before treatment is commenced. Most fractures are midshaft and require referral to a specialist center for application of one or two bone plates. There is a prolonged hospitalization time, up to several months, and the attempted internal fixation of these fractures should only be undertaken in valuable animals that have some breeding future.

HISTORY AND PRESENTING SIGNS

- Most horses are unable to bear weight on the affected leg.
- It is uncommon for the traumatic event to be seen by anyone on the stud farm, and there may be no external signs of trauma.

DIFFERENTIAL DIAGNOSIS

- Fracture of the humerus or ulna
- Fracture of carpal bones

DIAGNOSIS AND TREATMENT

- Non-weightbearing lameness indicates the likelihood of a long bone fracture.
- There may be some degree of swelling over the midradius, but this is not always the case.
- Close examination of the forearm reveals crepitus to be present on palpation and manipulation of the limb.
- Because penetration of part of the fractured bone through the skin will result in a much worse prognosis, it is important to establish early if the fracture is compound.

KEY POINT

If the horse requires transport to a facility to take radiographs for further evaluation, the limb should be immobilized as effectively as possible (see Chapter 19).

- Craniocaudal and lateral radiographs are essential to determine the type of fracture present.
- In horses of low economic value, more distal fractures of the radius may be successfully treated by external immobilization alone.

KEY POINT

The treatment of choice for radial fractures is internal fixation using two bone plates placed on the cranial and lateral aspects of the radius.

- Casts are contraindicated except for distal radius fractures as they end too close to the fracture

line and in most cases do not provide adequate stability.

Fractures of the Ulna

Fractures of the ulna are usually the result of kicks or sudden falls. Although the fractures cause dramatic clinical signs, the prognosis for full athletic recovery is excellent, particularly if the fracture does not involve the elbow joint. In foals, physeal fractures of the olecranon tuberosity are quite common.

HISTORY AND PRESENTING SIGNS

- Acute history of non-weightbearing lameness
- Presented with a "dropped elbow" appearance

CLINICAL FINDINGS AND DIAGNOSIS

- Limited or no weight bearing on the affected leg is a typical sign.
- There is a "dropped elbow" appearance to the leg, with the horse unable to extend the elbow.
- Fractures involving the elbow joint usually result in signs of more pain and reluctance to bear weight compared with fractures not involving the joint.
- Examination of the elbow region usually will show limited swelling and pain on palpation. In some cases, crepitus may be detected.
- A lateral radiographic view is most important for diagnosis. Because there may be considerable distraction of the proximal fragment, it is important to ensure that the radiographic field includes the area up to 15 cm (6 inches) proximal to the elbow joint. Fractures of the proximal radial physis may occur in combination with an ulnar fracture.

DIFFERENTIAL DIAGNOSIS

- Fracture of the proximal radius
- Fracture of the distal humerus
- Radial nerve paralysis

TREATMENT

- Conservative therapy may be tried in horses with nondisplaced fractures distal to the radiohumeral joint. The horse should be confined to a stall for 2 to 3 months. A splint to prevent carpal flexion may be helpful.
- More proximal fractures tend to distract due to the pull of the triceps muscle, and internal fixation provides the best results. Using the tension-band principle, a narrow dynamic compression plate is placed along the caudal border of the olecranon and ulna. If the proximal fragment is displaced some distance proximally, reduction of the fracture can be difficult. In young foals with physeal fractures, the proximal fragment is small and implants can weaken it dramatically. Heavily contoured plates are used to hold it in place.
- The prognosis for ulna fractures is quite good, even in cases where there is severe distraction of the proximal fragment. However, where there has been involvement of the elbow joint in the fracture, there may be secondary degenerative joint disease that may result in a chronic low-grade lameness.
- Physeal fractures in foals have a good prognosis, unless there is a Salter II type fracture with involvement of the elbow joint.

Hygroma of the Elbow ("Shoe Boil")

Hygroma of the elbow is a fluid-filled subcutaneous swelling at the point of the elbow. It results from trauma and may be due to the shoe causing pressure and rubbing on the elbow when the horse is lying down. Hence, the lay term for the condition is "shoe boil." It is also referred to as "capped elbow." If the condition is chronic, there may be extensive fibrous tissue formation rather than simple fluid accumulation.

HISTORY AND PRESENTING SIGNS

- Usually found in horses kept in stables
- Obvious swelling over the point of the elbow noted by owner or trainer

CLINICAL FINDINGS AND DIAGNOSIS

- A fluid-filled, nonpainful swelling on the point of the elbow is pathognomonic.
- This condition does not cause the horse any problems and is merely a cosmetic blemish.

KEY POINT

Lameness is not a feature of this condition, and if lameness is found, it is likely to be due to problems at an additional site.

- Radiographs are not necessary unless there are signs of pain on palpation of the area.

DIFFERENTIAL DIAGNOSIS

- Fractures of the ulna
- Soft-tissue trauma/foreign-body penetration
- Damage to tendon of triceps brachii muscle

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TREATMENT

- Treatment options include drainage and injection of long-acting corticosteroids, surgical drainage and insertion of a Penrose drain, and complete resection of the mass. Surgery is generally reserved for cases that fail to respond to medical therapy.
- Surgical drainage and the insertion of drains does not always result in resolution and loose skin over the olecranon often remains. Resection of the mass allows redundant skin to be resected and eliminates the chance of recurrence, provided that the inciting cause is removed. The technique can be performed standing to remove the risk of dehiscence during anesthetic recovery.
- Whatever treatment is used, further trauma to the olecranon must be prevented. This can be achieved by increasing the thickness of the bedding and the application of a "doughnut pad" to the pastern.

Intra-articular Elbow Lameness

Lameness arising from the elbow joint is very uncommon. However, in lamenesses that do not block out in the distal limb, the possibility of an elbow joint problem should not be discounted. Fortunately, intra-articular anesthesia of the elbow joint is very simple, allowing a definitive diagnosis to be made.

HISTORY AND PRESENTING SIGNS

- Usually a chronic forelimb lameness
- Lameness thought to be in the shoulder or foot because no swelling is present anywhere in the affected limb

CLINICAL FINDINGS AND DIAGNOSIS

- Chronic low-grade unilateral forelimb lameness typically is found.
- A careful clinical examination of the affected leg may not reveal any swelling, heat, or pain.
- Regional anesthesia commencing at the foot and ascending proximally to the carpus does not result in improvement in gait.
- Lameness is abolished after desensitizing the elbow joint (see Fig. 4-40).
- Radiographs may not reveal any abnormalities in the joint. In some cases, there are changes involving the anconeal process of the ulna, with joint irregularity and spur formation. These changes may only be found on a flexed lateral view of the elbow joint.

DIFFERENTIAL DIAGNOSIS

- Problems involving the shoulder joint
- Rupture of the medial collateral ligament of the elbow joint

TREATMENT

- Intra-articular medications (see Osteoarthritis) may result in temporary alleviation of the clinical signs. However, the prognosis for athletic soundness in horses with intra-articular elbow lameness is very poor.
- Arthroscopy of the elbow joint is possible and may be helpful in establishing a diagnosis if no radiographic changes are evident.

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SHOULDER PROBLEMS

Shoulder lameness is quite uncommon in horses. However, it is a favorite diagnosis of horse trainers and owners and those involved in chiropractic manipulation of horses. The difficulty often lies in convincing the client that the problem does not originate in the shoulder but is elsewhere in the leg.

> KEY POINT
>
> *In many shoulder problems, there are no localizing signs on clinical examination, and unless the problem is intra-articular, it may be difficult to establish a definitive diagnosis.*

Intra-articular problems are easily diagnosed by depositing local anesthetic into the shoulder joint. The technique for intra-articular anesthesia of the shoulder is not difficult and in most horses is well tolerated.

In cases where there is a suspicion of a shoulder problem, a careful examination of the shoulder should be carried out. Flexion, extension, and abduction of the shoulder should be performed to

determine whether there is any restriction of movement or pain. Particular note should be paid to the muscles around the shoulder for signs of atrophy and pain on palpation. In some conditions, applying flexion to the shoulder joint for 1 minute and trotting the horse off may accentuate the lameness.

Bicipital Bursitis

The bicipital bursa is interposed between the tendon of the biceps brachii and the intertubercular groove of the head of the humerus. Because the bursa is situated over the point of the shoulder, it is subjected to trauma, resulting in a bursitis. With open wounds the bursitis is often septic. Occasionally, the same injury may result in damage to the suprascapular nerve. On rare occasions, the bicipital bursa may become infected with *Brucella abortus* via a similar mechanism to that for fistulous withers.

HISTORY AND PRESENTING SIGNS

- Moderate to severe lameness found after a history of trauma to the shoulder
- Swelling and heat over the point of the shoulder

CLINICAL FINDINGS AND DIAGNOSIS

- Forelimb lameness with evidence of marked lifting of the head is a good indicator of shoulder lameness.
- Circumduction of the leg also may be seen as the limb is advanced. In most cases, pain can be elicited by palpation over the bicipital bursa.
- Pain may be elicited with flexion and extension of the shoulder joint.
- In chronic cases, atrophy of the shoulder muscles will occur.
- Ultrasound examination will demonstrate increased fluid in the bursa.
- Changes in contour of the humerus may be detected where there is bony involvement.
- If infection is suspected, a synovial fluid sample should be collected for total white cell count, total protein, and culture and sensitivity.

DIFFERENTIAL DIAGNOSIS

- Sweeney
- Fractures of the scapula
- Fractures of the humerus
- OCD of the shoulder

TREATMENT

- In nonseptic cases, rest and anti-inflammatory therapy is indicated.
- If sepsis is present, this should be treated with appropriate antibiotics.
- In chronic cases, surgical drainage and synovectomy have been used successfully.

Fractures of the Humerus

Fractures of the humerus are not as common as other long bone fractures and have a slightly better prognosis because of the success of conservative treatment. Humeral fractures are most commonly associated with falling but may be due to direct trauma such as a kick from another horse. Humeral stress fractures have also been reported in racehorses. Because the course of the radial nerve is intimately associated with the humerus, radial nerve paralysis is a potential complication. However, this is very difficult to assess preoperatively.

HISTORY AND PRESENTING SIGNS

- Non-weightbearing lameness of the affected leg
- "Dropped elbow" appearance of the leg

CLINICAL FINDINGS AND DIAGNOSIS

- In many cases there is swelling over the fracture site and abrasion of the skin.
- The degree of "dropped elbow" appearance will depend on the extent of the fracture displacement and overriding of the major fracture fragments.
- Crepitus may be difficult to detect in many humeral fractures.
- If a fracture is suspected, excessive manipulation of the leg should be avoided because trauma to the radial nerve is a possibility.
- Radiographs should be taken, and these may require general anesthesia. This could be difficult because the fracture may be worsened during recovery. Therefore, if adequate radiographs cannot be taken with the horse conscious, it may be better to postpone radiographs until the time of surgery.

DIFFERENTIAL DIAGNOSIS

- Fracture of the olecranon
- Fracture of the scapula
- Radial nerve paralysis

TREATMENT

- Oblique humeral fractures may heal with box stall confinement. This is because of the support of the large muscle mass when the horse does not bear weight on the leg. The healing that

results may not produce a horse that is athletically sound, but it may be sound for breeding purposes.
- The main complications in foals are the development of angular limb deformities in the opposite limb or flexural deformity in the affected limb.
- Open reduction and plate fixation via a cranial approach is indicated for transverse or comminuted fractures. The prognosis for adults is poor, whereas foals have a better prognosis. Two plates are required for most cases, although a single cranial plate may be used in foals less than 150 kg.

Fractures of the Scapula

Fractures of the scapula are relatively rare and most fractures of the scapula are due to falls. However, direct blows to the shoulder also may produce scapular fractures. Fractures are mostly simple and usually involve the supraglenoid tubercle and the glenoid cavity. Occasionally, the spine of the scapula will fracture, and in rare cases, there will be comminution, usually involving the neck of the scapula.

HISTORY AND PRESENTING SIGNS

- History of direct trauma is usual.
- Horses may present with mild lameness or, in severe comminuted fractures, may present unable to bear weight on the affected leg.

CLINICAL FINDINGS AND DIAGNOSIS

- Fractures of the scapula usually produce a severe lameness of acute onset, although fractures of the scapular spine may result in a more mild lameness.
- Palpation around the shoulder joint usually reveals pain, and there may be pain on flexion of the shoulder.
- Horses with longer standing fractures may present with muscle atrophy.
- Fracture of the scapula is confirmed by radiography. Supraglenoid tubercle fractures can be observed with standing radiographs, but general anesthesia is required for fractures of the neck and body of the scapula.

DIFFERENTIAL DIAGNOSIS

- Suprascapular nerve paralysis
- Fractures of the proximal humerus

TREATMENT

KEY POINT

Many fractures of the scapula will heal sufficiently well with box stall rest to permit pasture soundness.

- Fractures of the spine of the scapula can be treated conservatively and have a good prognosis.
- Fractures involving the neck of the scapula can be repaired by internal fixation using bone plates. Comminuted fractures with a lot of displacement have a poor prognosis. Fractures of the supraglenoid tubercle are difficult to repair successfully. Conservative treatment will generally result in pasture soundness and occasionally athletic soundness. Resection of the fractured fragment has also resulted in soundness in some horses. Successful internal fixation has been reported in horses less than 400 kg using 5.5-mm cortical screws and cerclage wire combined with partial tenectomy of the biceps tendon.

Osteochondrosis Dissecans of the Shoulder Joint

OCD of the shoulder is a condition that causes a severe lameness in yearling or 2-year-old horses. It generally involves the head of the humerus toward the caudal limit of the scapulohumeral joint. More details on OCD are given in the section on general musculoskeletal problems. Shoulder joint OCD is the most common form of shoulder lameness in young Thoroughbred horses.

HISTORY AND PRESENTING SIGNS

- Intermittent forelimb lameness in a yearling
- Lameness usually noted at the walk and at the trot

CLINICAL FINDINGS AND DIAGNOSIS

- Clinical signs are very similar to those for bicipital bursitis except that in most cases there is no pain on palpation of the shoulder. Muscle atrophy is common.
- Because of reduced weight bearing, affected horses often have an upright or club foot.
- Diagnosis is aided by intra-articular local anesthesia because most horses with OCD of the shoulder will show improvement of lameness afterward.
- Radiographs are necessary to determine the extent of the lesion(s) and the prognosis. These should be performed under general anesthesia.

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- Because the opposite shoulder joint also may be affected, radiographic examination of the unaffected shoulder also should be performed.

DIFFERENTIAL DIAGNOSIS

- Bicipital bursitis
- Shoulder muscle injuries
- Club foot

TREATMENT

- If untreated, OCD of the shoulder results in secondary osteoarthritis.
- Arthroscopic surgery of the shoulder joint is possible in young horses and enables visualization of the joint surface, removal of loose fragments, and curettage of the damaged subchondral bone. The prognosis is guarded, with less than 50% of horses being able to perform athletically.

Sweeney (Suprascapular Nerve Damage)

Sweeney is the term used to describe atrophy of the supraspinatus and infraspinatus muscles due to paralysis of the suprascapular nerve. Most cases result from trauma to the point of the shoulder, although it is possible that damage to the nerve can arise because of the nerve being stretched. This could occur if the shoulder joint were suddenly thrust caudally.

HISTORY AND PRESENTING SIGNS

- In chronic cases, atrophy is clearly visible.
- The shoulder can "pop" outward when the horse places weight on the affected leg.

CLINICAL FINDINGS AND DIAGNOSIS

- In early cases, before visible atrophy of the affected muscles, outward movement of the shoulder joint can be seen when the horse takes weight on the leg.

KEY POINT

Atrophy of the supraspinatus and infraspinatus muscles is readily apparent after several weeks, and the spine of the scapula becomes more prominent. This is the classical clinical sign of suprascapular nerve damage.

- With sweeney, there may be acute lameness immediately after the injury that improves after 7 to 10 days.
- Radiographs may be taken to ensure no bony lesions.

DIFFERENTIAL DIAGNOSIS

- Myopathies
- Disuse atrophy

TREATMENT

- In acute cases, stall rest and systemic anti-inflammatory therapy may result in resolution of signs. Nerve dysfunction will gradually improve over 6 to 10 weeks.
- If clinical signs persist for more than 12 weeks, surgery to free the suprascapular nerve is the treatment of choice.
- After the nerve has been dissected free of any scar tissue, a wedge of bone is removed from underneath the nerve. The bone removed should be no more than 1.5 cm^2 (0.5 $inch^2$), because fractures of the scapula have occurred during recovery when larger sections of bone were removed.
- Immediate improvement and return of function to the suprascapular nerve do not take place. However, improvement usually occurs over a period of 4 to 6 months. Approximately 80% of cases improve after surgery, although some muscle atrophy may remain.

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Hindlimb Abnormalities

Injuries to the foot, pastern, fetlock, and metatarsus are similar to those found at equivalent sites in the forelimb. Acute hindleg problems are generally due to problems in the distal limb, such as subsolar (hoof) abscesses, phalangeal fractures, and fractures of the sesamoid bones. Unlike foreleg lameness, which many owners and trainers are

aware of, hindleg lameness may remain undetected. Horses with chronic low-grade hindleg lameness may be presented for performance problems rather than specific lameness.

Details of the examination procedure are outlined in the Introduction. However, there are a few additional points about examining horses for hindleg lameness.

1. In chronic lameness, gluteal muscle atrophy is common and can be viewed standing directly behind the horse. This is an important sign of chronic upper (hock or above) hindleg problems.
2. The nerve distribution in the distal hindleg appears to be more variable than in the foreleg. This results in some nerve blocks (particularly the plantar digital) being less reliable and more difficult than in the foreleg.
3. Unless there is a distal limb problem, there may be few localizing signs (e.g., heat, swelling, pain) to indicate the site of the problem. For this reason, intra-articular local anesthesia of the hock and stifle is very important to enable a diagnosis to be established.

HOCK AND TIBIA PROBLEMS

Bog Spavin (Tarsal Hydrarthrosis)

Bog spavin is the common name given to distention of the tarsocrural (tibiotarsal) joint with synovial fluid. In cases of distention, the joint can contain more than 100 mL of synovial fluid. Bog spavin is chiefly a cosmetic problem, but because OCD also can present with similar features, radiographs of the hock should always be taken. The cause of the condition is unknown.

HISTORY AND PRESENTING SIGNS

- Usually found in young horses.
- Horses are usually presented because of gross swelling over the dorsomedial and dorsolateral aspects of the hock.

CLINICAL FINDINGS AND DIAGNOSIS

- Despite the degree of distention of the joint capsule, horses with bog spavin do not show signs of lameness.
- The fluid can be demonstrated to move from the medial to lateral aspects of the joint by palpation over the distended dorsomedial joint capsule.
- There is no heat or pain on palpation.
- Radiographs (lateral and oblique views) should be taken to ensure no chip fractures from the talus or OCD lesions. It is particularly important to examine the area of the intermediate ridge on the radiographs because small OCD lesions in this location can easily be missed.
- Synovial fluid examination is not necessary, but if a sample is taken, it will reveal low protein content (<20 g/L or 2.0 g/dL) and low cell count (<400 X 10^6/L).

DIFFERENTIAL DIAGNOSIS

- Tarsal chip fractures
- Thoroughpin
- OCD

TREATMENT

- Bog spavin requires no treatment, because the condition does not interfere with function.
- Because it is cosmetically undesirable, many clients request treatment.

KEY POINT

The most useful treatment consists of draining the excess synovial fluid from the tarsocrural joint (see Fig. 4-44) and injecting 200 to 250 mg of medroxyprogesterone acetate (Treatment No. 70) intra-articularly.

- The treatment has an antisecretory effect on synovia and will be successful in some but by no means all cases.

Bone Spavin

Bone spavin is an osteoarthritis usually involving the tarsometatarsal and centrodistal joints. In some cases, the proximal intertarsal joint may be involved.

KEY POINT

Bone spavin is the most common cause of chronic hindlimb lameness in horses.

All breeds of horses may be affected. The condition is progressive and results in a lameness that gradually worsens with time.

HISTORY AND PRESENTING SIGNS

- Poor racing performance
- Chronic hindlimb lameness
- Standardbreds also may have a history of not running straight in the cart, with the hindquarters moving toward the shaft of the sulky on the affected side.

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CLINICAL FINDINGS AND DIAGNOSIS

- Examination of the hindleg will usually reveal nothing significant, and there is no pain on flexion or palpation of the hock.
- Careful examination of the gait at the trot may show a lower arc of flight of the foot, in an attempt to limit the degree of hock flexion, when the horse trots.
- Application of the spavin test will usually accentuate the lameness when the horse is trotted off after 2 minutes of hindleg flexion.
- A positive diagnosis can be made by intra-articular local anesthesia of the tarsometatarsal and centrodistal joints. In most cases, the lameness will not be abolished, but there will be a definite improvement in the gait. In advanced cases it may be difficult to inject the centrodistal joint, and a tibial and peroneal nerve block may be required.
- Radiographs should be taken with the x-ray beam centered about 10 cm (4 inches) distal to the point of the hock. Changes are found most commonly on the lateral and plantarodorsal oblique views.
- Radiographic changes are found most commonly in and around the tarsometatarsal joint. Periarticular new bone growth is often found around the dorsal margins of the proximal aspect of the third metatarsal bone. There also may be narrowing of the joint space and bone sclerosis and lysis, most easily visible on the dorsomedial aspect of the joint. If there are radiographic changes involving the talocalcanean centroquartal joint (proximal intertarsal), the prognosis is poor for future athletic function. In milder cases of bone spavin, changes may be more common on the dorsolateral aspect of the hock, involving the tarsometatarsal and distal intertarsal joints. These changes may not be apparent on the plantarodorsal or lateral views.

KEY POINT

Because of the variable location of radiographic changes, it is important to take oblique radiographic views when examining the hock.

DIFFERENTIAL DIAGNOSIS

- Cunean tendon bursitis
- Stifle joint problems

TREATMENT

- Bone spavin is a progressive disease that may eventually result in ankylosis of the affected joints. Once ankylosis has occurred, the signs of lameness will abate.

KEY POINT

In the early stages of joint disease, intra-articular long-acting corticosteroids (Treatment Nos. 12 and 74) can be injected into the affected joints.

- Relief of the clinical signs varies from 2 weeks to 6 months after a single treatment. If a prolonged response is achieved, this form of treatment is preferred to arthrodesis because there are fewer potential complications.
- If the response to corticosteroids is inadequate or of short duration, arthrodesis should be considered. Two methods have been described: surgical and chemical. Intra-articular injection of sodium monoiodoacetate (100 mg per joint) is a low-cost alternative to surgery and results in reliable fusion. It is recommended that contrast material is injected and a radiograph taken to determine which joints will be affected, because occasional communication between the tarsometatarsal, centrodistal, and tarsocrural joints has been reported. Alternatively, surgical arthrodesis of the affected joint(s) can be performed using three 3.2-mm drill holes to remove subchondral bone and articular cartilage.
- Whichever technique is used, a postoperative exercise program is undertaken to hasten arthrodesis. After drilling of the joints, arthrodesis generally takes 6 to 12 months.

Capped Hock

Capped hock is a subcutaneous swelling that develops at the point of the hock as a result of trauma. It is a hygroma or traumatic bursitis and in some acute cases may be filled with a large amount of fluid.

HISTORY AND PRESENTING SIGNS

- Swelling over the point of the hock
- No lameness

CLINICAL FINDINGS AND DIAGNOSIS

- Swelling over the point of the hock without any lameness is a consistent finding.
- If the injury is acute, there will be pain on palpation over the swollen area, but in long-standing cases there will be no pain and only fibrous enlargement.

- Radiography is not necessary, unless there are signs of lameness.

DIFFERENTIAL DIAGNOSIS

- Thoroughpin
- Curb (plantar ligament sprain)
- Luxation of the superficial digital flexor tendon

TREATMENT

- If the swelling is long-standing, with only fibrous tissue present, no treatment will be successful. Surgery to improve the cosmetic appearance of the leg only should be attempted by the brave surgeon who has access to a good attorney.
- In acute cases, cold hosing and a topical anti-inflammatory preparation such as DMSO (Treatment No. 34) can be useful, followed by a Robert-Jones type of dressing (see Chapter 19).
- If a large amount of fluid is present, drainage of the fluid and a pressure bandage may be successful.

Curb (Plantar Ligament Desmitis)

Curb is the common name given to desmitis of the plantar ligament. The plantar ligament runs from the plantar aspect of the calcaneus to the fourth metatarsal bone. Curb is a condition most commonly found in Standardbred trotters or pacers. Although excessive hock angulation (so-called sickle hock) may contribute to the condition, it is not clear what produces the condition. Most cases of curb cause only mild lameness and resolve quickly with rest.

CLINICAL FINDINGS AND DIAGNOSIS

- The first sign of curb is swelling at the plantar aspect of the hock and about 8 to 10 cm (3.5-4 inches) distal to the point of the hock.
- In the acute stage there will be heat, swelling, and pain, and there may be a mild hindleg lameness.
- In chronic cases, swelling will be the only residual sign.
- Ultrasonography generally reveals a mottled hypoechogenicity and enlargement and will differentiate plantar ligament desmitis from tendonitis of the adjacent flexor tendons.
- Radiographs reveal only soft-tissue swelling on the plantar aspect of the calcaneus. However, in severe cases of trauma to the plantar ligament, there may be subsequent new periosteal bone growth along the plantar border of the calcaneus.
- If an apparent curb is seen in a foal or yearling, radiographs should be taken because tarsal collapse due to incomplete ossification of the third and central tarsal bones may have a similar appearance.

DIFFERENTIAL DIAGNOSIS

- Periligamentous or peritendinous effusion
- Tendinitis of the proximal flexor tendons
- Thoroughpin
- Tarsal collapse

TREATMENT

- In the acute stage, local anti-inflammatory measures such as cold water hosing, application of cold packs, and bandaging will help reduce the swelling.
- Anti-inflammatory drugs such as phenylbutazone (Treatment No. 88), given for 7 days, also can help to reduce the swelling.
- Topical preparations such as DMSO (Treatment No. 34) are useful in reducing the swelling and inflammation.
- Some practitioners advocate injection of corticosteroids locally into the area of the swelling. This results in immediate reduction in swelling but may encourage the trainer to put the horse back into full training before the damage has healed.
- Most cases of curb will resolve with 4 to 6 weeks of rest or light exercise. Horses in which there is structural damage to the superficial flexor tendon require a long period of rest and carry a poorer prognosis.

Fracture of the Talus (Tibial Tarsal Bone)

Fractures of the talus occur in a variety of configurations from small fragments of the distal lateral trochlear ridge or proximal medial trochlear ridge to complete sagittal fractures. Most are due to direct trauma. A normal vascular channel is often observed at the distal end of the medial trochlear ridge and should not be mistaken for a fracture. Small osseous densities can also be present at this site as a normal finding.

HISTORY AND CLINICAL SIGNS

- Variable degrees of lameness
- Distention of the tarsocrural joint

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CLINICAL FINDINGS AND DIAGNOSIS

- Tarsocrural joint effusion
- The degree of lameness depends on the severity of the fracture.
- Intra-articular anesthesia may be helpful where there is doubt about the significance of a radiographic finding.

DIFFERENTIAL DIAGNOSIS

- Fractures of other tarsal bones
- OCD

TREATMENT

- Most fracture fragments can be removed arthroscopically either via a dorsal or a plantar approach. Larger fragments may require arthrotomy for removal, but if acute, internal fixation is preferred.

Osteochondrosis Dissecans of the Tarsocrural Joint

The tarsocrural joint is the most common joint affected by osteochondrosis. The usual site is the distal intermediate ridge of the tibia, but the lateral trochlear ridge, medial trochlear ridge, and medial malleolus also may be affected.

HISTORY AND PRESENTING SIGNS

- Distention of the tarsocrural (tibiotarsal) joint
- Little or no lameness

CLINICAL FINDINGS AND DIAGNOSIS

- There is distention of the tarsocrural joint capsule with fluid and varying degrees of lameness depending on the extent of the lesion.
- The opposite hock also may be affected and therefore should be examined carefully.
- Radiographs will indicate the exact site and extent of the lesion(s). Important views are the lateromedial and two oblique views. Distal intermediate ridge lesions are best seen on the dorsomedial plantarolateral view.

DIFFERENTIAL DIAGNOSIS

- Fracture of the talus

TREATMENT

- If a small fragment is present, no treatment may be required apart from rest, although intra-articular medications such as sodium hyaluronate (Treatment No. 60) can be beneficial.

KEY POINT

The treatment of choice is arthroscopic surgery to remove the osteochondral fragments, curette the damaged articular surface, and lavage the joint. Resolution of effusion depends on the length of time it has been present. A period of 3 to 6 months of rest is usually advocated, depending on the extent of the lesion. Approximately 75% of horses will perform successfully after surgery.

Rupture of the Peroneus Tertius

The peroneus tertius is part of the stay apparatus of the hindlimb. Its origin is on the dorsolateral aspect of the distal part of the femur, and it inserts on the dorsal aspect of the proximal third metatarsal bone. Rupture of the peroneus tertius is an uncommon injury, but it produces typical clinical signs and therefore is easily diagnosed.

HISTORY AND PRESENTING SIGNS

- Abnormal hindleg carriage
- Extension of the hock with stifle flexion

CLINICAL FINDINGS AND DIAGNOSIS

- The horse usually bears weight without trouble but shows reduced flexion of the hock as the limb is advanced.
- The typical sign of rupture is that it is possible to flex the stifle while extending the hock. This is most easily seen by moving the hindleg backward.
- The limb also may appear to tremble as it is advanced and has a loose or slack appearance.

DIFFERENTIAL DIAGNOSIS

- The ability to extend the hock while the stifle is flexed is pathognomonic.

TREATMENT

- Rest is the only treatment that is feasible, and most cases will resolve with time. In most cases, 3 to 4 months of rest will give resolution of the problem. The early part of the rest period should take place in a box stall so that movement is restricted.
- Because cases are uncommon, there have been

no reports of the follow-up of a large series of cases of rupture of the peroneus tertius.

Stringhalt

Stringhalt is a condition that results in involuntary flexion of one or (usually) both hindlimbs. Where the condition is bilateral, it is found in horses that depend on pasture for nutrition (so-called Australian stringhalt). Therefore, it has been suggested that a pasture-derived neurotoxin may be important in the pathogenesis. In the United States, lathyrism (sweetpea toxicity) has been reported to result in a stringhalt-like syndrome, and in Australia, flatweed-dominated unimproved pastures have been incriminated in some cases. A common history in outbreaks of the disease in Australia is that horses are affected after a period of drought followed by substantial rain with fresh pasture growth. Most commonly this occurs in spring, and substantial numbers of horses have been affected within the same geographic area.

Unilateral stringhalt can also occur secondary to trauma to the extensor tendons on the dorsal aspect of the tarsus.

HISTORY AND PRESENTING SIGNS

- Involuntary flexion involving one or both hindlegs

CLINICAL FINDINGS AND DIAGNOSIS

- A typical goose-stepping gait with hyperflexion of the hindlegs is characteristic.
- This gait is more evident when the horse is first walked off and improves as the horse "warms up."
- The severity of the gait abnormality varies and may be accompanied by atrophy of the hindlimb muscles, and left laryngeal hemiplegia also may be found. If the condition has been present for weeks to months, atrophy of the muscles on the lateral aspect of the tibia is a consistent finding. Occasionally, a flexural deformity of the fetlock joints develops in severe cases.
- Because the condition appears to be a long nerve problem, the left recurrent laryngeal nerve may be affected; therefore, endoscopic examination of the larynx should be considered.

DIFFERENTIAL DIAGNOSIS

- Upward fixation of the patella

TREATMENT

- Although many cases will resolve spontaneously, this may take months to years. If the condition is thought to arise from plant toxins, removal of the horse to another paddock may give resolution. However, this may take several months.
- In cases that do not resolve or cases associated with trauma, surgical resection of the lateral extensor tendons may be tried, and success rates of approximately 80% have been reported. It is interesting, however, that where horses do improve with surgery, the improvement is rarely immediate and can take up to 7 to 10 days.

KEY POINT

Studies of nerve pathology have demonstrated widespread axonopathy and neurogenic muscle atrophy in bilateral cases of stringhalt. The validity of surgical treatment in these cases, therefore, may be open to question.

- The use of phenytoin (Treatment No. 89) has been reported to result in improvement in clinical signs while horses were on treatment. The dose rate recommended is 15 mg/kg given in capsule or powder form twice daily.
- There has been some success with the use of a GABA inhibitor, baclofen, given orally at a dose rate of 1 mg/kg three times daily (Malik and Kannegieter, personal communication, 1993). Some cases have shown a dramatic response within days of treatment, whereas others have continued to improve after ceasing therapy, despite the presence of clinical signs for more than 12 months. In other cases, there has been no significant improvement. Because baclofen is a centrally acting drug, it may be that there is a central and a peripheral component to stringhalt.

Thoroughpin

Thoroughpin is the term used to describe a tenosynovitis of the tarsal sheath. It often is associated with trauma, although in some cases there is no apparent cause. Where it is associated with trauma to the medial aspect of the talus, there also may be bony lesions of the sustentaculum tali.

KEY POINT

Thoroughpin should be differentiated from hematomas that can occur in the region of the tarsal sheath (false thoroughpin).

HISTORY AND PRESENTING SIGNS

- Trauma to the tarsus
- Fluid-filled swelling on the plantar aspect of the hock

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CLINICAL FINDINGS AND DIAGNOSIS

- A nonpainful, fluid-filled swelling just proximal to the point of the hock, on both medial and lateral sides, is typical. Variable degrees of lameness may be present.
- Radiography is recommended to rule out the possibility of bony lesions. Lesions of the sustentaculum tali are best observed on a dorsomedial-plantarolateral view or a flexed skyline view. This is performed by flexing the hindlimb and placing the x-ray plate along the plantar aspect of the proximal metatarsus, with the x-ray beam directed from above. Contrast radiography is useful to differentiate thoroughpin from false thoroughpin and may also outline soft tissue masses and adhesions within the sheath.
- Ultrasonography may also be useful to differentiate swelling of the tarsal sheath from false thoroughpin and is also important to assess the deep digital flexor tendon for lesions and to identify adhesions within the sheath.

DIFFERENTIAL DIAGNOSIS

- False thoroughpin
- Curb
- Gastrocnemius tendonitis

TREATMENT

- In acute cases of trauma, cold hosing and antiinflammatory drugs such as phenylbutazone are indicated.
- In more chronic cases, drainage of the fluid and injections of long-acting corticosteroids will generally reduce the swelling and alleviate lameness for varying periods. Arthroscopic exploration and debridement is indicated if soft tissue masses are detected on ultrasound examination and may also allow adhesions to be broken down and exostosis of the sustentaculum tali to be removed. The prognosis for return to soundness is poor in horses with bony lesions of the sustentaculum tali.

Tibial Fractures

Common fractures of the tibia include catastrophic diaphyseal fractures, tibial tuberosity fractures, fractures of the proximal physis, and incomplete tibial stress fractures. Tibial tuberosity fractures tend to occur after direct trauma to the stifle. Proximal physeal fractures also tend to follow trauma in young horses. These are nearly always Salter type II fractures with the metaphyseal fragment on the lateral aspect.

KEY POINT

Tibial stress fractures affect young horses in race training and can occur in any location but most commonly affect the lateral aspect of the proximal tibia.

Complete diaphyseal fractures in adults often are comminuted and open and therefore have a hopeless prognosis. In younger horses, fractures tend to be oblique or spiral and may be candidates for internal fixation.

HISTORY AND PRESENTING SIGNS

- Non-weightbearing lameness of sudden onset
- Visible alteration in the tibial contour
- Hindlimb lameness in a racehorse

CLINICAL FINDINGS AND DIAGNOSIS

Diaphyseal and Physeal Fractures

- Non-weightbearing lameness
- Swelling of overlying soft tissues
- Lateral angulation of the limb distal to the fracture
- Diaphyseal fractures often have skin penetration medially
- Lateromedial and craniocaudal radiographs will demonstrate the fracture

Tibial Tuberosity Fractures

- Severe lameness with swelling over proximal cranial tibia
- Effusion of the femoropatellar joint
- In young horses, radiographs of contralateral limb will help differentiate fractures from normal physis

Stress Fractures

- Hindlimb lameness with minimal localizing signs
- May be pain on palpation or rotation of tibia
- Lameness fails to block out with regional anesthesia
- Fracture lines are often not visible on radiographs but fracture callus may be observed
- Increased uptake of radioisotope in the area of the fracture will be observed on scintigraphy

DIFFERENTIAL DIAGNOSIS

- Femoral fractures
- Patellar fractures
- Stifle trauma
- Other causes of hindlimb lameness

TREATMENT

- Because of the very poor prognosis for diaphyseal fractures in adults, euthanasia should be considered. In foals, diaphyseal fractures are treated by double plate fixation. The prognosis is good for foals less than 4 months of age.
- Proximal physeal fractures have a relatively good prognosis. In neonates, cross-pinning may be used, whereas in older foals medial plate fixation is required.
- Tibial tuberosity fractures are generally repaired with a narrow dynamic compression plate and have a good prognosis provided the implants do not weaken the fragment excessively.
- Horses with tibial stress fractures require 4 to 6 months' rest. Scintigraphy should be repeated before commencing training.

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STIFLE AND HIP PROBLEMS

Fibrotic and/or Ossifying Myopathy

Fibrotic myopathy is thought to occur after injury to the semimembranosus or semitendinosus muscles. It results in a characteristic gait with a shortened cranial phase of stride and a sudden downward movement of the hoof immediately before placement on the ground. It is more common in horses used for cutting and roping. Falls, wire cuts, and intramuscular injections are possible causes. More recently, denervation atrophy of these muscles has also been implicated as a cause, and this may be a sequel to pelvic fracture with damage to the sciatic nerve.

HISTORY AND PRESENTING SIGNS

- Trauma or intramuscular injection
- Unusual hindleg gait

CLINICAL FINDINGS AND DIAGNOSIS

- Unilateral gait abnormality with shortened cranial phase of stride and stamping of the foot before weight bearing.
- Palpation of the affected muscles reveals a firm to hard area, usually in the middle to distal regions of the semitendinosus muscles. Usually there is no pain found on palpation of these areas.

DIFFERENTIAL DIAGNOSIS

- Neurologic problems
- Unilateral stringhalt

TREATMENT

- Surgical treatment is necessary to resolve the gait abnormality.

KEY POINT

Simple transection of the tendon of the semitendinosus muscle near its insertion on the caudomedial region of the proximal tibia will give good results in most cases of fibrotic myopathy.

Subchondral Bone Cysts of the Stifle

Subchondral bone cysts can occur in a number of sites in the stifle joint, but the medial femoral condyle appears to be predisposed. Subchondral bone cysts occur in high weight-bearing areas of joints secondary to osteochondral defects and have been experimentally induced at this site in the horse using artificially created cartilage and bone defects. The cause of the initial defect in clinical cases is unknown.

HISTORY AND PRESENTING SIGNS

- Young horse, usually 2 years old
- Hindlimb lameness

CLINICAL FINDINGS AND DIAGNOSIS

- Most commonly the hindleg lameness is unilateral. The degree of lameness can be quite variable and often does not correlate with the size of the cyst.

KEY POINT

Effusion of the medial femorotibial joint is rare, but approximately 50% of horses will have some femoropatellar effusion.

- The response to a flexion test is inconsistent.
- Local analgesia of the medial femorotibial joint is variable, and this is probably due to subchondral bone pain.
- Good-quality radiographs are essential, particularly a caudocranial view of the stifle. If the films are slightly underexposed, it is possible to miss the presence of a bone cyst. A flexed lateromedial view is a useful alternative. Close examination of the articular surface of the medial femoral condyle will usually reveal some degree of flattening, and a radiolucent area, often up to 2 to 3 cm (1 inch) in diameter, can be seen extending within the medial condyle.
- Although it is usual to have the condition presenting as a unilateral hindleg lameness, it is common to have lesions in both stifle joints. Therefore, it is essential that radiographs be taken of the apparently unaffected stifle.

DIFFERENTIAL DIAGNOSIS

- OCD of the stifle
- Hock lamenesses
- Ligament and meniscal injuries to the stifle

TREATMENT

- In small bone cysts causing little or no lameness, conservative therapy involving pasture rest for 6 to 12 months may result in a horse that is sound for athletic purposes.
- Surgical treatment is recommended for larger cysts because conservative treatment tends to be disappointing.

KEY POINT

Surgical treatment involves visualization of the medial femoral condyle with an arthroscope and curettage of the cavity of the cyst. Forage of the cyst rim, which used to be advocated, is no longer recommended because of evidence that it promotes cyst enlargement.

- Horses require 6 to 12 months of rest after surgery. Reported success rates range from 60 to 70%.

Gonitis: Stifle Lameness

Gonitis is the term used to describe inflammation of the stifle joint because of a variety of disorders. It is a general term indicating the region of pathology only and may reflect soft-tissue or bony lesions, although the latter are more common.

HISTORY AND PRESENTING SIGNS

- Hindleg lameness
- Distention of the femoropatellar pouches

CLINICAL FINDINGS AND DIAGNOSIS

- Distention of the femoropatellar pouches is characteristic.
- If there is any suggestion of a septic arthritis, a synovial fluid sample should be taken for a white cell count and total protein determination.
- Affected horses will show varying degrees of lameness, depending on the specific problem.
- Various techniques for manipulation of the stifle can be undertaken, but these give the investigator a great chance of being injured and seldom reveal worthwhile information.
- Intra-articular local anesthesia (see Figs. 4-47 and 4-48) may be used to confirm the site.
- Radiography of the stifle is essential and should include a minimum of caudocranial and lateral views. A flexed lateral view is also useful to assess the apex of the patella and the femoral condyles if a craniocaudal view is not possible. Also, a caudolateral-craniomedial view may show more subtle OCD lesions of the lateral trochlear ridge of the femur. If there is a history of trauma to the cranial aspect of the stifle, a flexed skyline view of the patella should be taken; otherwise, medial pole fractures will be missed. It is important to understand that a significant number of joints with no radiographic changes have soft tissue lesions.
- Ultrasound examination of the patellar ligaments, collateral ligaments, and abaxial parts of the menisci is important to establish the presence of soft tissue lesions.
- Arthroscopic examination of both the femorotibial and femoropatellar joints often is useful for assessing the menisci, cruciate ligaments, and cranial meniscal ligaments.

DIFFERENTIAL DIAGNOSIS

- Damage to medial or lateral collateral ligaments
- Meniscal damage
- Cruciate ligament damage
- OCD
- Subchondral bone cysts
- Tibial eminence fractures
- Septic arthritis
- Fractures of the patella
- Osteoarthritis

TREATMENT

- Treatment is dependent on the cause of the swelling.

KEY POINT

OCD lesions respond well to debridement of diseased cartilage and subchondral bone. Meniscal tears may respond to arthroscopic debridement of the meniscus if not too severe. The prognosis for more extensive meniscal damage or rupture of the cruciate ligaments is poor.

- Tibial eminence fractures can be removed arthroscopically, and the prognosis is good provided there is no damage to the cranial cruciate ligament.
- Small fragments of the base of the patella can be treated conservatively with a good prognosis. Fractures of the medial pole can be removed by arthrotomy and have a good prognosis for athletic soundness. Fragmentation of the apex of the patella is treated by arthroscopic debridement and has a good prognosis.
- Rest periods of up to 12 months may give resolution of osteochondritis, meniscal damage, and ligament rupture.
- Depending on the etiology of the gonitis, it may be possible to remove osteochondral fragments or gain access to the joint for other surgical treatments using arthroscopy.

Osteochondrosis Dissecans of the Stifle Joint

OCD affecting the stifle is a common condition, particularly in young Thoroughbred horses. The most common site affected is the lateral trochlear ridge of the femur.

HISTORY AND PRESENTING SIGNS

- Hindleg lameness
- Effusion of the femoropatellar joint capsule
- Horses 2 years old or less are usually presented

CLINICAL FINDINGS AND DIAGNOSIS

- Variable hindleg lameness that may be bilateral
- Obvious effusion involving the femoropatellar joint
- Effusion is usually bilateral, with lesions involving both stifle joints.
- Standard radiographic views should be taken. Lesions are most easily seen on the lateral view, and the lateral trochlear ridge and patella are most commonly affected.

DIFFERENTIAL DIAGNOSIS

- Meniscal injuries
- Collateral ligament injuries
- Cruciate ligament injuries
- Bone cysts

TREATMENT

KEY POINT

Conservative therapy may be considered in yearlings and should consist of stall rest and reduced digestible energy intake. If clinical signs progress, surgery is indicated. In older horses, arthroscopic surgery to debride the affected articular cartilage and subchondral bone is the treatment of choice.

- The prognosis depends on the extent of the lesion. Lesions localized to the lateral trochlear ridge have a good prognosis.
- A prolonged rest period of up to 6 months usually is required after surgery.

Tumoral Calcinosis (Calcinosis Circumscripta)

Tumoral calcinosis is a condition in which one or sometimes more circumscribed hard swellings are found. It is characterized by the formation of amorphous, calcified, granular deposits in the subcutaneous tissues that induce a granulomatous reaction. In most cases these are located over the lateral aspect of the proximal tibia, just distal to the stifle joint. Other sites are occasionally involved, and sometimes the lesions occur bilaterally. They are usually first noticed in weanlings or yearlings. The condition usually does not cause lameness.

HISTORY AND PRESENTING SIGNS

- Large firm swelling on the lateral aspect of the proximal tibia

CLINICAL FINDINGS AND DIAGNOSIS

KEY POINT

The typical appearance of a round hard swelling over the lateral aspect of the proximal tibia is very characteristic of tumoral calcinosis.

- The diameter of the swellings is approximately 3 to 12 cm (1-5 inches), and they are immobile.
- There is usually no lameness.
- If radiographs are taken, there will be a circumscribed radiopaque mass that has a somewhat granular appearance.
- If fine-needle aspiration is attempted, this will

indicate that the mass is extremely hard and calcified, and it will not be possible to obtain a sample.

DIFFERENTIAL DIAGNOSIS

- Soft-tissue abscess
- Foreign body
- Neoplasia

TREATMENT

- Surgery is the only feasible treatment, and although most are easily dissected, in many cases they will attach to the joint capsule of the stifle and are impossible to remove without entering the joint.

D KEY POINT

Because tumoral calcinosis causes only a cosmetic blemish, it is probably best left untreated except in cases in which lameness can be attributed directly to the lesion. If surgery is thought necessary, the owner should be made fully aware of the potential complications.

Upward Fixation of the Patella ("Locking Stifle")

Upward fixation of the patella is a condition that is found commonly in Shetland ponies and Standardbreds. It may be related to an excessively straight stifle conformation or the result of inadequate quadriceps muscle tone. Often it is seen in performance horses after a rest period and will resolve in most cases with increased fitness.

HISTORY AND PRESENTING SIGNS

- Inability of the horse to flex the limb
- Dragging of toe when the limb is advanced, with sudden hyperflexion
- Occurs when led out of the stall

CLINICAL FINDINGS AND DIAGNOSIS

- The typical gait of a horse with upward fixation of the patella is that the limb locks in extension and cannot be moved forward. The result of this is that the toe is dragged as the horse tries to move forward.
- This may occur in one or both hindlimbs.
- If the patella then unlocks, the hindlimb may suddenly hyperflex, so confusion with stringhalt is possible by horse trainers and owners.
- In milder cases it may not be possible to positively demonstrate the condition, which is intermittent, and reliance may have to be placed on the history.

DIFFERENTIAL DIAGNOSIS

- Stringhalt
- Damage to the proximal portion of the extensor tendons with adhesion formation
- Fibrotic myopathy

TREATMENT

- In most horses, increased fitness will result in resolution. Exercise up hills, which develops the quadriceps muscle group, may aid resolution.
- However, where the upward fixation occurs very frequently or fails to respond to conservative treatment, surgical treatment (medial patellar ligament desmotomy) is indicated. This is best performed under sedation using xylazine (Treatment No. 108) or detomidine (Treatment No. 28) and butorphanol (Treatment No. 15) (see Chapter 19) and local infiltration with 5 to 10 mL of mepivacaine (Treatment No. 72). A 2.5-cm (1-inch) incision is made between the middle and medial patellar ligaments, just proximal to the tibial crest. After a sharp stab incision is made in the fascia, a pair of blunt-pointed scissors is pushed through the fascia between the ligaments. This enables a blunt-nosed bistoury to be used to sever all fibers of the medial patellar ligament. Only one or two skin sutures are necessary, and the subcutaneous tissue is left unsutured. After surgery, horses should be rested for 1 month before resuming training.

KEY POINT

Because there have been several reports of osteochondral fragmentation of the apex of the patella after medial patellar desmotomy, conservative treatment should always be tried, with surgery reserved for severe nonresponsive cases.

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HIP AND PELVIS PROBLEMS

Femoral Fractures

Femoral fractures are a relatively common long bone fracture, particularly in foals. In younger foals, fractures may involve the distal growth plate. Femoral fractures in adults often are comminuted and repair is unlikely to be successful.

HISTORY AND PRESENTING SIGNS

- History of severe trauma or anesthesia
- Non-weightbearing lameness
- In some cases, a history of a loud crack or bang, like a gun going off

CLINICAL FINDINGS AND DIAGNOSIS

- Non-weightbearing lameness on affected hindleg
- The affected hindleg may appear shorter than the unaffected hindleg because of the overriding of the proximal and distal fragments and the comminution.

KEY POINT
With femoral fractures, crepitus may or may not be felt on manipulation of the leg, but if the area is auscultated with a stethoscope, crepitus usually can be heard.

- The fracture can usually be diagnosed from the clinical examination. Radiographs are indicated in foals, where attempts at surgical repair may be considered. In most adults, the prognosis is so poor that it is not worthwhile considering surgery. Therefore, radiographs may not be of economic benefit because general anesthesia is necessary.

DIFFERENTIAL DIAGNOSIS

- Fracture of the tibia
- Fracture of the patella
- Dislocation of the hip

TREATMENT

- Because of difficult access and extensive comminution, repair of femoral fractures in adults carries a hopeless prognosis. Conservative management in foals may allow healing, and good results may be achieved if the fractures is minimally displaced. Displaced fractures often result in a shorter and rotationally deformed limb. Surgical management involves the application of lateral and cranial broad dynamic compression plates and should be reserved for foals less than 3 months of age.

Dislocation of the Hip Joint

The hip joint is maintained by the round and accessory ligaments. If these ligaments are ruptured, dislocation of the hip joint will occur. This type of injury occurs secondary to trauma but is quite uncommon. In some cases of dislocation of the hip joint in ponies, it may be accompanied by upward fixation of the patella. Rarely, the round ligament is ruptured but the hip joint does not dislocate. The result of the instability is degenerative arthritis of the hip joint.

HISTORY AND CLINICAL SIGNS

- History of trauma or slipping on the leg
- Hindleg lameness
- Turning outward of the toe and stifle of the affected leg
- Inability to advance the hindleg

CLINICAL FINDINGS AND DIAGNOSIS

- Rupture of the round ligament of the hip joint is associated with shortening of the hindleg, with the stifle and toe rotated outward.
- Complete dislocation of the hip joint may not occur. When it does, there is a marked effect on gait, with the horse being reluctant to bear weight.
- The femur is rotated outward and the greater trochanter is more prominent than usual.
- Radiographs can be used to confirm the diagnosis, but it can be difficult to obtain views that demonstrate the dislocation. Standing films can be obtained (see earlier section on Radiography).

DIFFERENTIAL DIAGNOSIS

- Fractures of the femur
- Fractures of the pelvis
- Upward fixation of the patella

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TREATMENT

- Relocation of the hip joint may be attempted under general anesthesia, but this is difficult, and luxation is likely to recur. Open reduction has been described but is only an option in ponies or foals. Femoral head resection is also an option for achieving paddock soundness in ponies.

Pelvic Fractures

Pelvic fractures may occur due to direct trauma, but in racing horses they are mostly stress fractures. Fracture of the tuber coxae can occur when the prominence is caught on the gate or stable door. Most pelvic problems will resolve with time, but some fractures will produce a long-standing lameness.

HISTORY AND PRESENTING SIGNS

- History of trauma
- Hindleg lameness
- Change in contour of the pelvic region

CLINICAL FINDINGS AND DIAGNOSIS

- External swelling is generally only observed with fractures of the tuber coxae. There will generally be asymmetry of the pelvic prominences. The degree of lameness is variable, depending on the site of the fracture and whether the acetabulum is involved. Incomplete stress fractures of the ilium generally present with low-grade lameness.
- Chronic fractures will result in gluteal muscle atrophy. On rectal examination it may be possible to detect crepitus or changes in pelvic canal contour. Ultrasonography in the standing horse will generally confirm the diagnosis. Loss of continuity of the bone surface is detected or a change in the contour due to callus formation.
- Radiographs of the pelvis are possible in the standing horse but require a powerful x-ray machine. General anesthesia allows more extensive evaluation of the pelvis with radiographs.
- If the acetabulum is not involved, most pelvic fractures will heal satisfactorily in a period of 12 months. However, a few horses will be left with a chronic hindleg lameness even 12 months after a pelvic fracture. For this reason, a guarded prognosis should be given. Fractures of the acetabulum carry a worse prognosis, but a successful outcome is still possible if there is minimal comminution.

DIFFERENTIAL DIAGNOSIS

- Muscle atrophy due to other causes of hindlimb lameness
- Sacroiliac luxation

TREATMENT

- Rest is the only treatment possible. Three to four months in a stall is recommended initially, and periods of up to 12 months' rest may be necessary before lameness resolves.

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Back Abnormalities

Back problems make up only a small proportion of musculoskeletal problems in horses, accounting for less than 1 % of cases in most veterinary practices. However, in practices where there is a large proportion of pleasure, dressage, eventing, and hunting horses, back injuries are more common. Back problems are also more common in Standardbred trotters and pacers than in Thoroughbred gallopers. One of the major difficulties in establishing a diagnosis in horses with suspected back problems is achieving adequate radiographs of the region. Apart from radiographing the thoracic dorsal spinous processes, which can be achieved with most x-ray machines, those with a capability of more than 120 kVp and 500 mA are needed for radiographs to be taken of other areas of the back.

In some dressage horses, the rider may complain of various temperament problems, but the veterinarian may find nothing wrong. There may be subclinical back problems, but in some cases it may simply be that the horse is not responding well to the schooling or is being given conflicting signals by the rider. Such horses often present as "head shakers."

HISTORY AND PRESENTING SIGNS

- History of fall or trauma
- Temperament changes

- Poor performance
- Resentment of saddling
- Adverse reactions to being groomed
- Hindleg lameness
- Adverse reactions to a heavy rider

CLINICAL FINDINGS AND DIAGNOSIS

- The most consistent history is of a change in the temperament of the horse, with or without a loss of performance.
- The horse also may resent weight on its back and show signs of hindlimb lameness or reluctance to "stretch out."

KEY POINT

Examination should concentrate on evaluating the flexibility of the back together with assessment of any areas of asymmetry. Particular note should be taken of any excessively prominent spinous processes; deformities of the spinal column such as lordosis (sway back), kyphosis (roach back), or scoliosis (lateral deviation); and areas of hypersensitivity.

- Many horses with chronic back pain have wastage of the longissimus muscles. Muscle spasm is also a common feature and may be elicited with palpation.
- Firm pressure should be applied over the dorsal midline, palpating over each spinous process to determine if there is a localized area of pain. After this, the area over the longissimus dorsi muscles should be palpated at intervals of 2 to 3 cm (1 inch). Hypersensitivity may be found, or the horse may attempt to kick, indicating pain.
- Stroking over the dorsal midline with the blunt end of a pen will result in the horse crouching away (ventroflexion) from the pen, particularly over the thoracolumbar region. Over the caudal sacral region, firm stroking with a pen will result in the horse arching (dorsiflexion) its back. Stroking over the longissimus dorsi muscles normally results in lateral flexion of the thoracic spine away from the side being stroked. These responses may be abolished or modified when there are back problems, because movement of the back causes pain.
- Palpation over the tuber sacrale should be performed to determine if any hypersensitivity is present, which may indicate sacroiliac pain or pelvic stress fracture. Pain in this area is manifested as the horse flexing its hindlegs and crouching toward the ground when the region over the tuber sacrale has firm pressure applied. In some horses, there may be evidence of shifting of the sacrum, with the tuber sacrale being higher on one side than the other.
- If a localized area of pain is found, particularly over the thoracic spinous processes, it may be helpful to deposit 10 mL of a local anesthetic such as 2% prilocaine (Treatment No. 93) or mepivacaine (Treatment No. 72) to determine if there is an improvement in gait or in temperament.
- Radiographs may be indicated in some cases. The thoracic dorsal spinous processes are relatively easily radiographed. However, for radiographs of the vertebral bodies or the sacrum, general anesthesia is required together with a large-capacity x-ray machine.
- Rectal examination should be performed as a routine, although in most cases there are negative findings.

DIFFERENTIAL DIAGNOSIS

- Rhabdomyolysis ("tying up")
- Hindlimb lameness
- Temperament problems
- Rider/horse incompatibility
- Pelvic stress fracture

TREATMENT

- Checking on the equipment used, particularly the fitting of the saddle, is important because an ill-fitting saddle may result in a chronic back problem.
- Relief of muscle spasm is a major priority of therapy. A number of techniques can be used and these include acupuncture, manipulation, and corticosteroid injection. Poorly muscled horses should then undergo an exercise program consisting of unridden work to build up the muscle mass.
- Rest is also an alternative with prolonged periods of up to 12 months being necessary. Some cases of soft tissue injury to the back may be assisted by a course of oral phenylbutazone (Treatment No. 88) for several weeks, at a dose rate of 2.2 mg/kg once daily. In cases of sacroiliac pain, even prolonged rest may not result in the anticipated improvement. However, soft-tissue back injuries, particularly those which are muscular, may respond well.
- If there is evidence of overriding of the thoracic dorsal spinous processes, surgery is possible to resect one or more of the processes. The results of this surgery are not always predictable. Before such treatment is contemplated, it is im-

portant to see an improvement after infusion of local anesthetic into the affected area.
- Standardbred pacers with sacroiliac pain have been assisted by a change in training. Galloping these horses instead of hobbling them for their fast work may result in improvement in the clinical signs.

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General Musculoskeletal Abnormalities

Arthritis, Osteoarthritis, or Degenerative Joint Disease

Osteoarthritis (degenerative joint disease, DJD) is a common condition of all types of horses. In performance horses it predominantly affects the high-motion joints such as the carpal and fetlock joints. In pleasure horses it is more common in the low-motion joints such as the distal tarsal and proximal interphalangeal joints. Initiating factors include stress failure of subchondral bone due to repetitive loading, synovial inflammation due to trauma, and joint instability subsequent to ligament or meniscal damage. The degenerative process is perpetuated by bone and cartilage fragmentation, release of neurotransmitters and cytokines, and poor synovial perfusion due to increased joint pressures from effusion. This results in synovial proliferation and articular cartilage degeneration. Articular cartilage has limited healing potential and therefore its loss is irreversible. Severe cases in low-motion joints may result in arthrodesis.

HISTORY AND CLINICAL FINDINGS

- The most common clinical sign is lameness in the affected limb, with or without some degree of swelling around the joint.
- In high-motion joints there is usually some distension of the joint capsule and pain on extreme flexion of the joint. The joint capsule distension is best determined by direct palpation and comparison with the opposite joint. In low motion joints, effusion is often difficult to detect, but in advanced cases firm swelling may be present due to periarticular modeling of bone. In many cases, intra-articular analgesia is the only method of localizing lameness to these joints.
- The lameness may be aggravated by flexion of the joint for 1 to 2 minutes, after which the horse is trotted off.

DIAGNOSTIC TESTS

- Intra-articular local anesthesia is important to confirm the particular joint as the site of lameness, but pain associated with subchondral bone may not respond. Details of arthrocentesis are shown in Figures 4-24 to 4-49.
- Radiology is the most useful aid to diagnosis. The degree of narrowing of the joint space, the amount of periarticular bone modeling, and any calcification associated with origins or insertions of tendons and ligaments should be assessed. The dorsopalmar view is usually the best for assessing any reduction in the joint space, and it should be ensured that the primary beam is centered on the affected joint. Oblique views are often necessary to discern periarticular bone modeling. The lateral view of an affected joint is helpful to assess the extent of soft tissue swelling and changes in articular contours.
- Scintigraphy is useful, particularly in early cases where subchondral and periarticular bone modeling and remodeling are active. It is important to understand that a degree of subchondral bone modeling is normal in heavily exercising horses.

TREATMENT

The aim of therapy should be to reduce inflammation, neutralize cytokines and enzymes, and encourage synthesis of cartilage components, without any detrimental effect on the cartilage. At present there are no therapies that result in repair of articular cartilage. The best result is to delay the progression of the disease process while alleviating the signs and allowing the horse to be used for the purpose intended. If acute signs are present, rest may be desirable in combination with anti-inflammatory therapy, but in chronic progressive cases, rest tends to be of less use.

Hyaluronate (Treatment No. 60)

The mechanism of action of hyaluronate is unknown. Its major mode of action is as an anti-inflammatory, although its action is mild in comparison with corticosteroids. In equine joints it has been shown to reduce prostaglandin E_2 levels and to reduce inflammatory cell infiltration. It has little

beneficial effect on articular cartilage, but no detrimental effects have been observed. Hyaluronate is available in intra-articular and intravenous forms. There is some evidence that the intravenous form is more effective than the intra-articular form. There is also evidence that hyaluronate reduces the loss of proteoglycan from the articular cartilage that is associated with intra-articular corticosteroid administration when used in combination. On its own it appears to be most useful in cases with mild to moderate synovitis.

Intra-articular Corticosteroids (Treatment Nos. 12, 13, 74, 106)

Despite the availability of a range of therapeutic agents for osteoarthritis, the corticosteroids remain the most commonly used and useful intra-articular agents. Corticosteroids act at steroid specific receptors in cell cytoplasm to inhibit phospholipase A_2 and therefore both the cyclooxygenase and lipoxygenase pathways of the arachidonic acid cascade. Controversy exists over the use of corticosteroids in equine joints. This is primarily because adverse affects can be demonstrated in normal joints. A number of studies have demonstrated decreased proteoglycan concentrations in articular cartilage after corticosteroid injections, and at very high dose rates, chondrocyte necrosis and articular cartilage fibrillation occur.

KEY POINT

Although adverse effects have been attributed to intra-articular corticosteroids, more recent studies using therapeutic doses in models of osteoarthritis have demonstrated beneficial effects. In these models, corticosteroids reduce synovial inflammation, increase hyaluronate and proteoglycan synthesis and slow the progression of osteoarthritis.

Therefore, in cases of osteoarthritis, the corticosteroids are an effective treatment provided they are used at therapeutic dose rates. They are particularly useful in advanced cases of osteoarthritis where other therapeutic agents tend to give less reliable results. The duration of response varies tremendously between individual horses and is also dependent on the preparation used. Methylprednisolone acetate is a long-acting agent, betamethasone acetate has an intermediate duration of action, and dexamethasone is a short-acting agent. Care must be taken when using long-acting preparations in horses subject to drug testing because these drugs have prolonged excretion times, sometimes for up to 2 months after a single dose.

Polysulfated Glycosaminoglycans (Treatment No. 90)

Polysulfated glycosaminoglycans (PSGAGs) are synthetic heparinoids that have been shown to have therapeutic application in osteoarthritis. The mechanism of action of these drugs is unknown, but they have been shown to have anti-inflammatory properties, to inhibit cartilage degrading enzymes, to stimulate hyaluronate, and to promote proteoglycan synthesis. Septic arthritis has been reported after intra-articular administration of PSGAGs, and the drug has been shown to increase the infectivity of *Staphylococcus aureus.* For this reason it is now more commonly administered by the intramuscular route.

Pentosan Polysulfate

Like PSGAGs, pentosan polysulfate is a synthetic heparinoid that was previously used as an anticoagulant in human medicine. Its mode of action is also unknown, but in experimental animals it has been shown to stimulate hyaluronate and proteoglycan synthesis. Hyaluronate of increased molecular weight has been demonstrated in joint fluid after treatment. Inhibition of proteolytic enzymes such as stromelysin and decreased concentrations of tumor necrosis factor have been demonstrated. It has also been suggested that it may improve subchondral blood flow due to its anticoagulant/fibrinolytic activity. In the horse it has been used both intra-articularly and intramuscularly. In the dog an oral preparation also has been shown to be effective. Clinical experience suggests that it is highly effective in some cases of early osteoarthritis, whereas in longer standing cases it has little effect.

Topical Application of Dimethylsulfoxide (Treatment No. 34)

Dimethylsulfoxide (DMSO) is a hydroxyl scavenger with some direct anti-inflammatory properties. It is applied topically to the affected joint and appears to be useful in all stages of arthritis. It can be used in conjunction with intra-articular treatments. It appears to be of most use in cases of synovitis. Because DMSO acts as a vehicle to take other products through the skin, some veterinarians choose to mix this product with other anti-inflammatory drugs, such as phenylbutazone and flumethasone. In some countries, there are commercial preparations of DMSO combinations that can be applied topically.

Other treatments that are used include systemic anti-inflammatory and analgesic drugs such as the nonsteroidal anti-inflammatories (see Chapter 19).

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Azoturia

See Rhabdomyolysis.

Cellulitis

Cellulitis is an infection of the soft tissues of the limb that generally occurs after penetrating skin injuries. Many cases will present with no evidence of a skin wound. This may be because the wound is extremely small. It has been speculated that hematogenous spread is possible. In certain geographic areas, spider bites have been implicated. Cellulitis can also be associated with more open wounds.

HISTORY AND PRESENTING SIGNS

- Acute limb lameness
- Swelling of the affected limb
- History of trauma

CLINICAL FINDINGS AND DIAGNOSIS

- Swelling and heat associated with soft-tissue inflammation in the affected area of the leg, usually the distal limb.
- The degree of lameness depends on the severity of the cellulitis and can range from mild to severe.
- Radiographs may be necessary to ensure no changes in bony structures underlying the area of cellulitis. Ultrasound is useful to evaluate the structures involved including tendons, tendon sheaths, and joint capsules. Abscess formation also may be detected. If involvement of synovial cavities is suspected, a synovial fluid sample should be retrieved preferably from a site remote to the subcutaneous infection.

DIFFERENTIAL DIAGNOSIS

- Osteomyelitis/osteitis
- Tendon sheath infection
- Septic arthritis

TREATMENT

- Where the cellulitis is localized, local wound treatment using saline irrigation and povidone-iodine (see Disinfectants, Chapter 19) is effective in resolving the problem.
- Where there is more extensive tissue involvement, antimicrobial therapy is indicated. A combination of penicillin and gentamicin usually is effective. This should be combined with nonsteroidal anti-inflammatory therapy and pressure bandaging to reduce swelling. Abscesses should be drained surgically. Walking exercise should be commenced as soon as possible to improve venous and lymphatic drainage.

Fistulous Withers

Fistulous withers is the term used to describe supraspinous bursitis, due mostly to localized infection with *Brucella abortus.* Occasionally, the organism will localize in the atlantal bursa, causing "poll evil." These conditions are seen quite infrequently in veterinary practice today. However, it is still a difficult condition to treat successfully, because many cases tend to recur due to infection localizing in various tissues surrounding the bursa.

HISTORY AND CLINICAL FINDINGS

- The first sign of fistulous withers is sensitivity over the wither region together with swelling.
- The swelling is usually localized and fluid filled, although occasionally it is more diffuse.
- After a variable time course, a discharging sinus usually develops, with a purulent discharge.

DIAGNOSTIC TESTS

- Diagnosis is possible from the clinical signs, although serum may be taken for *antibody titers* and purulent material aspirated for *microbiology.*
- In cases where there is a discharging sinus, swabs for microbiology are not worthwhile because a range of bacteria will be cultured. However, in the early stages of the condition, before development of a discharging sinus, it is possible to aspirate fluid from the supraspinous bursa for culture.

DIFFERENTIAL DIAGNOSIS

- Trauma
- Osteomyelitis
- Foreign body

TREATMENT

- If the condition is diagnosed before the discharging sinus develops, administration of oxytetracycline (Treatment No. 80) at a dose rate of 3 to 5 mg/kg twice daily for 7 days may achieve resolution. However, after a discharging sinus has developed, there is seldom a response to antibiotic therapy.
- Radical surgery to establish effective drainage is the only useful treatment, but the success rate

is seldom better than 50%. Care must be taken regarding the discharge that contains *Brucella abortus* in low numbers. In particular, zoonotic infections should be avoided by rigorous hygiene.

Joint-Ill

See Septic Arthritis.

Lymphangitis

Lymphangitis is a condition that results in swelling of the leg and is thought to be due to a restriction in lymphatic flow, possibly due to a bacterial infection. *Corynebacterium pseudotuberculosis* has been cultured from affected horses, but in many cases bacterial culture will be negative. It can sometimes occur in outbreak form.

HISTORY AND CLINICAL FINDINGS

KEY POINT

By far, most cases affected involve the hindlimb(s), with swelling being apparent usually as far proximally as the hock and, occasionally, the stifle. In some cases, the distal limb may be enlarged up to two to three times normal size.

- Most horses will not show severe lameness of the affected limb, although they may show discomfort.
- There is usually no temperature rise or change in other cardinal signs.
- In most cases it is impossible to establish the causative agent.

DIAGNOSTIC TESTS

- Ultrasonography, if performed, will reveal subcutaneous fluid accumulation only. There is also a loss of definition of some of the structures.
- Bacteriology may be useful if a fluid sample can be aspirated aseptically. In most cases, however, it is impossible to procure an adequate sample for bacteriology.

DIFFERENTIAL DIAGNOSIS

- Infection of the tendon sheaths
- Septic arthritis
- Cellulitis

TREATMENT

- These cases should be treated with broad-spectrum antibiotics, systemic nonsteroidal anti-inflammatory drugs, and walking exercise. Pressure bandaging of the lower limb is also helpful. The response to treatment is often very slow. Even though the initial pain and lameness may resolve within a few days, the swelling can persist for many weeks and often recurs.
- Lymphangitis is a frustrating condition to treat.

Nutritional Secondary Hyperparathyroidism ("Big Head")

This disease is most common in horses grazing pastures that are predominant in tropical grasses such as buffel grass, kikuyu, and setaria. These grasses contain oxalates that bind calcium, making it unavailable for absorption. Similar effects can be found from horses being fed high grain rations or diets with excessive phosphorus.

HISTORY AND CLINICAL FINDINGS

- A shifting lameness may be the first sign of a problem, followed by reluctance to move, and, in severe cases, recumbency.
- Affected horses develop swellings of the maxilla and mandible. Some horses may present with spontaneous fractures of long bones or failure of the suspensory apparatus at its insertions.

DIAGNOSTIC TESTS

Urinalysis will enable diagnosis of calcium deficiency or calcium-phosphorus imbalance by enabling assessment of dietary calcium level. The calcium concentration and specific gravity of urine should be determined and then the following equation should be used:

$$\text{Ca excretion (mol/mOsm)} = \frac{\text{urine Ca concentration (mmol/L)} \times 0.04}{\text{specific gravity} - 0.997}$$

Calcium excretion values greater than 15 mol/mOsm indicate adequate dietary calcium.

DIFFERENTIAL DIAGNOSIS

- Epiphysitis
- Osteochondrosis
- Hypertrophic pulmonary osteopathy

TREATMENT

- For horses grazing tropical grasses, a supplement of 1 kg of rock phosphate in 1.5 kg of

molasses may be effective in prevention and treatment.
- In horses receiving excessive grain, the use of 20 to 40 g calcium carbonate, administered daily in the feed, is usually effective.

Osteochondrosis

Osteochondrosis is a term used to describe developmental pathology of articular cartilage and subchondral bone in young horses. Despite extensive investigations, the cause remains unclear but is probably a result of excessive forces on weak bone and cartilage. Lesions include osteochondral fragmentation or flap formation. Subchondral cystic lesions may occur secondary to articular cartilage and subchondral bone damage and are not specific osteochondrosis lesions. It is commonly found in growing horses and presents typically in yearlings and 2 year olds. Standardbreds are the most commonly affected breed, but the condition also is found in most other breeds, particularly fast-growing athletic horses. A variety of joints have been reported to be affected, including the phalangeal, fetlock, carpal, shoulder, tarsal, and stifle joints. Joints within the vertebral column are also affected, most commonly the cervical spine.

KEY POINT

There are many factors involved in the development of osteochondrosis, and it is impossible to blame a single cause.

Diets that are high in energy and have calcium-phosphorus imbalance will result in a high incidence of osteochondrosis in a susceptible population. Furthermore, one study has shown that diets marginal in copper may contribute to a higher incidence of osteochondrosis lesions. Foals supplemented to give a dietary value of 55 ppm copper had fewer cartilage lesions than those fed a diet containing 15 ppm copper.

HISTORY AND CLINICAL FINDINGS

- Joint effusion is the most common presenting sign, but in the shoulder joint where swelling is difficult to detect, horses will present with lameness and no obvious swelling. In other joints lameness is variable but is generally mild.
- Most horses will present as yearlings or 2 year olds.
- It is common for a horse to have several joints involved, and it is important to examine the joint contralateral to the affected one.
- In osteochondrosis involving the cervical spine, the resulting instability may lead to neurologic disease, with horses sometimes presenting as "wobblers."

DIAGNOSTIC TESTS

- Radiographs are essential not only for diagnosis but also for prognosis.
- Arthroscopy is sometimes used as a diagnostic procedure, but in most cases radiographs are sufficient to establish the diagnosis.

DIFFERENTIAL DIAGNOSIS

- Degenerative joint disease
- Acute arthritis
- Traumatic osteochondral fragments

TREATMENT

- In young horses with early or mild lesions, stall rest and dietary changes may result in resolution of the condition. It is important to reduce the dietary energy, and it may be of value to supplement the diet with copper to a total value of 55 ppm.

KEY POINT

The treatment of choice in most cases is arthroscopic surgery to debride affected articular cartilage and subchondral bone.

- Where there are extensive areas of damaged articular cartilage, the prognosis for athletic soundness is quite poor.

Osteomyelitis/Osteitis

Osteomyelitis is inflammation and infection of bone and its marrow cavity, which may result from either a localized infection after trauma or hematogenous spread. *Osteitis* is an infection of bone only. Hematogenous osteomyelitis occurs in young foals at the physis or in subchondral bone due to the presence of sinusoids or dilated vessels in which there is slow blood flow. A wide range of bacteria may be identified by culture. Localized infection after trauma to the leg is particularly common in the metacarpus and metatarsus and usually involves only the cortical bone.

HISTORY AND CLINICAL FINDINGS

- In localized osteitis after trauma (e.g., a wire cut to the distal limb), the most consistent finding is a purulent discharging sinus with a nonhealing

wound. There is often a history of intermittent discharge with apparent resolution of the problem for a period, followed by recurrence of discharge.
- Horses with this type of bone infection are lame initially, but when the condition is chronic, do not usually show lameness.
- Hematogenous osteomyelitis in foals generally causes lameness and swelling, although this often only becomes apparent when the infection is advanced. Subchondral bone infection often results in concurrent septic arthritis. There also may be concomitant septic arthritis involving the joint closest to the affected physis.

DIAGNOSTIC TESTS

- Radiology is essential to confirm the diagnosis. Typical radiologic findings are a localized area of bone lysis with a surrounding area of sclerosis (increased bone density). There is usually a sequestrum of bone that is evident as an area of increased density within the area of bone lysis. Where the localized bone infection is the result of external trauma, there is usually substantial periosteal new bone growth associated with the area of bone lysis, which is usually located within the cortex.
- If there is infection of the growth plate, a 12-gauge needle may sometimes allow collection of a sample of material in an aseptic manner for bacterial culture and sensitivity.

KEY POINT

If a discharging sinus is present, bacterial culture of the discharge should not be performed because the bacteria cultured will be secondary invaders. The results of such cultures lead to antibiotics being chosen that are likely to be worthless.

DIFFERENTIAL DIAGNOSIS

- Foreign body
- Localized cellulitis
- Local wound infection

TREATMENT

KEY POINT

If a localized bone infection secondary to a limb wound is present, antibiotic therapy is of little or no value.

- If necrotic bone is present, surgical removal is the most appropriate therapy. This is a simple technique that involves following the sinus tract and curetting the area of infected bone. Samples of bone from the infected area at surgery should be taken for microbiology rather than bacteriologic swabs. The wound should be left open to allow continued drainage. Systemic antibiotic therapy will hasten resolution.
- In hematogenous osteomyelitis, it is very important to establish the specific causative bacteria and the antibiotic sensitivity patterns. Prolonged antibiotic therapy (see Chapter 19) for up to 3 to 4 weeks is necessary. If the horse is treated early in the course of the infection, there may be resolution. Surgery may be possible if the affected area is localized. Local injection of antibiotics directly into the affected area or regional perfusion with intramedullary antibiotics may also be tried.

Rhabdomyolysis ("Azoturia," Myoglobinuria, "Tying up," "Setfast")

Muscular problems are common in working horses and range from stiffness and mild cramps to recumbency with myoglobinuria. The terminology for these conditions has been variable and has included "Monday morning disease," "tying up," "setfast," and "azoturia." From the histopathologic findings on muscle biopsy samples, the term *rhabdomyolysis* is the most accurate. The basic mechanism for the condition remains unknown, although a common history in many cases is several days without exercise while fed on grain followed by vigorous exercise. It was thought that muscle glycogen accumulated during rest, and this was used during exercise with the production of excessive amounts of lactate. It now seems unlikely that this theory is correct, but the pathogenesis remains elusive. Clinical observations throughout the world have indicated that fillies and mares are affected much more commonly than stallions and geldings. Young horses may have only one or two attacks of the condition and no further problems, which is probably why clinicians have reported favorable responses to a variety of treatments. Single, acute episodes with significant muscle damage, myoglobinuria, and profound increases in muscle enzymes are found most commonly during endurance rides. The chronic intermittent form of the disease is the most difficult to manage. Recently, a genetic component to the disease has been reported.

KEY POINT

Studies from U.K. racing stables have indicated that chronic sodium and/or potassium deficiencies may be involved in the pathogenesis of chronic intermittent rhabdomyolysis.

These findings have been established from fractional excretion of urinary electrolytes, and there has been resolution after appropriate electrolyte supplementation. Although vitamin E deficiency has been incriminated in the etiology of rhabdomyolysis, it is now clear that vitamin E deficiency is not a cause of the classic disease.

HISTORY AND CLINICAL FINDINGS

- In mild cases (so-called tying up), hindleg stiffness and a shuffling hindleg gait are seen. There may be pain on palpation over the gluteal muscles, usually in both hindlegs. This form of the disease may occur when the horses have received very limited amounts of exercise.
- In some cases, horses may only need exposure to some form of stress (e.g., placing the horse in the starting barrier, transport, etc.). This type of problem is more common in younger horses, where there may be a psychogenic factor involved.
- In some milder cases of rhabdomyolysis, poor performance may be the only feature of the disease.
- In more severe cases, there may be signs of severe pain with sweating, elevated heart rate, and reluctance to move. There also may be hard and painful locomotor muscles, the passage of dark-colored urine (myoglobinuria), and, occasionally, recumbency. This is more common in horses during protracted exercise, where significant fluid and electrolyte alterations occur.

DIAGNOSTIC TESTS

KEY POINT

Diagnosis is possible usually on clinical signs alone, but in mild cases it is helpful to collect a blood sample for estimation of muscle enzyme activities in the serum.

- The most useful measurements are creatine kinase (CK) and aspartate aminotransferase (AST). These enzymes will be greatly elevated over normal values (see Appendix 2, Hematology), and in severe cases the values may be greater than 100,000 U/L. More commonly in horses with the mild acute or chronic, intermittent form of the disease, values are in the range 1500 to 10,000 U/L.
- Horses with chronic, intermittent rhabdomyolysis frequently demonstrate increases in serum or plasma CK and AST after an exercise test (10-15 minutes of lunging). Blood samples should be collected within 30 minutes of exercise and 4 to 6 hours after exercise. A positive result is indicated if CK values double after exercise.
- Because recent research has shown that a number of horses with mild, chronic, intermittent rhabdomyolysis have electrolyte deficiencies, urinary electrolyte excretions should be determined. Alterations in the excretion of sodium, potassium, calcium, and phosphorus have been reported. Simultaneous collection of urine and blood for electrolyte and creatinine determinations should be performed, normal values must be established in the population studied because variations in feeding and management affect individual values.

DIFFERENTIAL DIAGNOSIS

- Aortoiliac thrombosis
- Colic
- Various causes of acute back pain
- Neurologic disease
- Laminitis
- Pleuropneumonia
- Muscle cramping

TREATMENT

A variety of different treatment regimens have been recommended, including thiamine, vitamin E and selenium, calcium borogluconate, anti-inflammatory drugs, tranquilizers, muscle relaxants, phenytoin, and dantrolene. In addition, many veterinarians have recommended the use of sodium bicarbonate in the feed to correct the purported acidosis. However, studies have demonstrated that metabolic acidosis is not a typical feature of horses with rhabdomyolysis, and bicarbonate use may not be justified.

Of all treatments used to treat rhabdomyolysis, few have been examined critically for their efficacy. The treatments that appear to give consistent results are as follows.

Severe Cases. Intravenous fluids (see Fluid Therapy, Chapter 19) should be used if any signs of shock are present and also to ensure that myoglobin does not produce renal damage, particularly in the face of hypovolemia. In addition, 0.2 mg/kg dexamethasone (Treatment Nos. 29 and 30) intravenously is useful in aiding relief of clinical signs. The use of nonsteroidal anti-inflammatory drugs such as phenylbutazone (Treatment No. 88) at dose rates of 2.2 mg/kg twice daily for 3 to 5 days may be indicated. If horses are in severe pain, the use of drugs such as detomidine (Treatment No. 28) at dose rates of 10 to 20 μg/kg or xylazine (Treatment No. 108) at a dose rate of 0.2

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mg/kg may be indicated. Because these drugs have a profound effect on blood pressure, they should not be used before correction of hypovolemia. After recovery, the horse should be rested for at least 6 to 8 weeks and then slowly brought back into training.

Mild Cases. Walking affected horses is sometimes useful, and most will recover without any further treatment. Intravenous thiamine and intramuscular vitamin E and selenium have been used widely in practice, and there is some empirical evidence that they may assist recovery. The use of nonsteroidal anti-inflammatory drugs such as phenylbutazone at dose rates of 2.2 mg/kg twice daily for 3 to 5 days may be indicated. Horses are usually rested for 3 to 4 days and then introduced to a gradually increasing exercise program. Lowering of the training intensity together with reduction of grain in the diet is often useful. Acepromazine (8-12 mg per 450-kg horse) can help alleviate the muscle spasm and promote peripheral vasodilatation in mild cases. In some cases, acepromazine at dose rates of 5 to 8 mg per 450-kg horse, administered before training, is useful in preventing further signs of the disease.

Chronic Intermittent Cases. The administration of the intracellular calcium-blocking agent dantrolene sodium (Dantrium) and the sodium and calcium channel-blocking agent phenytoin (Treatment No. 89) has been reported to aid in the prevention of recurrence of rhabdomyolysis. Dantrolene is expensive to use, difficult to achieve adequate circulating blood concentrations, and of questionable efficacy.

KEY POINT

Phenytoin is a useful adjunct to management changes in horses with the recurrent form of the disease. Ideally, dosage schedules should be based on determinations of plasma concentrations, with therapeutic levels considered to be in the range 5 to 10 μg/mL.

This is usually achieved by oral administration of 10 to 12 mg/kg phenytoin twice daily for 3 days, followed by 10 to 12 mg/kg once daily for 3 days. After this, the dose is reduced to 5 to 6 mg/kg once daily. The drug should be withdrawn at least 7 days before racing. The response to this drug is not consistent, and adverse effects include drowsiness, ataxia, and, rarely, seizures. During the first 3 days on the high-dose, twice-daily regimen, it is best to avoid serious training, because horses usually become a little depressed.

If urinary electrolyte clearances indicate marginal dietary sodium and/or potassium, electrolyte supplementation may be indicated. This usually involves adding 20 to 60 g sodium and/or potassium chloride to the feed.

Septic Arthritis

Septic arthritis may occur either in foals, where it is usually the result of septicemia ("joint-ill"), or in adult horses, where it results from penetrating injuries and/or occurs after intra-articular injection of therapeutic agents. Because of the destructive effects on articular cartilage of bacteria, high leukocyte concentrations, and resultant enzyme liberation, early diagnosis and treatment are essential if irreversible damage to the joint is to be avoided. The most common mistake made by equine practitioners in treating suspected septic arthritis is that of commencing antibiotic therapy before collecting synovial fluid samples for culture and sensitivity testing. The use of antibiotics, even if ineffective, will make the subsequent culture of bacteria difficult.

HISTORY AND CLINICAL FINDINGS

- The earliest clinical sign is a reluctance to bear weight associated with marked heat, swelling, and pain in the affected joint(s). In adults there tends to be extensive periarticular edema and joint effusion, whereas in foals, swelling tends to be primarily of the joint capsule.
- There may be signs of a septicemia or bacteremia, including elevated heart rate and temperature, particularly in foals with "joint-ill," although in many cases the lameness develops when the initial disease process resolves. In the early stages of septic arthritis, horses appear depressed and may be inappetent. Temperature can fluctuate greatly during the course of the day but is usually in the range of 39 to 40°C (102.5-104°F).

DIAGNOSTIC TESTS

Synovial Fluid Analysis

If septic arthritis is suspected, a synovial fluid sample should be collected to confirm the diagnosis, and this should be done before any antimicrobial therapy is commenced. It is imperative that the sample be collected with a sterile technique.

Synovial fluid samples should be collected into EDTA for total and differential white cell counts and total protein measurement and into a plain sterile container for bacteriology. White cell counts in excess of 5000 X 10^6/L with greater than 80% neutrophils and total protein values

greater than 30 g/L (3 g/dL) indicate septic arthritis. In the acute stages of septic arthritis, before antibiotic therapy, white cell counts in synovial fluid are often greater than 30,000 X 10^6/L, and the percentage of neutrophils is in excess of 95%.

Synovial fluid samples for culture and sensitivity can be injected directly into blood culture bottles or centrifuged, discarding the supernatant, and resuspending the deposit in enrichment media. (For further details, see Chapter 17, Clinical Bacteriology). If antibiotics have been used before collection of the synovial fluid samples, antibiotic removal devices can be used to assist in recovery of bacteria.

Synovial Membrane Biopsy

In some cases, bacteria cannot be recovered from synovial fluid, particularly when antibiotics have been used before bacteriologic sampling. If arthroscopic surgery is performed as a therapeutic procedure, a synovial membrane biopsy can be used to obtain tissue for bacteriologic culture and sensitivity testing. The sample should be submitted in a sterile container to the laboratory.

DIFFERENTIAL DIAGNOSIS

- Fractures
- Osteomyelitis (particularly of the epiphyses)
- Infection of tendon sheaths
- Foot abscess
- Infection of the navicular bursa

TREATMENT

- Bactericidal antibiotics should be commenced immediately. Penicillin therapy is rarely adequate on its own because gram-negative bacteria and penicillin resistant *Staphylococcus* sp. are often involved. Therefore, penicillin should be combined with gentamicin (Treatment No. 56) or amikacin (Treatment No. 4). The treatment subsequently can be modified when the bacteriologic results become known. If early treatment is undertaken, appropriate systemic antibiotic administration will result in good recovery of function.
- Parenteral therapy should be combined with local antibiotics. Gentamicin or amikacin can be used intra-articularly or by regional perfusion for joints of the distal limb through either the intraosseous or intravenous route distal to a tourniquet applied proximal to the affected joint. Intra-articular therapy can be repeated daily.
- The joint should be irrigated with 4 to 5 L of a balanced electrolyte solution. Ideally, this is done under arthroscopic visualization to allow assessment of the articular cartilage, debridement of any infected subchondral bone, more thorough removal of fibrin clots, and resection of proliferating synovium. If improvement in clinical signs is not evident after 48 hours, this can be repeated and the portals widened on completion to allow better debridement and fibrin removal.
- The inclusion of additives to the irrigation fluid is controversial. DMSO (10% solution) has been advocated due to its anti-inflammatory and antibacterial properties, but there is no definitive evidence that it is beneficial. Povidone-iodine has also been suggested, and at a concentration of 0.01%, it does not affect articular cartilage but has not been shown to improve the elimination of bacteria. The injection of antibiotics after lavage is probably more effective. Systemic nonsteroidal anti-inflammatory therapy is also indicated to reduce swelling and improve weight bearing.
- Antibiotic therapy should be continued for 1 week after sepsis has resolved. This is based on absence of lameness and synovial fluid cell counts of less than 5000 X 10^6/L.
- The prognosis for these cases depends on the duration of infection and the degree of damage to the articular cartilage. Degenerative joint disease is a common sequel to infection as a result of the damage to articular cartilage and synovial membrane.

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CHAPTER 5

Respiratory System

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Diseases of the respiratory system are common in horses, particularly in North America and Europe where infectious respiratory conditions often result in significant wastage and poor performance in athletes. The combination of climate, the need for housing during the colder months resulting in close contact between animals, stressful activities such as racing and transport, and the presence of a variety of respiratory viruses results in the high incidence of respiratory disease. Infectious respiratory disease often produces the greatest morbidity in younger horses and can be particularly difficult to manage on stud farms and in racehorse populations.

Most infectious respiratory diseases are considered to be initiated by viral infections, in which the affected horse shows signs of depression, inappetence, and fever. Commonly, the offending agent is cleared by the host. However, where the horse's defenses are compromised, secondary bacterial infections may result in more severe bronchitis, pneumonia, or possibly pleuropneumonia.

The challenge in respiratory disease is in making a specific diagnosis. Signs of respiratory tract involvement are usually self-evident and include respiratory noise during exercise, nasal discharge, cough, dyspnea, and fever. It is only after careful examination and utilization of relevant diagnostic aids that a correct diagnosis can be made and an appropriate therapy instituted.

SIGNALMENT AND HISTORY

Signalment is important because younger horses often suffer from infectious diseases (e.g., *Rhodococcus equi* in foals and rhinopneumonitis in weanlings and yearlings), whereas older animals tend to be afflicted more commonly with chronic disorders (e.g., chronic allergic respiratory disease). A thorough **history** is essential when one is presented with a horse suspected of having a respiratory disorder. The exact complaint, duration of the disorder, a history of exposure to stress or other horses with similar signs, recent changes in management, vaccination schedules, and responses to treatment (if any) should be determined. Some of the specific questions related to various respiratory disorders are outlined in Chapter 2.

EXAMINATION PROCEDURE

Respiratory disease frequently results in generalized systemic signs, making it important to assess carefully the animal's overall health status and to perform a specific examination of the respiratory tract. Initial evaluation of a horse with respiratory disease should include assessments of general body condition, quality of the hair coat, rectal temperature, heart rate, pulse quality, and hydration status (see Chapter 1). After these appraisals, a detailed examination of the respiratory tract should be performed. This is best done initially at rest and in a location free from distracting outside noise.

Initial Inspection

The examiner should begin by gaining a general impression of the horse's respiratory rate, effort, pattern, and evidence of inspiratory or expiratory noise. Abnormalities such as dyspnea, flaring of the nostrils, guarded inspirations, or a marked diphasic expiratory effort with an increased contribution by the abdominal musculature should be noted. The presence of a nasal discharge should also be investigated. Serous nasal discharge is more common early in the course of viral respira-

tory infections, whereas mucopurulent exudate is more likely with a bacterial disease. It also should be noted whether the discharge is unilateral or bilateral, malodorous, or more profuse when the head is lowered or the horse is eating (as often occurs with guttural pouch empyema). It should be ascertained whether the horse has a cough, and if so, its frequency and character should be determined.

Detailed Examination

Examination begins with inspection of the head and neck for any obvious asymmetry. Care should be taken to examine the nostrils for symmetry, equality of airflow and presence of abnormal respiratory sounds, odor (which may indicate turbinate necrosis, guttural pouch disease, or pleuropneumonia), or palpable abnormalities in the nostrils. Examination of the mucous membranes should follow to check for color, cyanosis, or hemorrhages. The area between the mandibles and the parotid region is then examined to determine if there is any enlargement of lymph nodes. Percussion of the maxillary and frontal sinuses should be performed to determine if there is any fluid present or associated pain. The presence of fluid will usually result in a dull sound, and the horse may also show signs of pain on percussion. The maxillary sinuses lie dorsal to the facial crest, and the frontal sinuses lie on either side of the dorsal midline, with their centers approximately at the level of the eyes. Determination of alterations in the resonance of maxillary sinuses is more easily done if the horse's mouth is open, by placing a finger in the interdental space, while percussing. The larynx is then palpated to determine if any asymmetry of the dorsal cricoarytenoid muscles can be felt. This is done by standing on the left side of the horse facing toward the head and placing the index fingers under the tendon of the sternocephalicus muscle on either side of the neck and palpating the anterior dorsal part of the larynx. If there is atrophy of the dorsal cricoarytenoid muscle (as occurs in idiopathic left laryngeal hemiplegia), the muscular process of the arytenoid cartilage will be more prominent on the affected side than the unaffected side. Any scarring or thickening of skin that could suggest a previous laryngoplasty or laryngotomy should be noted. If there is any indication of excessive prominence of the left muscular process of the arytenoid cartilage, indicating left laryngeal hemiplegia, a laryngeal adductor test ("slap test") and upper respiratory tract endoscopy may be useful.

KEY POINT

The "slap test" can be done without an endoscope while palpating the muscular processes on the left and right sides.

This is usually done by having an observer slap alternately the left and right chest walls gently with the open hand. This should result in the muscular process on the opposite side adducting, felt as a "flicking" or movement of the process. Similarly, slapping of the right thorax should result in the muscular process of the left arytenoid cartilage flicking. In horses with idiopathic left laryngeal hemiplegia, the flicking of the left muscular process does not occur or is reduced in response to the slap test on the right side of the chest. Squeezing firmly at the junction of the cricoid cartilage and trachea will induce a cough in many horses, and in horses with pharyngitis, laryngitis, or tracheitis, pressure in this region may evoke a paroxysm of coughing. Palpation of the cervical trachea should then be performed to assess if there is stenosis or other deformity.

Auscultation

Careful auscultation of the chest on both left and right sides and over the cervical trachea is important if pulmonary disease is suspected. Auscultation should be performed in a quiet environment to optimize the chances of detecting abnormalities. The normal margins of the thoracic cavity are level with the tuber coxae at the 18th rib, midthorax at the 15th rib, and shoulder at the 11th rib and then curve down to the level of the elbow.

KEY POINT

Because of the quiet respiration of most horses (particularly fat animals), it may be helpful to accentuate the respiratory sounds by placing a large plastic bag over the horse's nose (Fig. 5-1).

A rebreathing bag will stimulate increased rate and depth of respiration by lowering the inspired oxygen concentration and increasing the carbon dioxide concentration. The result is large respiratory excursions, and air movement through both large and small airways is more easily heard. Care should be taken when using this technique in horses with painful pleural conditions because discomfort may be increased, associated with deep respiratory efforts.

KEY POINT

Application of a rebreathing bag is not usually required in foals, and restraint alone

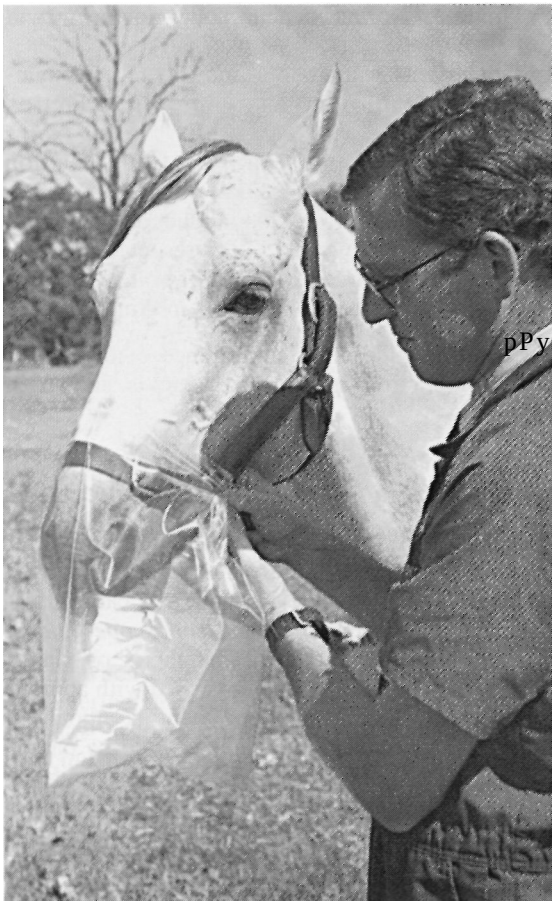


Figure 5-1. Auscultation of the chest using a rebreathing technique. A plastic bag is placed over the horse's nose, and the resulting increase in arterial carbon dioxide tension and decrease in arterial oxygen tension results in an increase in tidal volume and respiratory rate.

generally produces some degree of hyperventilation.

Another complication associated with rebreathing bags is the induction of paroxysmal coughing, which occasionally occurs in horses with highly sensitive airways (e.g., chronic allergic respiratory disease). Such a reaction will preclude effective auscultation of the respiratory tract.

Under normal conditions, inspiratory sounds are louder than expiratory sounds, with all sounds being slightly louder on the right side of the thorax. Sounds over the carina are more audible than those in other parts of the respiratory tract. Abnormalities include increases in the intensity of respiratory sounds (particularly if expiration is louder than inspiration), areas of dullness, radiation of heart sounds over a greater area than normal, crackles (discontinuous sounds), wheezes (continuous sounds), and pleural friction rubs (loudest at the end of inspiration). A common normal finding when auscultating the chest is the presence of intestinal sounds within the thoracic cavity.

Percussion

Percussion of the chest can be useful for detection of fluid or pleural pain. Percussion is performed using a plexor and pleximeter or a rubber hammer as a plexor and a spoon as a pleximeter (Fig. 5-2) or by using the thumb and middle finger to flick the chest. By working along the chest in parallel lines from dorsal to ventral and anterior to posterior, the whole chest can be covered. Dull areas indicate consolidation near the lung surface or the presence of fluid.

DIAGNOSTIC AIDS

Endoscopy

Direct visual examination of the respiratory tract with flexible endoscopes is probably the most widely used diagnostic aid in equine practice. Most endoscopes in use today have an external diameter of 8 to 10 mm and range in length from 60 cm to 3 m. Examination is likely to be most safely performed with the horse restrained in stocks (if possible). Sedation usually is not necessary and may influence the findings of some examinations, because function of the arytenoid cartilages may be modified after administration of sedatives. After application of a twitch, the endoscope is passed up the ventral nasal meatus. Depending on the indication, examination of the nasal passages, nasomaxillary ostium of the paranasal sinuses, ethmoid region, pharynx, lar-

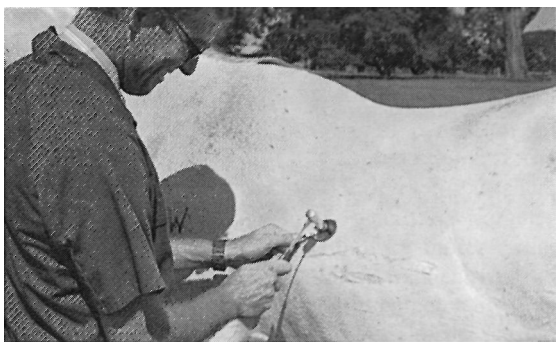


Figure 5-2. Percussion of the chest using a metal spoon as a plexor and a patellar hammer as a pleximeter.

ynx, guttural pouch openings (and the internal linings of the pouches), trachea, and some other parts of the lower airways can be carried out. A line drawing showing the normal larynx and pharynx as viewed from the nasopharynx is presented in Figure 5-3. Longer endoscopes can be passed distally as far as some of the mainstream bronchi. Endoscopy is a particularly valuable tool for examination of the upper respiratory tract and enables diagnosis of laryngeal hemiplegia, aryepiglottic entrapment, pharyngeal cysts, and lymphoid hyperplasia. The technique is also often integral to the detection of nasal tumors, polyps, and mycoses; ethmoid hematomas; guttural pouch empyema and mycosis; pus or blood draining from the nasomaxillary opening; exercise-induced pulmonary hemorrhage; and the accumulation of large volumes of abnormal fluid or pus within the trachea.

Laryngeal function tests that are useful during endoscopy include nostril occlusion to stimulate laryngeal abductor and adductor movement and flushing water through the endoscope to stimulate swallowing. The first breath taken by the horse after swallowing results in full abduction of the larynx. We have found a laryngeal scoring system useful to record the findings on the clinical record at the time of endoscopy. We use the system of Lane (1993), which describes five gradings of laryngeal appearance:

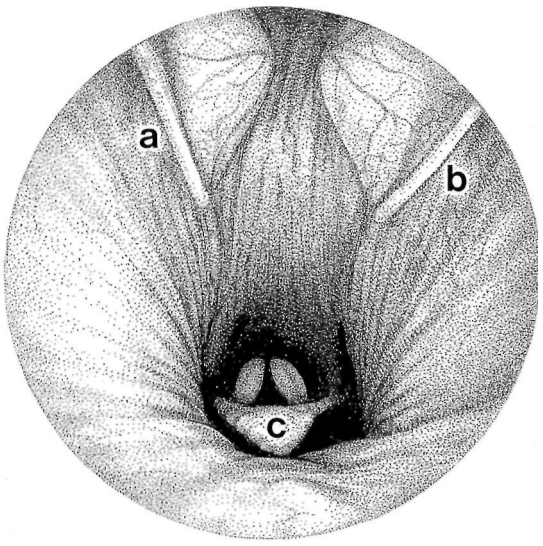


Figure 5-3. Line drawing of the larynx and pharynx as seen via an endoscope positioned in the nasopharynx. The soft palate lies ventrally under the epiglottis (c), and dorsally the right (a) and left (b) guttural pouch openings are visible.

Grade 1: Normal synchronous movement and full abduction of arytenoid cartilages.

Grade 2: Asynchronous movement, but symmetric arytenoids at rest and full abduction achieved.

Grade 3: Asymmetry at rest, full abduction possible.

Grade 4: Inability to fully abduct left arytenoid cartilage, some movement still present.

Grade 5: No movement of left arytenoid cartilage, which is resting on or near midline.

The normal endoscopic appearance of the larynx at rest and after nostril occlusion is shown in Figure 5-4.

Catheterization of the Guttural Pouches

This procedure is useful for collecting samples for bacterial and fungal culture and for irrigating the pouches. There are two methods for catheterization of the pouches, *blind* and *endoscope-guided.* For the blind technique, a Chamber's or plastic mare uterine infusion catheter is used with approximately 3.75 cm at the end bent at about 20 degrees. The distance the catheter is to be passed to gain access to the guttural pouch can be determined by measuring the distance between the lateral canthus of the eye and the nostril. A mark is placed on the catheter as a reference point when it is inserted. The catheter is passed via the ventral meatus, with the tip pointed down. When inserted to a depth of about 2 to 5 cm from the mark, the catheter is rotated laterally through about 150 degrees, and the end of the catheter in the nostril is moved into a dorsal and lateral position over the alar fold. The catheter is advanced, and contact with the pharyngeal wall can be sensed. When the horse swallows, the guttural pouch openings will dilate, and the catheter can be passed into the pouch. Once the catheter passes through the opening, it can continue to be advanced with less resistance than if it were pushing into the pharyngeal wall. Passage of the catheter is achieved more easily if an endoscope is passed up the contralateral nostril and the catheter is observed as it is passed into the pouch opening. Samples for microbiologic analysis can then be collected by infusion of a sterile lavage solution (e.g., normal saline) with subsequent aspiration. Obviously, this technique also provides a mechanism by which solutions for therapy can be infused into the pouch.

A number of methods have been described for the placement and maintenance of indwelling catheters in the guttural pouch. These include Foley catheters (27Fr) with the balloon inflated with

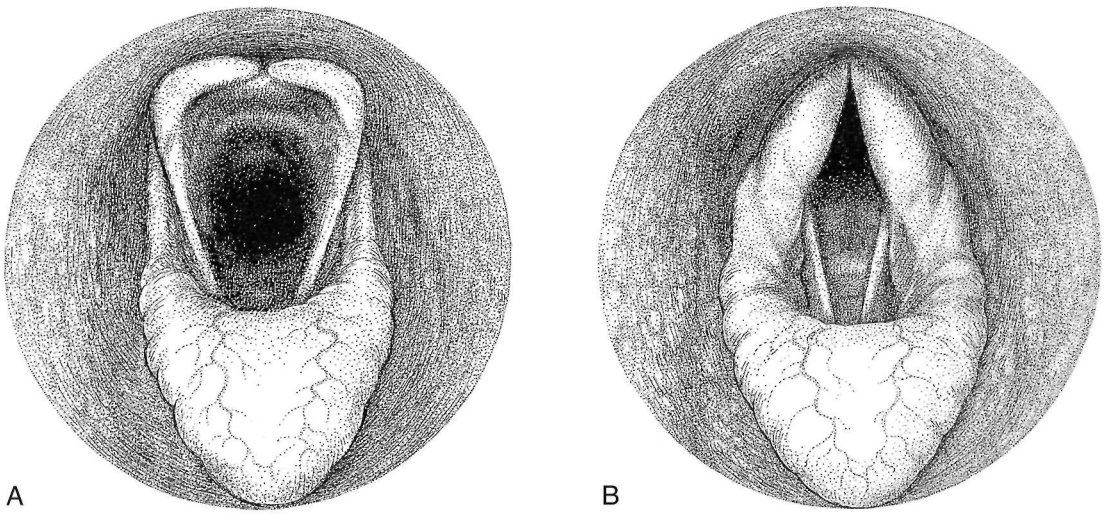


Figure 5-4. Line drawings of the endoscopic appearance of the larynx in the horse after respiratory stimulation showing arytenoid abduction *(A)* and during quiet breathing *(B).*

saline when in the pouch. Alternatives include an intrauterine catheter with a "ram's horn" tip or a catheter with a curled grapple at the tip constructed from polyethylene (PE 240) tubing. The grapple is made by heating the tubing in hot water and then wrapping it around the barrel of a 25- to 30-mL syringe. Although these catheters provide an excellent means for repeated pouch infusions, they may induce considerable local irritation to the mucosa of the guttural pouch.

Endoscopic examination of the guttural pouches is an important part of the respiratory examination procedure and can be achieved relatively easily with a little practice. It is best performed using a narrow endoscope (e.g., 10-mm diameter). A guide wire or stiffener is passed into the biopsy portal of the endoscope. The endoscope is then directed into the ipsilateral ventral meatus. The endoscope is positioned such that the biopsy portal is lying against the lateral pharyngeal wall close to the dorsal-most point of the pouch opening. The guide wire is then directed into the pouch opening, and the endoscope is rotated medially through 180 degrees and advanced into the medial compartment of the guttural pouch.

Transtracheal Aspiration

A number of techniques have been described for lavage and aspiration of the tracheobronchial tree. Transtracheal aspiration allows aseptic collection of samples from the lower respiratory tract for cytologic and microbiologic studies.

KEY POINT

For collection of samples under the most aseptic conditions for microbiology, a transtracheal wash and aspirate is the optimal method.

Contamination of sampling catheters and aspirates with bacteria from the nasal passages and pharynx is avoided, and the findings are more likely to reflect the real bacteriologic situation in the lower respiratory tract. To perform the technique, an area over the middle to lower trachea is clipped and surgically prepared with appropriate skin disinfection. Horses may require the application of a twitch or administration of a small dose of sedative before the procedure. A small volume (1-2 mL) of local anesthetic is injected under the skin over the trachea (Fig. 5-5) and a small stab incision is made (Fig. 5-6). The operator applies surgical gloves, and the trachea is palpated and stabilized with one hand. A 12- or 14-gauge needle or catheter is inserted in a downward direction through the skin incision into the lumen of the trachea (Fig. 5-7), taking care to penetrate between the tracheal rings and not to damage the tracheal rings on the opposite side of the trachea. If resistance to passage of the needle or catheter into the trachea is detected, this may indicate that the needle or catheter is against a tracheal ring. A 5Fr or 6Fr sterile dog urinary catheter is then passed down the trachea to the level of the carina. Because the ends of these catheters are sealed, it may be useful to cut off the end to allow passage of tenacious secretions. An alternate method uses



Figure 5-5. Transtracheal aspiration. The skin over the ventral midline of the midcervical region is clipped, and after appropriate skin disinfection, a small amount (1-2 mL) of local anesthetic is injected to raise a bleb.

a 60-cm drum catheter-needle combination (Deseret Co., Sandy, UT). The catheter should be inserted 50 to 60 cm at least to the level of the thoracic inlet (where the trachea lies in the horizontal plane and secretions may accumulate). Sterile saline (20-50 mL) is injected into the trachea (Fig. 5-8) and aspiration is performed (Fig. 5-9). If there is little or no yield of fluid on aspiration, the catheter can be repositioned slightly, and aspiration can be repeated. If this is still unsuccessful, the flushing/aspiration procedure can be repeated. In addition, the horse's head can be lowered to further improve the chance of a successful aspiration. Some horses will cough during infusion of saline, which may increase the yield of mucopurulent material. If there is severe coughing, the catheter may be dislodged proximally to lie around the laryngeal opening.

After collection of the sample, the urinary catheter is removed first, followed by the introducer.



Figure 5-6. Transtracheal aspirate. After clipping and appropriate skin disinfection, a stab incision is made through the skin and subcutaneous tissues with a no. 11 scalpel blade.

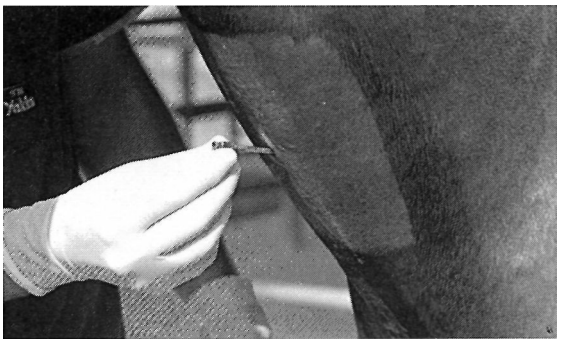


Figure 5-7. Transtracheal aspirate. A large-bore (12-gauge) needle is inserted between the tracheal rings into the lumen of the trachea.

This sequence may not be possible if a needle is used as an introducer, because retrograde movement of the catheter may result in severance of the catheter tip in the trachea. However, a piece of tubing that fits down the lumen of the trochar through which the urinary catheter can be inserted before placement may help to prevent catheter damage and limit subcutaneous contamination.

Complications of the procedure include the following:

- Damage to the tracheal rings, which can be reduced if care is taken when the needle is inserted.
- Breakage of the catheter within the trachea. This is rare but if it occurs, most horses cough up the catheter within 30 minutes with no untoward side effects.
- Cellulitis at the site of tracheal puncture. This is probably the most common adverse effect and may be reduced in severity if the catheter is

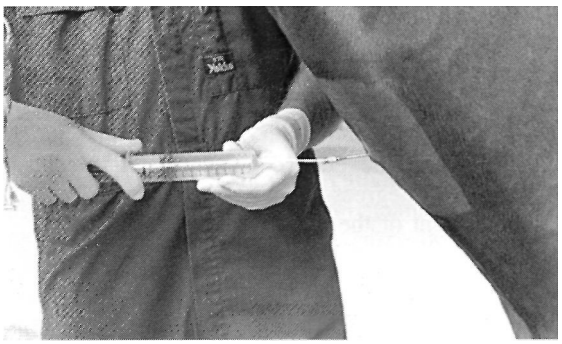


Figure 5-8. Transtracheal aspirate. A sterile dog urinary catheter (8Fr) is inserted through the needle into the trachea so that it comes to lie around the area of the carina, and sterile phosphate-buffered saline (50-200 mL) is injected through the urinary catheter.



Figure 5-9. Transtracheal aspirate. Aspiration of mucopurulent material from the lower airway.

removed prior to extirpation of the introducing needle and a bandage is applied for 24 hours after the procedure. Alternatively, application of Superglue to the wound edges to seal them together and gluing of an adhesive strip over the wound gives good results. The adhesive strip can be removed in 24 to 48 hours.

Administration of systemic antibiotics may be considered if malodorous or purulent material is collected or if the initial Gram stains indicate bacterial infection. If swelling does occur, local therapy with hot saline packs and topical dimethyl sulfoxide (DMSO) may be useful.

KEY POINT

Samples may be collected for cytologic study via a catheter inserted into the lower airway through the biopsy channel of a flexible endoscope.

However, because there is contamination by bacteria resident in the upper respiratory tract, such samples are unreliable for microbiologic study. The operator can visualize the areas of accumulated mucoid or purulent material, direct the catheter appropriately, and harvest samples using a similar technique to that described for the transtracheal method. Overall, contamination of samples is reduced if the endoscope is disinfected before the procedure.

Recently, "guarded" catheters for insertion through the nasopharynx for collection of tracheobronchial aspirates have been described (Darien microbiology aspiration catheter, Mill-Rose Labs., Inc., Mentor, OH). These catheters substantially decrease the chance of contamination of samples, with recent studies indicating that the sample quality can be as good as that obtained using traditional techniques. The advantage of the technique is that the procedure using the Darien catheter is far less invasive than transtracheal aspiration.

After collection, the aspirate is placed in a sterile container (or kept in the sterile syringe) and subjected to cytologic, bacteriologic, and possibly mycologic examination (see Chapter 16).

Bronchoalveolar Lavage

This procedure has been used much more widely in recent years and is designed to collect fluid and cells from the distal airways and alveoli. It is a simple technique that often provides useful information about the integrity and constituents of the lower respiratory tract. Bronchoalveolar lavage (BAL) can be performed "blind" or with a flexible fiberoptic endoscope. When the endoscopic technique is used, the horse may be sedated (xylazine at a dose rate of 0.2-0.4 mg/kg IV is suitable) and the tip of the endoscope passed into the lower respiratory tract and wedged in the smallest possible airway. With the blind technique, an equine BAL tube (Bivona, Inc., Gary, IN) is passed into the lower airway and wedged into position (Fig. 5-10). Passage of the tube into the trachea is facilitated if an assistant extends the horse's head. Obviously, the operator has little knowledge of which respiratory unit the lavage tube is wedged in. However, radiologic studies have shown that the tube usually lodges in the dorsocaudal lung. Between 50 and 300 mL of saline is infused in 50-mL aliquots, and it is aspirated immediately after infusion.

Subsequent to collection, samples are observed for color and subjected to cytologic (total and differential nucleated cell counts) examination.

Complications during BAL include the following:

- Kinking of the tube, once the cuff is inflated and fixed in the distal airway, results if the horse pulls away and the tube is replaced without deflating the cuff. The tube often kinks within the pharynx or in some cases can be coughed up, allowing it to enter the mouth and subsequently be chewed.
- Contamination of the sample with pharyngeal microbial flora and debris.
- Paroxysmal coughing. This may upset the horse owner or trainer. In most cases, coughing usually is transient and generally occurs when the tube is being passed and reaches the carina.

KEY POINT

BAL will provide information only about the portion of the lung sampled. This may not be indicative of the overall condition of the lung.

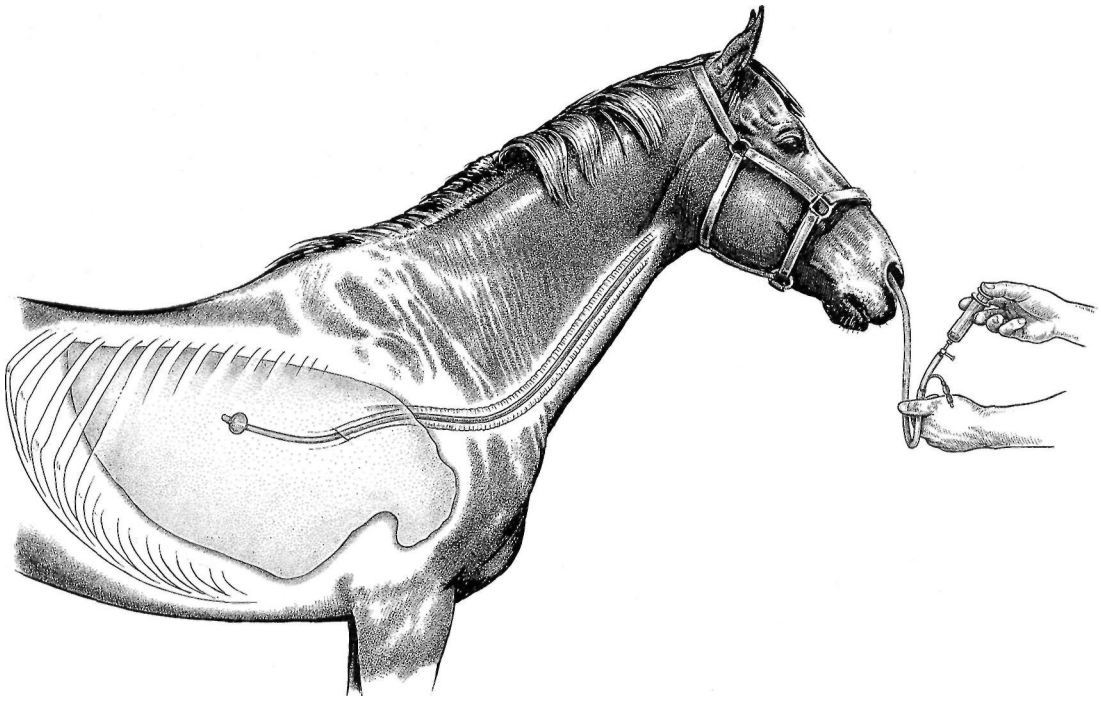


Figure 5-10. Technique of bronchoalveolar lavage. After the catheter is lodged in a distal bronchus, the balloon on the catheter is inflated with 5 mL of air. Sterile saline (50-200 mL) is then flushed into the lung and aspirated for cytologic analysis.

Ultrasound Examination

Over the past 10 to 15 years, the advent of portable ultrasound equipment has made this form of examination of a horse's thorax feasible. As a result, ultrasonography is now widely used by equine practitioners, particularly in cases where lower respiratory tract pathology is suspected. It is most suitable for detection of accumulations of fluid within the pleural space, consolidation of the lung parenchyma, and abscessation. In addition, if fluid has accumulated within the thorax, some indication regarding the quality of that fluid can be obtained. Both linear and sector scanners may be used to examine the equine thorax. Sector scanners provide the best quality images but are more expensive than linear scanners (commonly used for pregnancy diagnosis). Both types of scanners can provide quite acceptable images of the equine lung and pleural space. Sector transducers with frequencies of 2.5 and 3.5 MHz provide the penetration required to view intrathoracic structures. However, as a consequence, image resolution is not as good. Linear array transducers with adjustable focusing properties allow better quality images to be obtained. It is not necessary to clip the hair over the region to be imaged. However, because intact hair traps air, which dramatically interferes with the quality of the image, it needs to be "wetted" down with alcohol, mineral oil, or other suitable acoustic coupling gel. In long-coated horses, clipping of the hair may be necessary.

The boundaries of the lung are described earlier in this chapter. Such anatomic landmarks are important when imaging the thorax. A systematic approach should be adopted such that the entire lung field is examined. Movement of the scan head from dorsal to ventral along sequential intercostal spaces will allow imaging of most of the lung field. Particular care should be taken when examining the cranioventral thorax because this is the most common site for fluid accumulation. Elevation and cranial retraction of the forelimb may allow better imaging of cranial thoracic structures. Fluid containing small amounts of particulate matter may create little acoustic reflection (anechoic), whereas accumulations of inflammatory debris (cells and fibrin) are echogenic. Anaerobic infections may be indicated by the appearance of gas bubbles within pleural fluid accumulations. Small areas of pleural irregularities and superficial lung consolidation may present as reverberation artifacts or comet tails originating close to the visceral-pleural interface. Larger areas of pulmonary consolidation usually are identified

within the ventral lung fields and provide a more echogenic image resembling that of liver, hence the term "pulmonary hepatization." Thoracic ultrasonography provides a noninvasive and tangible means of monitoring lower airway disease progression and response to treatment.

KEY POINT

Thoracic ultrasonography also has been used recently for the detection of pulmonary abscessation associated with R. equi *infections and has proved to be a sensitive and useful technique for early diagnosis of the problem.*

Thoracocentesis

If a dull area or fluid line is suspected by auscultation or percussion or fluid is imaged with ultrasound examination or radiography, thoracocentesis is indicated to reveal if fluid is present and the cytologic and microbiologic characteristics of that fluid. In general, thoracocentesis should be performed in the ventral third of the thorax, with care being exercised to avoid the heart. Although not imperative, ultrasound-guided thoracocentesis is the optimal method. For initial sampling, if only a small amount of fluid is suspected, a 3.75-cm needle or 6- to 7.5-cm teat cannula or catheter can be used.

KEY POINT

Aseptic technique is important, and therefore, an area of skin over the sixth, seventh, and eighth intercostal spaces at least 5 x 5 cm^2 above the level of the olecranon should be clipped, shaved, and aseptically prepared.

Local anesthetic is infused subcutaneously and into the intercostal muscles and sufficiently deep to include the parietal pleura. A stab incision is made in the skin with a small blade, and the cannula is inserted cranial to the rib border to ensure that the intercostal vessels and nerves, which course down the caudal aspects of the ribs, are not damaged. The cannula is advanced into the pleural space, and fluid is aspirated (Fig. 5-11). If large amounts of fluid are present or if the fluid has a high concentration of fibrin material, larger bore catheters can be used. Blunt-tipped thoracic drainage tubes designed for human use are ideal and are available in a variety of sizes. These may be sutured in place and plugged if repeated drainage is required. Alternatively, "condom" valves may be attached to allow continued fluid drainage and to reduce the chance of a pneumothorax. A condom valve involves taping a condom over the end of the thoracic drain. The tip of the condom is cut to create a one-way valve.

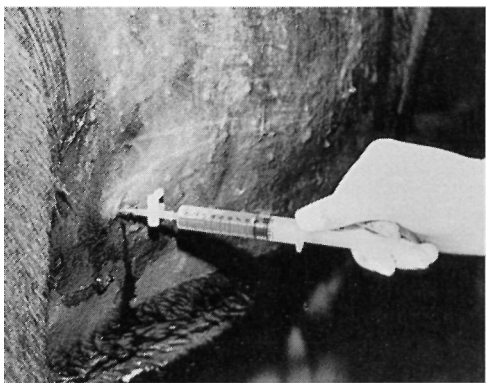


Figure 5-11. Thoracocentesis showing position of cannula in the eighth intercostal space.

KEY POINT

Thoracocentesis should be performed on both sides of the thorax because different bacteriologic results can be found.

The quantity and quality of fluid obtained during this procedure may provide valuable information for the clinician. In the normal horse, little or no fluid is obtained from the thorax. In conditions where pathology exists, more than 25 L of fluid may be drained from each hemithorax. In addition to fluid volume reflecting pathology, the color of the fluid, degree of opacity, presence of fibrinous material, and odor are all useful indicators of the severity of disease. With infectious causes of pleural effusion, the fluid will often become more opaque and malodorous and contain fibrin clots. The presence of a fetid odor may indicate the presence of anaerobic bacteria.

Cytologic examination of normal pleural fluid reveals a sterile fluid with a total protein concentration below 30 to 35 g/L (3-3.5 g/dL) and a nucleated cell count less than the peripheral white cell count and usually less than 8000 to 10,000 nucleated cells X 10^6/L (8000-10,000/μL). Of these cells, less than 60 to 70% should be neutrophils, with the balance macrophages and lymphocytes. With pleuropneumonia, the volume of fluid increases, as does the cell count (predominantly neutrophils) and total protein concentration. The pH and glucose content of pleural fluid also may be good indicators of disease. Under normal conditions, pleural fluid pH is similar to that of peripheral blood (7.4), and the glucose concentration is greater than 2 mmol/L (40 mg/dL). In cases of

pleuropneumonia, pH and glucose concentrations often fall below these values.

Thoracic fluid should be placed into appropriate preservatives for cytologic and microbiologic examinations. For cytologic examination and protein determination, samples should be placed in EDTA tubes used for preservation of peripheral blood samples. Samples for determination of glucose concentration should be submitted in fluoride oxalate preservative. Methods for management of samples for bacteriologic studies are described in Chapter 16. Particular care must be taken with sample collection when anaerobic infections are suspected if one is to attempt bacterial culture.

Radiography

Radiographic examination of the respiratory tract can be helpful in the diagnosis of sinusitis, nasal masses (e.g., neoplasia and ethmoid hematomas), tooth-root problems, guttural pouch disorders (e.g., empyema and chondroids), and tracheal stenosis. If the clinician has access to equipment of suitable size and power, thoracic disorders (e.g., pneumonia, abscessation, pleural effusion, neoplasia) can be diagnosed. Complete radiographic evaluation of the adult equine thorax may require multiple overlapping views and in most instances is limited to standing left and right projections. Because of this, lesions can be assessed in only two dimensions. In the standing horse, the four standard radiographic views are dorsocranial, dorsocaudal, ventrocaudal, and ventrocranial projections. Radiography of the thorax in foals usually can include lateral and dorsoventral views and can be particularly helpful if pneumonia (e.g., *R. equi)* is suspected.

Arterial Blood Gas Analysis

Assessments of pulmonary function can be aided by the measurement of the partial pressures of oxygen and carbon dioxide within the arterial blood. The partial pressure of carbon dioxide in the arterial blood ($Paco_2$) reflects alveolar ventilation and is therefore an indicator of hypoventilation (e.g., chronic obstructive pulmonary disease). In contrast, the Pao_2 is related to the inspired oxygen tension, alveolar ventilation, and the effectiveness of gas exchange across the alveolar-capillary membrane. Thus, in an animal breathing ambient air, with a normal $Paco_2$ (reflective of normal alveolar ventilation), hypoxemia indicates impaired efficiency of gas exchange within the lung. An example is provided by diseases causing severe lung consolidation (e.g., pneumonia), in which there is mismatching between ventilation and perfusion to large areas of the lung.

KEY POINT

Although many veterinary practices do not own blood gas machines, most hospitals have this apparatus and are willing to process samples for veterinarians for an arranged fee. Samples can be stored on ice for several hours after collection, making this procedure one worthy of consideration, even for the busiest of practitioners.

The two most frequently used sites for collection of arterial blood samples from the conscious horse are the common carotid artery and the transverse facial artery. Collection of a sample from the *carotid artery* is undertaken after aseptic preparation of the skin dorsal to the jugular groove on the right side of the neck. The carotid artery can be palpated as a cord-like structure deep to the jugular vein on the dorsal side of the groove (under most circumstances no pulse will be detected) running alongside the trachea. A 3.75-cm, 18- to 19-gauge needle is directed into the vessel. It may be necessary to perform several manipulations with the needle after penetration of the skin to obtain access to the lumen of the artery. Once the needle is in place, blood will be expelled from the vessel under pulsatile pressure (Fig. 5-12). The needle is stabilized with the fingers of one hand, and a 2- or 5-mL syringe is connected to the hub. This syringe should have been heparinized, with a small volume of 1000 IU/mL sodium heparin filling the dead space.

KEY POINT

After collection of the sample, the syringe should have all air bubbles expelled from the lumen and a cap or capped needle should be attached.

This is necessary because air bubbles lead to spurious results during analysis. After expulsion

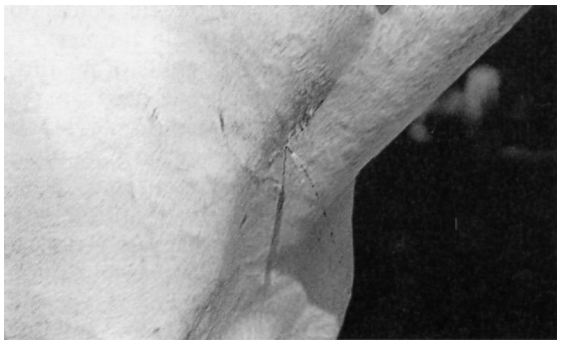


Figure 5-12. Position of 18-gauge, 3.75-cm needle in the right carotid artery to permit arterial blood gas sampling.

of the air, the needle should be capped with a rubber stopper (a Vacutainer lid is ideal), and the sample should be assayed immediately or stored in an ice water bath until analysis. Samples remain stable in an ice bath for 4 to 6 hours after collection. The horse's body temperature should be taken at the time of collection of the sample to allow correction during the assay procedure. An increase in body temperature to 40°C will have a major effect on results for arterial oxygen tension, increasing values by around 20 mm Hg. After removal of the needle, pressure should be maintained over the arteriopuncture site for several minutes to reduce the possibility of hematoma formation.

Alternative sites for collection of samples in the conscious standing horse are the *transverse facial artery* and the *palmar digital artery*, over the sesamoid bones. The transverse facial artery is located just lateral to the lateral canthus of the eye and is easily palpated. The site is aseptically prepared, the artery is stabilized with the fingers of one hand, and a 21- to 23-gauge, 1- to 2.5-cm needle is inserted into the vessel lumen. Deposition of 0.2 to 0.3 mL lidocaine under the skin over the vessel using a 25-gauge needle may prevent sensitive horses from moving their heads during arteriopuncture. With the palmar artery, this is easily palpated over the abaxial surface of one of the foreleg fetlock joints. The forelimb is held up and a 23-gauge needle inserted into the vessel and the sample collected. Collection and assay of the sample are performed as described for samples obtained from the carotid artery. In foals, an arterial sample can be collected from the great metatarsal artery in a laterally recumbent foal.

At sea level, normal horses have Pao_2 values in the range of 85 to 100 mm Hg and $Paco_2$ values in the range of 35 to 45 mm Hg. Hypoxemia (Pao_2 <80 mm Hg) usually occurs with severe pulmonary disease or ventilation-perfusion mismatching (e.g., severe consolidating pneumonia or general anesthesia). Hypoxemia in association with hypercapnia ($Paco_2$ >45 mm Hg) reflects hypoventilation. The most common diseases that cause hypoventilation are chronic allergic respiratory disease and severe pneumonia.

Sinuscentesis

In the adult horse, the dorsal margin of the maxillary sinus is a line from the infraorbital foramen caudally running parallel with the facial crest to the medial canthus of the eye. Rostrally, the limit is a line from the facial crest to the infraorbital foramen, and the caudal margin lies in a transverse plane on the rostral side of the root of the orbital process of the zygomatic bone. In the foal, the ventral limit is much higher because of incomplete eruption of the teeth. The maxillary sinus is divided into two compartments (rostral and caudal) by a complete oblique septum.



Figure 5-13. Sinuscentesis. Stab incision through a bleb raised by local anesthetic previously placed in the skin and subcutaneous tissues over the maxillary sinus.

The frontal sinus is also paired. The rostral limit is defined by the point at which the facial bones diverge. The caudal limit is near the level of the temporomandibular joint.

Sinuscentesis of the maxillary sinus is performed using local anesthesia, normally with the horse sedated. Because the frontal sinus communicates with the caudal compartment of the maxillary sinus, sinuscentesis also allows sampling of material associated with the frontal sinus. An area of skin is shaved, aseptically prepared, and local anesthetic is infiltrated under the skin. A stab incision is made in the skin and a sterile Steinmann pin is used to puncture the bony roof of the sinus (Figs. 5-13 and 5-14). A small sterile urinary catheter is introduced through the hole, and aspiration is performed (Fig. 5-15). The sample can be submitted for microbiologic and cyto-

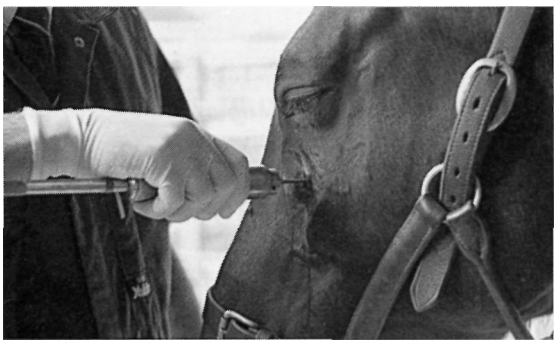


Figure 5-14. Sinuscentesis. A hole is drilled into the maxillary sinus with a Steinmann bone pin.

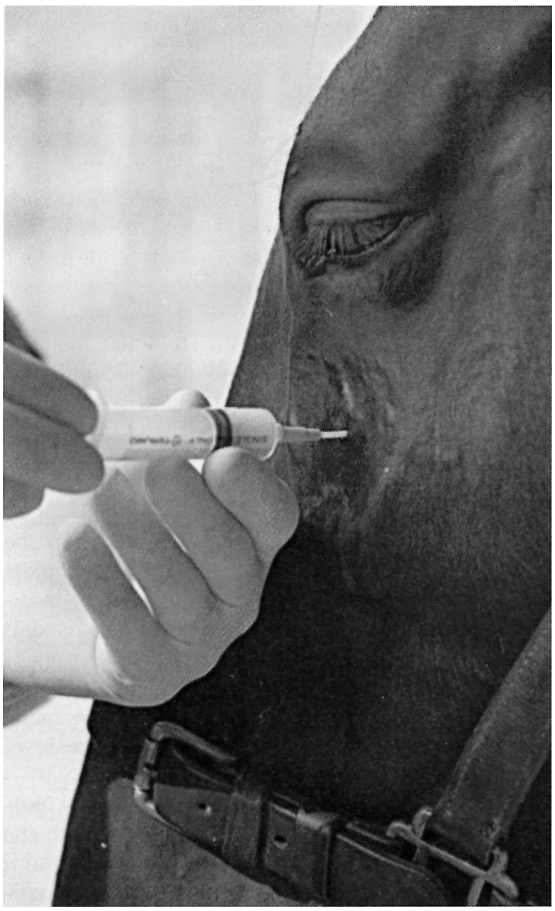


Figure 5-15. Sinuscentesis. A catheter is placed in the maxillary sinus to permit aspiration of any fluid.

logic examination. Radiographic identification of fluid accumulations before sinuscentesis may assist in obtaining a diagnostically useful sample. After collection of the sample, the catheter is removed and a single skin suture is inserted. Visual inspection of the sinus using a bronchoscope or arthroscope can be done and may provide an indication of the characteristics of the sinus lumen.

Upper Respiratory Tract Diseases

GENERAL NOSE AND THROAT PROBLEMS

Atheroma

Atheroma is a sebaceous cyst that creates a firm round swelling in the nasoincisive notch (false nostril).

HISTORY AND PRESENTING SIGNS

- Swelling near nostril
- Young horse

CLINICAL FINDINGS AND DIAGNOSIS

- Atheromas usually occur unilaterally in young horses.
- Lesions are painless, may enlarge with time, but rarely cause obstruction to airflow.
- They are easily diagnosed, because external swelling is evident over the dorsal aspect of the nasal diverticulum or false nostril.
- The contents of the cyst are usually fluid, although in some cases they may be caseous in nature.

DIFFERENTIAL DIAGNOSIS

- Congenital defects of the nostril
- Local infections (e.g., abscesses)
- Tumors (e.g., sarcoids)

TREATMENT

KEY POINT

Because of their location, atheromas seldom have any effect on airflow, but treatment is usually requested from a cosmetic point of view.

- The atheroma can be removed surgically in the standing sedated horse after regional infusion of local anesthetic or using an infraorbital nerve block. Alternatively, general anesthesia can be used to permit cyst removal. An incision is made over the dorsal aspect of the cyst and the lining is carefully dissected away from the subcutaneous tissues. The skin incision is then sutured.
- An alternative method involves incising into the cyst from the undersurface. The cyst lining is then removed, and routine closure is performed. This procedure, although a little more complicated, results in less chance of postoperative scarring. It is important to ensure ventral drainage.

Chondritis of the Arytenoid Cartilages

Chondritis is a condition most commonly affecting young racehorses in which one or both of the arytenoid cartilages undergo dystrophic changes, including enlargement, inflammation, mineralization, and the deposition of granulation tissue. The

net result of these changes is a reduction in the laryngeal luminal diameter. The cause of the condition is not known, although trauma to the cartilages, particularly by stomach tubes, and infection have been implicated. This condition is reported to be most common in North America.

HISTORY AND PRESENTING SIGNS

- Decreased exercise capacity and inspiratory stridor are the most common signs. Signs are manifested during higher-speed exercise and are the result of restricted airflow at the larynx.
- Onset of signs is often progressive.
- In severe cases, dyspnea may be present at rest.

CLINICAL FINDINGS AND DIAGNOSIS

- Palpation of the larynx may reveal abnormalities in the shape of the affected cartilage.
- Diagnosis is usually confirmed during endoscopic examination of the larynx. Changes may be mild, consisting of hyperemia, ulceration, and granulation tissue formation at the dorsomedial aspect of the cartilage(s). Advanced cases often include a thickening and change in shape of the arytenoid cartilage, reduction in the size of the laryngeal opening, and dystrophic tissue (chondroma formation) often projecting axially into the laryngeal opening.
- Unilateral involvement is most frequently observed, but bilateral lesions also occur. There may be evidence of "kissing" lesions, in which the chondritic tissue contacts the opposite arytenoid cartilage.
- Lateral radiographs of the larynx may reveal mineralization of the affected tissues.

DIFFERENTIAL DIAGNOSIS

- Idiopathic left laryngeal hemiplegia
- Subepiglottic/pharyngeal cysts
- Neoplasia of the larynx/pharynx (e.g., squamous cell carcinoma), including hypertrophic dystrophy of the arytenoid cartilages

TREATMENT

KEY POINT

In acute forms, where laryngeal obstruction is almost complete, a temporary tracheostomy and systemic antibiotic and anti-inflammatory therapy may give transient relief of the problem.

- Removal of the affected cartilage is the treatment of choice for long-term solution of this problem. A number of techniques have been described, including arytenoidectomy, subtotal arytenoidectomy, and partial arytenoidectomy.
- Unilateral arytenoidectomy is the most frequently used technique in racehorses, with the success rate for return to athletic endeavors reported to be about 45% to 60%. Mild dysphagia is a common complication of this procedure.
- The use of a neodymium:yttrium-aluminum-garnet (Nd:YAG) laser has been described for resection of excessive granulation tissue on the corniculate process of the arytenoid cartilage in the standing horse under endoscopic guidance. The Nd:YAG laser also can be used in conjunction with a ventral laryngotomy to allow improved dissection of the auricular cartilage from its muscular attachments.
- If the problem is bilateral and removal of both arytenoid cartilages is required, there is a poor prognosis for return to racing.

Epiglottic Entrapment

Epiglottic entrapment is a relatively common disorder in which the aryepiglottic folds (thick bands of mucous membrane that attach to the ventral surface of the epiglottis and extend caudodorsally to merge with mucous membranes that join the corniculate processes of the arytenoid cartilages) envelop the dorsal surface of the epiglottis. This derangement can occur because the subepiglottic epithelium is relatively loose, thereby allowing for elevation of the epiglottis during swallowing. Entrapment occurs when this loose tissue folds over the apex and lateral aspects of the epiglottis.

KEY POINT

Entrapment may result in a functional obstruction to airflow and a variety of clinical signs, but in mild cases it may result in no functional problems.

It has been suggested that this condition has a congenital basis, particularly in horses with epiglottic hypoplasia, although this hypothesis is questionable. Intermittent epiglottal entrapment has been reported that may complicate diagnosis and treatment in select cases.

HISTORY AND PRESENTING SIGNS

- Respiratory noise
- Thoroughbred and Standardbred horses
- Coughing

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CLINICAL FINDINGS AND DIAGNOSIS

- Signs are variable, with some horses showing no clinical manifestations of the disorder and entrapment being demonstrated only during a routine rhinolaryngoscopic examination.
- When entrapment does result in clinical signs, one of the major signs is turbulent airflow resulting in respiratory stridor (inspiratory and expiratory noise) and poor athletic performance.
- The severity of clinical signs depends on the degree of occlusion of the airway due to the entrapment. This is a function of the amount of tissue that is entrapped, the inflammatory response occurring in the surrounding tissues, and whether there is coexisting dorsal displacement of the soft palate.
- In some cases, mild entrapment causes no problems, as the condition has been found during routine examinations of well-performed horses.
- Diagnosis is best made by rhinolaryngoscopy, at which the entrapped epiglottis can be seen to have a distinctive V-shaped aryepiglottic fold lying over its dorsal aspect. The normal crenated appearance of the edges of the epiglottis disappears, and the vessels normally apparent on the dorsal epiglottis cannot be seen. Ulceration of the rostral margin of the entrapping mucosa may be observed in approximately 50% of cases.

DIFFERENTIAL DIAGNOSIS

- Soft palate displacement
- Idiopathic left laryngeal hemiplegia
- Subepiglottic cysts
- Laryngeal chondritis
- Ethmoid hematomas
- Diseases causing nasal deformity/obstruction
- Rostral displacement of the palatopharyngeal arch
- Epiglottic deformity

TREATMENT

- If the performance of the horse is not affected, no treatment may be necessary. In one survey of approximately 500 normal Thoroughbred horses that were examined endoscopically, 10 had entrapment. None of these animals had evidence of decreased performance.
- If performance is impeded, surgery to divide the aryepiglottic membrane is indicated. Traditionally, resection was achieved via a ventral laryngotomy and eversion of the excess mucosa. Alternative methods of correction include the use of curved hooked bistouries, electrocautery, and, more recently, the Nd:YAG laser under endoscopic guidance. Some surgeons perform these procedures in the standing horse and others via the oral or nasal route in the anesthetized animal. The results of a recent survey showed that resection using a bistoury per nasum was successful in 22 of 29 cases, with 4 horses developing recurrent entrapment. In the same study, topical application of an anti-inflammatory throat spray was successful in alleviating the condition in a small number of horses suffering from intermittent epiglottic entrapment.
- Occasionally, the epiglottis is hypoplastic, and in these cases, dislocation of the soft palate may be a subsequent result, causing continued respiratory noise and impaired performance.
- Radiographs (lateral projections) of the larynx and epiglottis obtained before surgery is performed for correction of the entrapment may provide the clinician with an indication of whether hypoplasia or other anatomic distortion of the epiglottis exists. This allows the practitioner the ability to provide the owner or trainer with a clearer idea of prognosis before surgery is undertaken.
- Complications associated with aryepiglottic resection include coughing/mild dysphagia postoperatively; nasal discharge; cicatrization of subepiglottic remnants; and, accidental division of the soft palate.

Epistaxis

Epistaxis, or bleeding from the nose, is a relatively common disorder, particularly in racehorses, as one of the sequels to exercise-induced pulmonary hemorrhage. In some cases, there is no doubt about the cause, for instance, when epistaxis follows the passage of a stomach tube. However, in many cases, the source of the blood may be difficult to determine, requiring a thorough investigation of the horse's respiratory tract. The possible sites of origin include the following:

7. ***Nasal Cavity***
   a. *Trauma* (e.g., blunt trauma, nasogastric entubation/endoscopy)*
   b. *Infection (e.g., sinusitis, conchal necrosis, fungal granuloma)*
   c. *Polyps*
   d. *Amyloidosis*
   e. *Iatrogenic (passage of a stomach tube or endoscope)*
   f. *Neoplasia*
   g. *Foreign body*
2. ***Ethmoid Labyrinth***

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   a. *Progressive hematoma* (may also occur in sinuses)*
3. ***Guttural Pouches***
   a. *Guttural pouch mycosis**
   b. *Empyema*
   c. *Ruptured longus capitis muscle*
4. ***Lungs***
   a. *Exercise-induced pulmonary hemorrhage**
   b. *Abscess*
   c. *Neoplasia*
   d. *Severe bacterial pneumonia*
   e. *Fungal pneumonia*

HISTORY AND PRESENTING SIGNS

- Blood discharging from one or both nostrils. Can vary from a trickle and intermittent to profuse and life-threatening.
- Signs can be associated with exercise.
- There may be a history of trauma to the head or nose.

CLINICAL FINDINGS AND DIAGNOSIS

- Blood at the nostrils is self-evident. The important factors to be determined are the characteristics of the discharge, its volume, and the relationship of the discharge to any recent event (e.g., exercise, nasal intubation).
- Epistaxis may occur as frank blood, and this is often seen after trauma to the nasal conchae or hemorrhage from the guttural pouches.
- Serosanguineous fluid often accompanies conditions in which chronic inflammatory changes occur in association with the hemorrhage. Examples are ethmoid hematomas, sinusitis, hemorrhage from neoplasms, and abscesses.
- It is important to determine whether the epistaxis occurs in association with other systemic signs, such as fever, depression, inappetence, or cough.
- Diagnosis is based on careful accumulation of information from the history and physical examination. Important details of the history include whether the hemorrhage occurs in association with exercise or subsequent to an infectious or traumatic process involving the respiratory tract. A thorough inspection of the nares and other parts of the upper respiratory tract, including percussion of the sinuses and palpation over the guttural pouches and larynx and trachea, should be undertaken.
- Endoscopy often is vital in determining the site of hemorrhage. A careful and thorough examination of the airway is necessary, paying particular attention to ensuring that sites are examined from which hemorrhage is common. These include the nasal meatus, nasomaxillary openings, ethmoid labyrinth, guttural pouch openings (and possibly the pouches themselves if hemorrhage is detected), and the trachea.
- Additional diagnostic aids include radiography of suspected sites of hemorrhage, possibly ultrasound examination, transtracheal lavage or BAL, and aspiration if previous hemorrhage from the lungs is suspected. Also, judicious use of relevant clinical laboratory data to assess the degree of systemic response may be helpful if the problem appears to be more generalized. Measurements such as total and differential leukocyte counts and fibrinogen concentration may indicate an underlying inflammatory process.
- The conditions described in this section that are known to cause epistaxis are dealt with as individual conditions elsewhere in this chapter.

*Most common causes.

DIFFERENTIAL DIAGNOSIS

- Trauma (e.g., blunt trauma, nasogastric intubation/endoscopy)
- Infection (e.g., sinusitis, conchal necrosis, fungal granuloma)
- Foreign body
- Nasal polyps
- Amyloidosis
- Progressive hematoma of the ethmoid or sinuses
- Guttural pouch mycosis or empyema
- Exercise-induced pulmonary hemorrhage
- Severe acute pneumonia
- Pulmonary abscess
- Respiratory tract neoplasia
- Fungal infections of the respiratory tract
- Thoracic trauma

TREATMENT

- Treatments for the individual conditions are described in the relevant sections.

Ethmoid Hematoma

Hematomas of the ethmoid and paranasal sinuses are slowly progressive tumors that resemble hemangiomas in many ways but apparently are not neoplastic or metastatic. The most common location for such lesions is in the ethmoid (labyrinthal) region, with the paranasal sinuses less commonly affected. Given the progressive nature of the disease, older horses are most commonly affected. However, the condition has been observed in younger animals.

HISTORY AND PRESENTING SIGNS

- Nasal discharge that may be unilateral or bilateral
- Epistaxis

CLINICAL FINDINGS AND DIAGNOSIS

- Intermittent epistaxis apparent at rest and after exercise in an older horse is the most consistent clinical sign. This is usually mild and unilateral and occurs because the progressive enlargement of the tumor results in its outstripping its blood supply. As a result, the surface undergoes necrosis, and hemorrhage occurs.
- Severe cases may invade the surrounding tissues, resulting in dyspnea, particularly during exercise.
- Endoscopy is useful to determine the location of the tumor, which usually is located dorsally in the nose. The surface of the hematoma usually glistens and is green/red in color. Hemorrhage may be seen originating from the tumor. Bleeding is common if the hematoma is traumatized by the endoscope. If a horse has a history of epistaxis not necessarily related to exercise, the ethmoid region should always be examined for the presence of a progressive hematoma.
- Radiography is an integral part of the diagnosis to assist in outlining the soft tissue mass. If lesions are present in the paranasal sinuses, radiography provides the most effective means for delineation of the tumor.
- Histopathologic examination of a biopsy specimen or samples collected after removal of the lesion reveals tissue types consistent with those described for ethmoid hematomas. The tissue is vascular, with evidence of repeated hemorrhage and reorganization. Based on their histologic appearance, these lesions have been described as congenital vascular anomalies, hence the term *angiomatosis.* Surface tissue is composed predominantly of epithelium and fibrous tissue.

DIFFERENTIAL DIAGNOSIS

- Guttural pouch mycosis/empyema
- Trauma
- Primary or secondary sinusitis
- Foreign body
- Fungal infection
- Neoplasia (e.g., fibroma/fibrosarcoma, squamous cell carcinoma, adenocarcinoma)
- Conchal necrosis
- Amyloidosis
- Exercise-induced pulmonary hemorrhage

TREATMENT

KEY POINT

Surgical removal of the tumor masses may provide palliation of the disease for variable periods. Unfortunately, recurrence is common, particularly when the tumor is only excised or removed with a wire snare.

- Surgical approach to the tumor is often difficult, requiring a frontonasal flap or sinus flap, and is complicated by the potential for significant blood loss. This may be reduced by using temporary bilateral carotid artery occlusion.
- Cryofreezing of the tumor and removal of the mass while still frozen have been advocated to decrease the chance of hemorrhage and improve the potential for removal of most of the diseased tissue. Cryosurgery also has the advantage of producing cell death in residual tumor tissue. At least three freeze-thaw cycles are advocated to optimize cellular destruction. Freezing to temperatures lower than —20°C provides the most satisfactory results. Disadvantages of cryosurgery are possible damage to local healthy tissues, including mucosa, the infraorbital nerve, and possibly the cribriform plate. In addition, nasal discharge may persist for weeks postoperatively.
- More recently, injection of the tumor with formalin under endoscopic guidance has proved useful and is reported to curtail tumor growth and hemorrhage in a select group of cases.

Fourth Branchial Arch Defects

The term *fourth branchial arch defect* (4-BAD) has been adapted from the condition reported in humans to describe a number of abnormalities observed in the equine larynx/pharynx. During embryogenesis it is thought that the extrinsic laryngeal musculature and cartilages develop from the fourth branchial arch. The intrinsic laryngeal musculature and structures develop from the sixth branchial arch. Traditionally, the endoscopic finding of rostral displacement of the palatopharyngeal arch (RDPA) has been used to describe a single entity. It appears, however, that RDPA may in fact be one of the recognizable signs in the syndrome of 4-BADs.

HISTORY AND PRESENTING SIGNS

- Abnormal respiratory noise
- Belching/eructation
- Nasal discharge

- Coughing
- Recurrent colic
- Poor performance

KEY POINT

It should be noted that many horses with 4-BAD may in fact show no overt clinical signs, and the condition is diagnosed when routine endoscopic examination of the upper airway is performed.

CLINICAL FINDINGS AND DIAGNOSIS

- Endoscopic examination of the pharynx/larynx is fundamental in the diagnosis of this condition. Persistent rostral displacement of the palatopharyngeal arch close to the corniculate processes of both arytenoid cartilages together with cricopharyngeal dilation is highly suggestive of 4-BAD.
- Radiography can also assist in making a definitive diagnosis. In lateral radiographs the palatopharyngeal arch may be seen rostrodorsal to the corniculate processes in conjunction with a column of air extending from the cricopharynx to the cervical esophagus. Contrast studies using barium or fluoroscopy during swallowing may demonstrate the lack of a cricopharyngeal sphincter and the absence of primary esophageal peristalsis.
- Laryngeal palpation may reveal abnormalities of the thyroid/cricoid cartilages.

DIFFERENTIAL DIAGNOSIS

- Idiopathic laryngeal hemiplegia
- Displacement of the soft palate
- Pharyngeal lymphoid hyperplasia
- Pharyngeal paralysis
- Arytenoid chondropathy/chondritis
- Wind sucking (aerophagia)

TREATMENT

- To date there has been no effective surgical or medical treatment of this condition, and it appears that clinically affected horses have a poor prognosis for athletic activity.

GUTTURAL POUCH PROBLEMS

The guttural pouches communicate with the pharynx through the slit-like epipharyngeal orifices on the dorsolateral walls of the pharynx. The function of these pouches is unclear, but because of their location and the fact that the epipharyngeal orifices open during swallowing, extension of bacterial upper respiratory infections into the guttural pouches may occur. Several disorders involving the guttural pouches may occur and can limit performance.

HISTORY AND PRESENTING SIGNS

- Nasal discharge
- Epistaxis
- Distention in the region of Viborg's triangle

Guttural Pouch Empyema

Bacterial infection and pus accumulation in the guttural pouch can occur as a secondary event to upper respiratory tract infections, particularly infections due to *Streptococcus equi* ("strangles"). The exact pathogenesis of guttural pouch empyema is uncertain. However, it is possible that the condition may occur as a result of chronic suppurative pharyngeal lymphandenitis and or a dysfunction of mucociliary clearance mechanisms resulting in accumulation of purulent material.

CLINICAL FINDINGS AND DIAGNOSIS

KEY POINT

Apart from poor performance, the major clinical manifestation of guttural pouch empyema is a persistent unilateral or bilateral nasal discharge.

- This discharge usually is increased when the horse swallows, lowers its head, or the region of the guttural pouches is palpated.
- There may be little or no swelling in the region of Viborg's triangle and few other systemic signs.
- The condition may be diagnosed on endoscopic examination of the pharynx. Purulent material can be seen discharging from the guttural pouch opening(s), particularly when the horse's head is lowered or after swallowing.
- At times, passage of the endoscope into the pouch may be necessary to confirm the diagnosis.
- Radiography of the pharynx also may confirm the presence of fluid or accumulated material within the guttural pouches.
- Aspiration of a sample of purulent material via a catheter may be useful for confirmation of the diagnosis (cytology, Gram stain) and culture of the offending bacteria.

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DIFFERENTIAL DIAGNOSIS

- Primary or secondary sinusitis
- Ethmoid hematoma
- Guttural pouch mycosis
- Trauma
- Foreign body
- Fungal infection of the nares or lower respiratory tract
- Neoplasia (e.g., fibroma/fibrosarcoma, squamous cell carcinoma, adenocarcinoma)
- Conchal necrosis
- Amyloidosis

TREATMENT

KEY POINT

The most common infecting organisms are Streptococcus *spp., particularly* S. equi, *var.* zooepidemicus *and* S. equi *var.* equi. *These organisms are sensitive to penicillin, so local lavage and systemic therapy are often successful.*

- Parenteral procaine penicillin G (Treatment No. 84) at a dose rate of 15 to 20 mg/kg (15,000-20,000 IU/kg) ql2h, combined with local irrigation with warm isotonic polyionic solutions containing crystalline penicillin (Treatment Nos. 85 and 86) once or twice daily for 5 days will usually resolve the infection.
- Catheterization of the guttural pouch is performed using a plastic mare uterine infusion catheter with the tip bent (see Guttural Pouch Catheterization). Catheters can be inserted blind or with the aid of an endoscope to view the pharynx.
- In severe cases, particularly those in which the purulent material has become caseous, surgical drainage of the guttural pouch may be required. In chronic cases, the inspissated pus eventually becomes solid and forms chondroids. A number of techniques providing surgical access to the guttural pouch have been described. The most common technique is performed through Viborg's triangle. However, we have found that the dorsal (hyovertebrotomy) approach just cranial to the atlas allows better exposure. Care must be taken to avoid vital structures such as the vagus and glossopharyngeal nerves and the internal carotid artery.

Guttural Pouch Mycosis

Guttural pouch mycosis occurs more commonly in horses in the northern than those in the southern hemisphere. The common sites for mycotic plaques to occur are overlying the internal carotid artery within the medial compartment and over the external carotid artery within the lateral compartment of the guttural pouch. It is unclear whether the mycosis is a primary lesion or occurs secondarily after an arterial wall aneurysm. The most common presenting sign is epistaxis, occurring as a result of erosion through the carotid artery, which in advanced cases may be fatal.

CLINICAL FINDINGS AND DIAGNOSIS

KEY POINT

The most common signs associated with guttural pouch mycosis are epistaxis and dysphagia due to involvement of cranial nerves VII, IX, X, and XI, which traverse the region of the guttural pouches.

- Other signs may include parotid pain, nasal discharge, abnormal head posture, and Horner's syndrome.
- Positive diagnosis is made on the basis of history, clinical signs, and endoscopy, in particular by introducing the endoscope into the guttural pouch, where a typical diphtheritic membrane, hemorrhage, and discharge may be seen.
- Care should be taken during the passage of the endoscope into the pouches because severe hemorrhage may be induced if the mycotic lesion is traumatized.
- The specific fungal pathogen producing the infection is often not identifiable, although *Aspergillus* spp. (ubiquitous fungi) are commonly cultured from affected pouches.

DIFFERENTIAL DIAGNOSIS

- Primary or secondary sinusitis
- Ethmoid hematoma
- Guttural pouch empyema
- Trauma
- Foreign body
- Fungal infection of the nasal passages or paranasal sinuses
- Neoplasia (e.g., fibroma/fibrosarcoma, squamous cell carcinoma, adenocarcinoma)
- Conchal necrosis
- Amyloidosis
- Rupture of the longus capitis muscle

TREATMENT

- Topical treatment using the same methods described for empyema may be attempted.

KEY POINT

Because lesions are often confined to the dorsal aspect of the pouch, delivery of medications (e.g., 1% miconazole [Treatment No. 76], 1-5% ketoconazole [Treatment No. 65], enilconazole as a 33.3-mg/mL solution [Treatment No. 37], amphotericin B [Treatment No. 7]) to the site of infection can be difficult, and response is often slow. Daily infusions for 4 to 6 weeks may be necessary.

- If the internal carotid artery is involved, ligation of this vessel is important because profuse life-threatening hemorrhage can occur. This provides the most useful and effective means for controlling epistaxis in affected animals. Several techniques are described; however, the use of a balloon catheter inserted into the artery to a point beyond the area of fungal infection and ligation of the vessel proximal to the lesion appear to provide the most satisfactory results. In severe cases of epistaxis, blood transfusions may be required.
- Systemic treatment of guttural pouch mycosis with intravenous ketoconazole (Treatment No. 65) has been reported, although this agent has no specific in vitro activity against *Aspergillus* spp. Amphotericin B (Treatment No. 7) also may be a possible drug for systemic administration, although a number of untoward side effects have been reported with prolonged use. These include nephrotoxicity, phlebitis near the site of injection, anorexia, and signs of depression. Newer antifungal agents include itraconazole at 2.6 mg/kg per os ql2h.
- It is questionable whether topical antimycotic agents are required in the treatment of this disease. Repeated guttural pouch catheterization may increase the risk of iatrogenic trauma and hemorrhage. Recent reports suggest that topical medication is not necessary for the complete resolution of the mycotic lesion if carotid arterial ligation/occlusion is successful.
- Complications of guttural pouch mycosis are fatal epistaxis, pharyngeal paralysis and dysphagia, laryngeal hemiplegia, cranial nerve dysfunction (including CN VII, Horner's syndrome), otitis media/interna, osteomyelitis of the stylohyoid bone/temporomandibular joint, and aspiration pneumonia.

Guttural Pouch Tympany

This condition is a developmental disorder and is therefore most common in foals. The precise etiology of the disease has not been defined, and it has been suggested that this disease has a functional rather than a mechanical basis, such that air is trapped within the guttural pouch or pouches, producing tympany. Suggestions have been made that the disease occurs because of a malformation of one or both epipharyngeal orifices, resulting in air becoming trapped in the guttural pouch. This has not been proven.

CLINICAL FINDINGS AND DIAGNOSIS

KEY POINT

Marked, nonpainful distention of one or both sides of the neck in the region of Viborg's triangle in foals or weanlings is the usual presenting sign.

- Percussion over the swollen area will reveal tympany.
- If the enlargement is sufficiently great, obstruction to airflow may occur, producing dyspnea. In severe unilateral cases, swelling may extend to the opposite side of the neck despite an absence of the disorder in that pouch.
- Complications include dysphagia, nasal discharge, secondary infections of the pouch (empyema), and aspiration pneumonia.
- Affected foals may make a "snoring" sound when sucking.
- Diagnosis is usually based on the physical examination and pathognomonic appearance of the swollen pouch.
- Apart from swelling extending into the pharynx, endoscopic findings are usually unremarkable.
- Radiographs, in the dorsoventral plane, are helpful if there is a question as to whether the condition is unilateral or bilateral.
- Additionally, aspiration of air from a distended pouch may assist in diagnosis.

DIFFERENTIAL DIAGNOSIS

- Guttural pouch empyema
- Congenital malformations

TREATMENT

- Temporary relief may be afforded by insertion of a catheter into the pouch via the nasopharynx, needle aspiration, or digital pressure on the pouch.
- A commonly practiced, potentially permanent method of treatment involves surgical correction of the problem.
- In unilateral cases, fenestration of the septum between the two pouches often affords relief.
- In bilateral cases, the treatment of choice is

enlargement of the pharyngeal openings of one or both guttural pouches. This is done using a procedure in which the pouch is entered via Viborg's triangle. If only one opening is enlarged, fenestration of the septum is imperative. An alternate and less traumatic method involves insertion of a balloon-tipped catheter into the pouch via the nasopharyngeal opening. The balloon is filled with water to assist in maintaining it in position. This catheter is left in place for several weeks or longer, with the intention of producing a permanent ostium into the pouch.

- A technique for external fistulation of the affected pouch has also been described, and more recently transendoscopic Nd:YAG laser fistulation through the medial septum has been shown to provide a relatively noninvasive alternative for treatment.
- The prognosis for this disorder is often good if the foal does not have significant complications (e.g., aspiration pneumonia) and if the condition is unilateral. However, dysphagia after surgical intervention has been reported irrespective of the technique used.

NASAL PASSAGE PROBLEMS

There are a number of disorders that can involve the nasal passages. These include foreign bodies, amyloidosis, fungal diseases, disorders of the nasal septum, neoplasia, polyps, and necrosis of the conchae.

HISTORY AND PRESENTING SIGNS

- Nasal discharge
- Respiratory noise
- Dyspnea

CLINICAL FINDINGS AND DIAGNOSIS

KEY POINT

Manifestations of diseases of the nasal passages may include epistaxis, nasal discharge, malodorous breath, alterations in airflow, and respiratory noise.

- Careful collation of the information obtained from the history and physical examination is invaluable if a correct diagnosis is to be made.
- Judicious use of diagnostic aids (e.g., endoscopy, radiography, biopsy, and collection of samples for cytologic and microbiologic examination) is important.
- Characteristics of the specific disorders include the following.

Foreign Bodies

- Often induce "head shyness," epistaxis, and obvious discomfort for the horse.
- Malodorous nasal discharge is common.
- Endoscopy and radiography are often useful for diagnosis.

Amyloidosis

- Horses of all ages have been reported to deposit glycoprotein in various body organs in response to continued immunologic stimulation such as repeated vaccination, chronic infection, and neoplasia.
- A common site for deposition of amyloid is the upper airway, including the nasal passages, particularly the nasal septum and conchae.
- Clinical manifestations include nasal discharge, epistaxis, reduced exercise tolerance, and possibly dyspnea and weight loss.
- Lesions are firm and nodular or plaque-like with fragile, smooth walls that are easily traumatized.
- Diagnosis can be made by clinical examination, endoscopy, a history of chronic antigenic stimulation, biopsy, and histopathology.

Fungal Infections

- Although quite rare, fungal diseases of the upper airways are more common in tropical and subtropical regions.
- Fungi implicated include *Conidiobolus*, *Cryptococcus*, *Coccidioides*, *Aspergillus,* and *Rhinosporidium.*
- Clinical signs of fungal infections of the upper airway may include mucopurulent and possibly malodorous nasal discharge, epistaxis, dyspnea, respiratory noises, and submandibular and parotid lymphadenopathy.
- Diagnosis is based on clinical signs, endoscopic appearance, smears and culture of the nasal discharge and of material from the lesions, and histopathologic examination.
- Endoscopically, the fungal lesions may consist of cream-colored, plaque-like accumulations, progressing to ulcerated granulomatous lesions in advanced cases.

Disorders of the Nasal Septum

- Usually this is a congenital disorder that becomes obvious with induction of respiratory stridor when the horse undertakes exercise.
- Attention may be directed toward septal disorders earlier in the horse's life if they involve malformations of the other nasal bones.
- Other causes of nasal septum dysfunction in-

clude trauma, septal and conchal necrosis secondary to severe respiratory tract infections, amyloidosis, and fungal infections. Mucoid degeneration and deformity of the nasal conchae also can be present as a result of nutritional secondary hyperparathyroidism.
- The most common clinical manifestation is respiratory stridor (louder on inspiration) and dyspnea during exercise.
- Facial deformity may be present.
- Diagnosis is based on a history of trauma, infection, or evidence of facial deformity.
- Deformities in the rostral portion of the septum can be detected by palpation and visual inspection. Lesions located more caudad may require inspection with an endoscope.
- Dorsoventral radiographs also may provide useful information about the degree of tissue disturbance present.
- Biopsy samples collected from local lesions may help identify specific etiologic agents in certain cases.

DIFFERENTIAL DIAGNOSIS

- Ethmoid hematoma
- Tooth-root abnormalities
- Trauma of fractures of the nasal region
- Atheroma of the false nostril
- Epistaxis
- Tracheal stenosis
- Idiopathic laryngeal hemiplegia
- Arytenoid chondritis

TREATMENT

Foreign Bodies

- Removal with forceps with or without the aid of an endoscope may be possible.
- In severe cases, more invasive procedures may be indicated.

Amyloidosis

- Removal of the chronic antigenic stimulus may reduce the continued deposition of amyloid. However, it is likely that this has no effect on the glycoprotein already deposited.
- If the amyloid deposits are restricted to well-defined, circumscribed lesions, surgical removal of the lesions may be beneficial.
- Corticosteroid administration appears to provide no benefit to affected horses.

Fungal Infections

- In many cases, surgical removal of the masses is successful.
- The antifungal agents amphotericin B (intravenously and locally; Treatment No. 7) and ketoconazole (intravenously; Treatment No. 65) may be effective in lesions that are not extensive. Ketaconazole is often the drug of choice and is administered at a dose rate of 300 mg/kg IV ql2h for 7 to 10 days. A less expensive alternative is the oral administration of potassium iodide at a dose rate of 20 g/500 kg q24h until regression of signs.

Disorders of the Nasal Septum

- Surgical removal of the septum is indicated if respiratory stertor causes reduced performance or if there are areas of necrotic tissue present.
- Resection of the septum is a relatively complex procedure, and blood loss can be profuse.
- In cases of nutritional secondary hyperparathyroidism, the underlying calcium and phosphorus imbalance must be corrected to prevent further progression of disease.

Nasal Polyps

Nasal polyps are slow-growing pedunculated tumors originating from the nasal mucosa, septum, and on occasion the tooth roots. Lesions are frequently unilateral and result from proliferation of connective tissue in response to chronic inflammation or from hypertrophy of the mucosa.

HISTORY AND PRESENTING SIGNS

- Occur in horses of all ages.
- Progressive dyspnea, particularly with exercise, is common.
- Nasal discharge, frequently malodorous, is common. Epistaxis is rare.
- In some cases, owners, trainers, or handlers may see a polyp in the naris of the affected side.

CLINICAL FINDINGS AND DIAGNOSIS

- Diagnosis can be made on direct inspection of the nasal cavity if the polyp extends to near the external naris. As polyps grow, they may invade the entire lumen of the nasal cavity on the affected side and even protrude out the nostril. Lesions up to 30 cm long have been reported.
- Polyps are usually creamy white and pedunculated and have a smooth surface.
- Diagnosis can be confirmed with endoscopy. In extensive cases, polyps may be seen extending caudally beyond the nasal meatus when viewed through the opposite nostril.
- Radiographs also may assist in delineation of the margins of the mass.
- Confirmation of the diagnosis may be made with

biopsy, which will reveal mature and immature fibrous tissue covered with epithelium.

DIFFERENTIAL DIAGNOSIS

- Neoplasia (e.g., fibroma/fibrosarcoma, squamous cell carcinoma, adenocarcinoma)
- Foreign body
- Fungal infection
- Amyloidosis
- Nasal malformations
- Conchal necrosis

TREATMENT

- Surgical removal is the treatment of choice and is often quite feasible. Methods used vary according to the size and location of the polyp.
- Because of the potential for regrowth if all abnormal tissue is not removed, most procedures involve direct access to the lesion and complete excision.
- Access to a polyp near the external naris can be gained by opening the nostril between the false nostril and nasoincisive notch. Removal of polyps located further caudad usually requires trephination or creation of a bone flap though the nasal bones to the site of the polyp.
- It must be remembered that polyps have an excellent vascular supply, and hemorrhage is often extensive, requiring packing of the nostrils.
- Removal of lesions via the external nares using a wire snare often provides only temporary relief because the polyps regrow as a result of inadequate resection using this technique.
- Use of the Nd:YAG laser may allow easier, less invasive access for effective surgical ablation or coagulation of the polyp mass.

LARYNGEAL, PHARYNGEAL, AND TRACHEAL PROBLEMS

Idiopathic Left Laryngeal Hemiplegia

Idiopathic laryngeal hemiplegia (ILH) is a disorder occurring mainly in young, large Thoroughbred and draught horses and affecting the left side of the larynx. The incidence of ILH is probably in the range of 3 to 8% in Thoroughbred horses, but a higher incidence has been reported in draught horses. ILH results from demyelination of the left recurrent laryngeal nerve (and some other long peripheral nerves), denervating the muscles of the larynx, particularly the cricoarytenoideus dorsalis (CAD), which is responsible for abduction, and the cricoarytenoideus lateralis (CAL), the adductor. Although it is known that the lesion results from repeated bouts of segmental demyelination resulting in a distal axonopathy, the pathogenesis of the disorder remains obscure. In left laryngeal hemiplegia, atrophy of the left CAD muscle often can be determined by palpation. The result of the failure of abduction of the left arytenoid cartilage is a partial upper respiratory tract obstruction, which produces an inspiratory dyspnea. It usually becomes evident at 2 or 3 years of age. A heritable basis for the disease has been proposed but not definitively established.

HISTORY AND PRESENTING SIGNS

- Respiratory noise during fast exercise
- Noise becoming progressively worse
- Poor performance
- Thoroughbred horse, 2 to 3 years old

CLINICAL FINDINGS AND DIAGNOSIS

- The first sign of laryngeal hemiplegia is a respiratory noise, usually evident during fast exercise, and/or reduced performance capacity.
- The noise may vary from a whistle (early stages) to a roar and is inspiratory.
- The condition is progressive, and therefore the noise eventually becomes evident at slower gaits.

KEY POINT

On clinical examination, the larynx also should be palpated to determine if atrophy of the dorsal cricoarytenoid muscle can be felt.

- If atrophy of the CAD muscles is present, the muscular process of the left arytenoid cartilage, normally not very prominent, will be more prominent than on the right side and can be felt as a bump (see Fig. 1-14). Note should be taken of any scarring or thickening of skin that could suggest a previous laryngoplasty or laryngotomy.
- If there is any indication of more prominence of the left muscular process of the arytenoid cartilage, a laryngeal adductor test (slap test) should be performed while palpating the muscular process on left and right sides. Slapping the left thorax gently with the open hand should result in adduction of the right muscular process, and this can be felt as a flicking or movement of the process. Similarly, slapping of the right thorax should result in flicking of the muscular process of the left arytenoid cartilage. In horses

with ILH, arytenoid adductor function is lost before abductor function; therefore, flicking of the left muscular process does not occur or is reduced in response to the slap test. However, the response also may be lost in horses with cervical spinal cord disease and should not be considered specific for ILH.

- The laryngeal depression test is useful in horses immediately after exercise. With this procedure, the muscular process of each arytenoid cartilage is depressed while the horse's respiratory effort remains elevated after exercise. Horses with ILH will often make a more profound stertorous sound when the left side is depressed as compared with the right side.
- Positive diagnosis is only possible with use of a rhinolaryngoscope, where an atypical appearance of the larynx will be found. Features include an asymmetric appearance, a kink in the aryepiglottic fold, and a failure to abduct the left arytenoid cartilage, which may be most obvious immediately after the horse swallows. Nostril occlusion or use of the slap test during endoscopic examination (with the horse not sedated) improves the chances of diagnosis of ILH by allowing direct observation of abductor and adductor function.
- Asynchronous movement of the arytenoid cartilages is a normal finding in some horses and appears not to progress to clinical laryngeal hemiplegia.
- The availability of high-speed treadmills has provided a mechanism whereby endoscopy can be performed during exercise. This procedure may assist in the diagnosis of ILH, particularly in those cases in which diagnosis is equivocal at rest. Recent investigations have suggested that a percentage of horses suffering from grade 3 dysfunction may experience limitations in performance (i.e., reduced arterial oxygen content) similar to those suffering from grade 4 to 5 dysfunction (see also Diagnostic Aids, p. 189).

DIFFERENTIAL DIAGNOSIS

- Soft palate displacement
- Epiglottic entrapment
- Subepiglottic cysts
- Laryngeal chondritis/chondropathy
- Rostral displacement of the palatopharyngeal arch
- Diseases causing nasal deformity/obstruction (e.g., sinusitis, neoplasia)
- Fourth branchial arch defects

TREATMENT

- Surgical treatment is possible, and the combination of laryngoplasty ("tie back") and ventriculectomy (± vocal cordectomy) has given best results to date. The current technique of laryngoplasty involves insertion of material such as Mersilene, Dacron, Polydek, or Ethibond to mimic the action of the atrophied left CAD muscle so that the left arytenoid cartilage is permanently abducted. A ventriculectomy is performed to create adhesions between the thyroid and arytenoid cartilages, thereby holding the arytenoid cartilage in an abducted position, because the suture material usually pulls through the cartilage in 2 to 3 months. A recent study demonstrated, however, that largyngoplasty in conjunction with ventriculocordectomy does not necessarily improve upper respiratory function when compared with laryngoplasty alone.
- Although the initial appearance of the larynx after surgery is often good, the left arytenoid cartilage often sags inward after several months, causing a partial obstruction.
- The major complication of surgery is chronic coughing, which results from food particles entering the trachea, causing a chronic tracheitis. Other complications include incisional sinus tract formation and wound dehiscence.
- The result of this procedure is that 50 to 60% of horses will have little or markedly reduced inspiratory noise after surgery, and most horses will have improvement in airflow. Some authors have suggested that up to 70% of younger horses (i.e., <2 years old) and those with grade 3 laryngeal dysfunction may have successful outcomes after surgery. Most horses require 3 months' rest after surgery, before recommencing training.
- Arytenoidectomy has also been evaluated as a treatment method for ILH. Early investigations of subtotal arytenoidectomy in Standardbreds did not show an improvement in upper airway mechanics. However, more recently, the technique of partial arytenoidectomy performed after experimental induction of ILH reduced upper airway inspiratory impedance, although not to within normal limits. Nonetheless, this technique may be an alternative in cases in which laryngoplasty has failed.
- The technique of nerve pedicle grafting has been described as a technique that attempts to normalize laryngeal function during exercise. The procedure involves implantation of a nerve-muscle pedicle graft, over the site of the atrophied CAD muscle, created from the omohyoideus muscle and associated first cervical nerve. The surgical technique is purported to result in fewer postoperative complications when compared with laryngoplasty. However, although it appears that reinervation of the CAD can occur as early as 3

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months postoperatively, it may take up to 12 months before laryngeal function is improved sufficiently to permit racing. Additionally, better results are apparently obtained in horses with CAD paresis compared with those suffering from complete paralysis of the left CAD (i.e., grade 5 laryngeal dysfunction). It would appear, therefore, that this technique would be best suited to young horses (2-3 years old) with grade 3 to 4 laryngeal dysfunction that may have a longer-term racing future. When more immediate results are required and with older animals suffering from more advanced ILH, traditional laryngoplasty with or without ventriculocordectomy or arytenoidectomy appears to be the best option.

Pharyngeal Lymphoid Hyperplasia

Pharyngeal lymphoid hyperplasia (PLH) is a condition commonly afflicting young horses in training. The disorder promotes controversy, because disagreement exists as to the significance of the lesions, although many authorities now seem to agree that PLH is a normal physiologic finding in younger horses. It is thought that PLH is a manifestation of an immunologic response of the mucosa within the pharynx. Because young horses have the greatest density of lymphoid follicles within the pharynx, it is not surprising that PLH is more common in this age group. Possibly, viruses that damage the upper respiratory tract, inhaled irritants, and pollutants contribute to the induction of hyperplasia. Classically, horses with PLH have a chronic cough and poor performance, which may be a sequel to a viral upper respiratory tract infection.

HISTORY AND PRESENTING SIGNS

- Horses may have a chronic cough.
- Reduced performance may be reported in some horses.
- PLH may be an incidental finding on endoscopic examination of the upper airway.

CLINICAL FINDINGS AND DIAGNOSIS

- Diagnosis of PLH is made by rhinolaryngoscopic examination, which reveals hyperplasia of the lymphoid tissue ("follicles") on the roof of the pharynx.
- The degree of hyperplasia can be graded (see below). There also may be some discharge associated with a general inflammatory response.

KEY POINT

Care must be taken in interpretation of endoscopic findings with regard to PLH, because there are a number of studies that demonstrate that PLH has little or no effect on respiratory gas exchange.

- Racing performance is unlikely to be impaired by PLH under most circumstances, although in severe cases (grades III-IV), decrements in performance and a chronic cough may occur.

Grades of PLH

Grade I: Very few, small, inactive follicles in the pharynx.

Grade II: More follicles with a wider distribution than seen in grade I. Follicles can be seen on the dorsal and lateral pharynx. Some follicles are edematous.

Grade III: Many more, larger, pink/red follicles than in grade II. The pharyngeal tonsillar tissue is often hyperplastic. Follicles may be seen on the soft palate.

Grade IV: More severe manifestations of the changes seen in grade III. Many large edematous follicles with a wide distribution, including the dorsal and lateral pharyngeal walls, soft palate, and possibly the epiglottis. Small polyp-like lesions may be observed.

DIFFERENTIAL DIAGNOSIS

- Idiopathic laryngeal hemiplegia
- Soft palate displacement
- Epiglottic entrapment
- Subepiglottic cysts
- Viral respiratory disease
- Retropharyngeal abscessation

TREATMENT

- A number of treatments have been described for more severely affected horses. Often rest for 8 to 12 weeks is sufficient to resolve the problem.
- In North America, surgical management of PLH by means of cryosurgery, chemical cautery, electrocautery, and more recently, coagulative necrosis using the Nd:YAG laser have been used.
- However, because PLH often is self-limiting, such treatments may be indicated only in the most severe cases.
- Most treatments require a period of convalescence/reduced training of 4 to 6 weeks, sometimes in conjunction with anti-inflammatory and antibiotic therapy.

Retropharyngeal Abscessation

The most common cause of retropharyngeal abscessation in the horse is severe lymphadenopathy that accompanies *S. equi* var. *equi* infections ("strangles"). A much less common cause of cellulitis and abscessation in this site is pharyngeal trauma. Because of the pyogenic nature of the organisms that often induce this pharyngeal abscessation or cellulitis, systemic manifestations of these disorders are common.

HISTORY AND PRESENTING SIGNS

- Retropharyngeal abscessation often follows a history of strangles in the affected animal or the herd.
- Signs frequently result from the presence of a space-occupying lesion and include local swelling and possibly dyspnea.
- In severe cases, dysphagia and nasal discharge may be present in addition to respiratory embarrassment.

CLINICAL FINDINGS AND DIAGNOSIS

- Diagnosis is based on history, clinical manifestations, and clinical pathologic findings.
- Palpation of the retropharyngeal region may reveal pain and swelling. Most commonly this is unilateral and is apparent as a reduction in the lumen size of the nasopharynx.
- Systemic manifestations include fever, signs of depression, inappetence, and lower respiratory tract disorders (possibly due to aspiration). Coughing may be present.
- Endoscopy may reveal distortion of the pharynx and possibly the larynx with a diminished airway diameter. If the endoscope can be passed into the guttural pouch, distortion of the medial compartment on the affected side is the most frequent observation, and in some cases enlarged lymph nodes can be observed ventromedially.
- On radiographic examination of the pharyngeal area, a large soft tissue density impinging on the pharynx and larynx is often visible.
- Clinical pathologic findings may reveal a leukocytosis, hyperfibrinogenemia, hyperproteinemia (hyperglobulinemia), and other changes depending on the degree of systemic involvement.

DIFFERENTIAL DIAGNOSIS

- *S. equi* var. *equi* infection ("strangles")
- Pharyngeal trauma
- Diseases causing upper airway obstruction (e.g., subepiglottic cysts, guttural pouch empyema, laryngeal paralysis, laryngeal chondritis)
- Guttural pouch tympany
- Guttural pouch/pharyngeal cysts

TREATMENT

- In most cases, medical management of retropharyngeal abscessation provides resolution of the problem, although the treatment often needs to be maintained for several weeks.
- Administration of appropriate doses of antimicrobial agents is the mainstay of this form of therapy (see Chapter 18). Most cases are the result of infections with *Streptococcus* spp. Penicillin is the drug of choice for these infections because it is bactericidal, adequate concentrations can be achieved in the circulation and in the diseased tissue, and the drug is inexpensive. Procaine penicillin administered intramuscularly at a dose rate of 15,000 to 20,000 IU/kg (15-20 mg/kg) ql2h is appropriate (Treatment No. 84). If longer term antimicrobial therapy is required, trimethoprim-sulfonamide may be used at 15 mg/kg q12h of the combined agents (Treatment No. 108).
- Signs of depression, inappetence, and retropharyngeal pain may respond favorably after the administration of nonsteroidal anti-inflammatory drugs (see Chapter 18). These should be used only after the initial fever abates, because a decrease in temperature is a useful indicator of response to antimicrobial treatment.
- In cases in which severe airway obstruction is present, a temporary tracheostomy may be indicated.
- Surgical drainage of the abscess cavity may be required in some severe cases, although this is often difficult because of the complex anatomy of this region. Surgical access to the retropharyngeal abscess can be achieved through a similar approach to that used for laryngoplasty. The abscess can be aspirated to yield appropriate bacteriologic samples and then lanced. Drainage can be facilitated by using fenestrated Silastic tubing, through which the abscess cavity also can be lavaged.

Soft Palate Dislocation (Dorsal Displacement of the Soft Palate)

Dorsal displacement of the soft palate (DDSP), which usually occurs during peak exercise, causes a narrowing of the nasopharyngeal airway, creating turbulence on inspiration and expiration. However, it appears that the greatest amount of airflow impedance occurs during expiration. Soft palate

displacement may be intermittent or persistent. The intermittent form is most common in racehorses, causing temporary obstruction of the airway at high speeds. The soft palate normally forms an airtight seal around the larynx, together with the pharyngeal arch, to allow efficient and laminar movement of air down the respiratory tract during exercise. It appears that the soft palate is innervated primarily by the vagus nerve, because local anesthesia of cranial nerve X may result in DDSP. Some authors suggest that the guttural pouches be examinined in cases of DDSP, because inflammation/infection may be noticed in the vicinity of the vagus nerve as it courses along the medial compartment. However, this association needs to be verified.

KEY POINT

If dislocation of the soft palate occurs, it becomes displaced from its normal position under the epiglottis and lies over the opening of the larynx. This results in turbulent and inefficient airflow with a great reduction in performance.

Severely affected horses may become weak and attempt to mouth breathe. Swallowing (repeated attempts may be required) usually results in replacement of the soft palate to its normal position. Although soft palate displacement may be a sequel to a variety of inflammatory diseases of the upper airway and hypoplasia of the epiglottis, in most cases the cause for the displacement is not defined. It may be due to caudal displacement of the larynx, associated with activity of the sternothyrohyoideus and omohyoideus muscles during exercise.

HISTORY AND PRESENTING SIGNS

- Gurgling noise during fast exercise
- Dyspnea during exercise
- Repeated attempts to swallow during exercise
- Usually in racehorses

CLINICAL FINDINGS AND DIAGNOSIS

KEY POINT

Soft palate displacement usually occurs during fast exercise, particularly when the horse is "pulling hard" or "on the bit."

- Trainers, jockeys, and drivers often describe horses as making a gurgling noise or "choking up," and there is a dramatic reduction in performance associated with this.
- Endoscopic examination at rest usually shows no abnormalities.
- Endoscopic examination after high-intensity exercise may reveal soft palate dislocation. However, on many occasions, no abnormalities are detected because the dislocation usually occurs during high-speed exercise and the soft palate is immediately replaced in its normal position.
- Manual occlusion of the nostrils may assist in inducing displacement of the soft palate. Diagnosis is therefore often made by exclusion of other upper respiratory problems, together with the typical history.
- Treadmill endoscopy, with a bit in the horse's mouth and the use of long reins, may result in palate displacement in susceptible horses. Horses need to be exercised for several minutes at higher speeds before the treadmill endoscopic examination, because often the displacement of the soft palate occurs only when the horse shows signs of fatigue.

DIFFERENTIAL DIAGNOSIS

- Idiopathic laryngeal hemiplegia
- Epiglottic entrapment
- Subepiglottic cyst
- Pharyngeal lymphoid hyperplasia
- Sinus/pharyngeal cyst
- Guttural pouch empyema/abscess
- Nasal polyp
- Atheroma
- Congenital palatal defects (e.g., cleft palate)

TREATMENT

KEY POINT

In some cases, application of a tongue tie, figure of 8, or cross-over nose band or use of a straight bit will result in elimination of the problem. Alternatively, rest from exercise may be helpful.

- When these techniques are not successful, a variety of surgical procedures have been described. These include surgical removal of the sternothyrohyoideus and omohyoideus muscles. Myectomy of the sternothyrohyoideus muscle can be performed with the horse standing, and training can be resumed within 12 to 14 days in most cases. Myectomy of both muscles requires more extensive dissection, and general anesthesia is indicated. Tenectomy of the sternothyroid tendon close to its insertion onto the thyroid cartilage can be combined with myectomy. Approximately 50 to 60% of horses can be ex-

pected to show improvement after myectomy (assuming the epiglottis is not hypoplastic). However, the efficacy of the surgery is still not clear.

- In cases in which the myectomy provides little or no relief, a staphylectomy can be performed. This involves resection of a portion of the caudal border of the soft palate. This procedure results in improvement in some horses, although the exact reason for this benefit is unknown. A recent study showed a successful result in approximately 60% of horses that underwent staphylectomy. There is a strong contraindication to this procedure if the epiglottis is hypoplastic, because the operation will only worsen the soft palate displacement. Additional complications associated with this surgery include the potential for tracheal and/or pulmonary aspiration of feed if too much of the soft palate is resected.
- Epiglottic augmentation using polytetrafluoroethylene (Teflon) paste has also been described as a treatment for DDSP and has potential for use in horses with epiglottic hypoplasia.

Subepiglottic Cysts (Pharyngeal Cysts)

The most common location for cysts within the pharynx is in the subepiglottic area, although cysts have been reported near the larynx and soft palate and in other sites within the pharynx. It has been proposed that subepiglottic cysts are the result of a defect in embryologic development and are derived from remnants of the thyroglossal duct. In all cases, cysts usually contain a viscous, yellow-tan, mucus-like material. Cysts are most frequently reported in Standardbred and Thoroughbred racehorses. A congenital form of the disorder occurs in foals.

HISTORY AND PRESENTING SIGNS

- Respiratory stridor during exercise and reduced performance are common.
- Dysphagia may be reported but is rare.
- A chronic cough, dyspnea, and dysphagia may be reported in foals with the disorder.

CLINICAL FINDINGS AND DIAGNOSIS

KEY POINT

The most common clinical signs in older horses (2-5 years old) that are associated with subepiglottic cysts are reduced performance capacity and an inspiratory and expiratory respiratory noise that is often prominent only during exercise.

- The dyspnea becomes more pronounced with increasing exercise intensity.
- Dysphagia and nasal discharge are uncommon manifestations of this disease in adult horses.
- Subepiglottic cysts have been reported infrequently in foals. In this age group, the cysts are often extremely large when diagnosed and produce dyspnea at rest and, possibly, nasal discharge, dysphagia, and an increased potential for aspiration pneumonia.
- Subepiglottic and pharyngeal cysts are diagnosed on endoscopic examination. In adults, the cysts are usually 1 to 5 cm (0.5-2 inches) in diameter and are smooth-walled, lying under or lateral to the epiglottis. During examination of the pharyngeal area, it is important to make the horse swallow, because some cysts may not be visible until this maneuver is accomplished.
- Lateral radiographs of the laryngeal/pharyngeal region also may be of value in defining cystic lesions.
- In foals, the cysts are often much larger at the time of diagnosis and are usually obvious during endoscopic examination. If there is dysphagia, careful examination of the chest should be undertaken for signs indicative of aspiration pneumonia. If there is a suspicion of pneumonia, radiographs of the thorax may be useful.

DIFFERENTIAL DIAGNOSIS

Adults

- Idiopathic laryngeal hemiplegia
- Epiglottic entrapment
- Pharyngeal lymphoid hyperplasia
- Sinus cyst
- Guttural pouch empyema/abscess
- Nasal polyp
- Atheroma
- Epiglottic malformation

Foals

- Congenital deformities of the upper airway
- Disorders resulting in dysphagia and dyspnea (e.g., guttural pouch tympany, retropharyngeal abscessation)
- Viral or bacterial pneumonia

TREATMENT

- Therapy involves surgical removal of the cyst. Currently, several techniques are available.
- One treatment involves a ventral laryngotomy so that direct access to the cyst is achieved. The

cyst is then dissected free from the surrounding tissue. Healing is by second intention, and convalescence takes about 4 to 8 weeks. This is the technique of choice in foals and horses with particularly large lesions.

- The alternate technique involves removal of the cyst via an oral approach using short-term general anesthesia, an oral speculum, and a wire snare. Although the surgeon gains less favorable visual access to the cyst, because of the nature of the lesions, recurrence is rare. This latter procedure has been made more popular by some clinicians because the postoperative convalescence period is greatly reduced, being only 1 to 3 weeks before the horse can return to training. This technique is not suitable for all cases because access to the cyst depends on its size and location and the lumen diameter of the horse's mouth.
- The use of Nd:YAG lasers for extirpation of the cysts via an oral approach also has been reported. The cystic structures can be excised using direct contact or via noncontact coagulation.
- Whichever surgical technique is adopted, care should be taken so as to not resect excessive amounts of subepiglottic tissue, which may result in cicatrical contraction and subsequent DDSP.

Tracheal Stenosis

Also referred to as "scabbard" trachea or dorsoventral flattening of the trachea, tracheal stenosis is a condition that most commonly has a congenital origin. Ponies and miniature horses constitute the breeds most frequently afflicted. In the congenital form of the disease, much of the length of the trachea is involved. Acquired forms of tracheal stenosis may result from direct trauma to the cervical trachea, resulting in a partial narrowing and hence a restriction to airflow.

HISTORY AND PRESENTING SIGNS

- Demonstration of a collapsed trachea is often an incidental finding. However, presenting complaints by owners may include poor performance, dyspnea (during exercise), and chronic paroxysmal coughing, particularly in association with exercise.

CLINICAL FINDINGS AND DIAGNOSIS

KEY POINT

Clinical manifestations of a collapsed trachea are relatively rare, with the diagnosis often being made as an incidental finding at necropsy. In animals in which clinical signs are apparent, stertorous respiration with some degree of respiratory stridor is usually present.

- Chronic, paroxysmal bouts of coughing may be stimulated by tracheal palpation or exercise.
- Some animals show clinical manifestations similar to those seen in horses suffering from chronic obstructive pulmonary disease (COPD).
- Diagnosis can be made by palpation of the cervical trachea, which may reveal a flattening at the site of the abnormality.
- Endoscopic examination of the lumen of the trachea is a valuable diagnostic tool, as are lateral radiographs of the cervical trachea.

DIFFERENTIAL DIAGNOSIS

- COPD
- Retropharyngeal abscessation
- Pneumonia
- Upper airway obstruction (e.g., foreign bodies, neoplasms, sinus cysts)
- Tracheal ring hypoplasia

TREATMENT

- Several reports exist that describe attempts to correct the stenosis. These are difficult, are often associated with complications, and are usually undertaken only by specialist surgeons.
- If the animal is not required to perform athletic endeavors and is not coughing too severely, the best advice may be not to attempt correction of the problem. Retirement of animals to a more sedentary lifestyle may result in palliation of signs.

SINUS PROBLEMS

Sinus Cysts

Sinus cysts are fluid-filled cavities developing principally in the maxillary sinuses. The cause of the cysts is unknown, although most cysts have an epithelial lining. One suggestion is that sinus cysts have a similar pathogenesis to ethmoid hematomas, but this is unlikely. A congenital form has been described in foals.

HISTORY AND PRESENTING SIGNS

- Long-term, clear, unilateral nasal discharge is commonly reported.

- History of progressive facial swelling.
- Dyspnea and exercise intolerance may occur.

CLINICAL FINDINGS AND DIAGNOSIS

- The most common findings on examination are a clear to mucoid nasal discharge, facial swelling, and dyspnea.
- There appear to be two groups of animals affected by sinus cysts: animals less than 1 year old (probably a congenital form) and mature horses (>9-10 years old).
- Despite the propensity of nasal cysts to distort the paranasal sinuses and tooth roots, they rarely invade the nasal cavity.
- Involvement of the frontal sinuses is rare.
- Diagnosis is based on the signalment, history of progressive swelling in the paranasal sinus region, physical examination findings, and the results of diagnostic tests.
- The two most important diagnostic tests are endoscopy and radiography.
- Endoscopic examination of upper airway when the paranasal sinuses are diseased can give the appearance of the sinuses impinging into the airway. This is reflected by the ventral concha being enlarged.
- Radiography of the affected area usually reveals fluid-filled cavities within the sinuses. The multiloculate form of the cysts is most common, although single fluid-filled cavities may be identified in some horses.
- Sclerosis of surrounding bone is common, as is displacement of tooth roots.
- Sinuscentesis may reveal moderately viscous fluid of a flaxen-amber color. There are few cells on cytologic examination and no bacteria.

DIFFERENTIAL DIAGNOSIS

- Primary or secondary sinusitis
- Congenital malformations of the nasal cavity or facial bones other than sinus cysts
- Neoplasia of the paranasal sinuses (e.g., squamous cell carcinoma, fibroma/fibrosarcoma, adenocarcinoma)
- Mucocele (young horses)
- Ethmoid hematoma
- Trauma

TREATMENT

- Extirpation of the sinus cyst is the treatment of choice. An incision and bone flap are used to gain access to the affected sinus.
- The cyst and its lining are dissected free of the surrounding sinus tissues. Considerable hemorrhage may accompany the procedure.
- In many cases, the postoperative course is relatively uncomplicated, with the incidence of recurrence being relatively low.
- Aesthetically favorable facial remodeling after extirpation of the cyst frequently occurs, making the longer-term prognosis more promising than might have been expected on initial diagnosis.

Sinusitis

The maxillary and frontal sinuses have communications with the nasal cavity, and the caudal compartment of the maxillary sinus communicates with the frontal sinus via the frontomaxillary opening. As a result, upper respiratory tract infections can extend into these regions, resulting in primary sinusitis.

KEY POINT

Primary sinusitis occurs most frequently in younger horses, as the incidence of infectious respiratory tract disorders is greater in this age group than in older horses.

In addition, the last four upper cheek teeth extend into the maxillary sinus, so infection of the tooth roots can manifest as a secondary sinusitis (see Tooth-Root Abnormalities and Secondary Sinusitis).

HISTORY AND PRESENTING SIGNS

- Chronic, persistent, unilateral, mucopurulent nasal discharge that increases during exercise is commonly reported.
- Some horses demonstrate dyspnea, particularly in response to exercise.

CLINICAL FINDINGS AND DIAGNOSIS

- Distortion of the facial contours is rare but can be seen in chronic cases.
- Stertorous breathing during exercise may occur.
- Systemic signs (e.g., fever, inappetence, weight loss) occur occasionally, as does epiphora.
- Extension of infection in severe cases of frontal

sinusitis has been associated with meningitis and neurologic dysfunction.
- Diagnosis of primary sinusitis can be supported by a history of upper respiratory tract infection and the clinical signs.
- An oral examination should be performed if secondary sinusitis due to tooth-root problems is suspected.
- Accumulation of fluid in the sinuses may be indicated by a change in resonance during percussion of the sinuses.
- Endoscopy may reveal pus draining from the nasomaxillary opening. This is located in the caudal region of the dorsal meatus.
- Radiography is frequently of great assistance in revealing fluid within the sinus. Chronic cases may show osteolysis and mineralization.
- Sinuscentesis is a valuable tool for confirmation of infection and elucidation of the causative organisms. *S. equi* var. *zooepidemicus* and *S. equi* var. *equi* are implicated most frequently.

TREATMENT

KEY POINT

Systemic antibiotics are rarely successful in resolving the problem. However, in some mild cases remission may occur.

- Penicillin (procaine or aqueous) is the drug of choice for infections due to *S. equi* var. *zooepidemicus* and *S. equi* var. *equi* (Treatment Nos. 83-86).
- More aggressive therapy involves lavage and drainage of the sinus with polyionic solutions (0.5-2 L warmed to near body temperature) infused once or several times per day through a catheter or polyethylene tubing inserted through a trephine hole. Catheters can be sutured in place for repeated lavage. Lavage provides mechanical flushing of the sinus and is often an effective method for removal of the purulent material and debris.
- In more extensive infections of the maxillary sinus, a maxillary bone flap technique may be used to gain access to the entire sinus for removal of mineralized and necrotic tissue. Postoperative care requires lavage and appropriate systemic antibiotics.

Tooth-Root Abnormalities and Secondary Sinusitis

Because the roots of the upper premolar and molar teeth project into the maxillary sinus and are close to the ventral nasal meatus, any disorders involving the tooth roots may lead to narrowing of the nasal cavity, obstruction to airflow, local infections (including pulpitis), and secondary sinusitis. Conditions such as fractures, patent infundibula, chronic ossifying alveolar periostitis, tooth displacement, dental malposition, tumors of the tooth roots, or maleruption can cause such problems. Dental disease is most common in mature horses.

HISTORY AND PRESENTING SIGNS

- Nasal discharge that is often unilateral and malodorous is common.
- Dyspnea may be reported. This occurs secondary to narrowing of the nasal passages.

CLINICAL FINDINGS AND DIAGNOSIS

KEY POINT

Nasal discharge, dyspnea (respiratory stertor), malodorous breath, and rarely sinus tracts to the skin may indicate tooth-root problems, particularly those involving local infections.

- Distortion and swelling over the maxillary region may be seen.
- Dysphagia is rare.
- Thorough oral examination (often requiring sedation and use of a mouth gag), endoscopic examination, and radiography aid diagnosis.
- When performing an oral examination in a horse with suspected tooth-root problems, specific things to be checked for are evidence of cracked teeth, dental malposition, fetid breath, drainage of purulent material from around tooth roots, patent infundibula, or pulpitis.
- Evidence of nasal obstruction may be revealed by passage of a stomach tube.
- Absence of signs on physical examination does not preclude the existence of tooth-root disease.
- Endoscopy may reveal purulent material draining from the ventral compartment of the maxillary sinus into the nasal cavity via the nasomaxillary opening. This is likely if secondary sinusitis exists. Distortions of the nasal passages also may be noted.
- Radiographs often reveal the presence of a tooth-root or apical granuloma. A zone of local osteolysis with a sclerotic margin is common.
- Diagnosis of the obstruction is possible by passage of a stomach tube, which will meet an obstruction at the site of narrowing of the ventral nasal meatus.
- *Micronema delatrix,* a saprophytic nematode found in decaying humus, may invade the nasal passages and paranasal sinuses on occasion and

result in clinical signs similar to those described in this section. There appears to be a high risk of spread of the organisms to other organs, including the lower mandible, brain, and kidneys. Diagnosis is based on the results of biopsy examinations.

DIFFERENTIAL DIAGNOSIS

- Primary sinusitis
- Sinus cysts
- Congenital malformations of the nasal cavity or facial bones
- Trauma
- Foreign body
- Ethmoid hematoma
- Nematode infection (e.g., *M. delatrix)*
- Neoplasia (e.g., fibroma/fibrosarcoma, squamous cell carcinoma, adenocarcinoma)
- Conchal necrosis
- Amyloidosis

TREATMENT

- Most tooth-root problems usually develop slowly, and by the time the horse presents for examination, removal of the diseased tooth and other infected local tissues is the most appropriate course of action. This is a difficult procedure and is most easily performed via a sinus flap. Postoperative care includes antibiotic coverage (see Chapter 18) and local wound therapy. The latter may consist of packing the alveolar socket with gauze. The packing is changed every other day, and the socket is flushed with warm saline or dilute antiseptic. New gauze is wedged back in the socket. Umbilical tape is tied around the pack before insertion, and the loose end of the tape is directed to exit at the skin surface, where it is secured to another gauze roll. This procedure requires a mouth gag, and most horses require sedation. A less risky procedure involves packing the alveolar socket with acrylic at the time of surgery. Securing the acrylic in the socket can provide a challenge. The aim of both procedures is to allow the socket to fill with granulation tissue while preventing the egress of food from the mouth into the socket or sinus. In summary, tooth removal is often time-consuming, and complications occur relatively frequently. However, with time most tooth sockets will heal satisfactorily.
- *M. delatrix* is sensitive to Ivermectin (200 μg/kg orally; Treatment No. 62). However, because this organism can invade other tissues, therapy is often not successful.

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Lower Respiratory Tract Diseases

Bacterial Pneumonia

Bacterial pneumonia is one of the most common diseases affecting the lower respiratory tract in adult horses. Bacterial pneumonia is frequently the result of some stressful event that produces some degree of immunocompromise and is often secondary to viral respiratory disease.

HISTORY AND PRESENTING SIGNS

KEY POINT

History of exposure to stressful situations is common (e.g., transport, anesthesia, training, weaning, congregations of large numbers of horses).

- The horse will usually have a history of signs consistent with a previous viral respiratory tract infection

CLINICAL FINDINGS AND DIAGNOSIS

KEY POINT

Increases in respiratory rate or heart rate, dyspnea, fever, mucopurulent nasal discharge, signs of depression, and inappetence are common features of this disease.

- In racehorses, poor performance (acute onset) may be noted, as are signs of distress after racing or training.
- Auscultation of the thorax may reveal harshness, wheezes, and "gurgling" respiratory sounds, and these may be accentuated by the use of a rebreathing bag.
- Definitive diagnosis is obtained with the use of

transtracheal lavage and aspiration (see previous section on diagnostic aids). This procedure allows for cytologic and bacteriologic analysis of samples collected. Appropriate antibiotic selection can then be made on the basis of culture and sensitivity results.

- BAL also may provide a very useful indication of the cytologic and to a lesser extent bacteriologic (due to the potential for contamination by bacteria resident in the upper airway when the tube is passed) characteristics of the airway in the caudal lung region. However, culture using the Darien catheter system, described in the section on diagnostic aids, is an alternative to the more invasive procedure of transtracheal aspiration.
- The most common aerobic bacterial species involved in the induction of pneumonia in horses are members of the families Lactobacillaceae (gram-positive) and Enterobacteriaceae (gram-negative). The most frequently isolated bacteria from cases of pneumonia include *Streptococcus* spp., *Actinobacillus* spp., *Klebsiella* spp., and *R. equi*.
- Anaerobic bacteria are being implicated with greater frequency as causative agents in bacterial pneumonias in adult horses. As a result, submission of samples for anaerobic culture, particularly in cases with severe clinical signs, may be indicated (see Chapter 16).
- Hematologic studies will give some guide to the severity of the infection in acute cases. Anticipated abnormalities include a leukocytosis, with neutrophilia, and increased serum fibrinogen and globulin concentrations if the disease process has been active for more than a few days.

KEY POINT

Pleuropneumonia and pleural effusion often occur secondarily to pneumonia, and therefore additional physical examinations {including careful auscultation) should be undertaken in horses not showing a favorable clinical response to therapy. Particular consideration should be given to repeating the transtracheal aspiration, ultrasound examinations, and/or thoracocentesis if there is a lack of response to therapy. Repeated measurement of the animal's leukocyte count in refractory cases will provide information pertaining to response to treatment.

DIFFERENTIAL DIAGNOSIS

- Viral respiratory tract infections
- Pleuropneumonia
- Acute respiratory distress

TREATMENT

- Systemic antibiotic therapy, preferably with bactericidal antibiotics at maximum therapeutic dose rates, should be undertaken.
- Selection of antibiotic therapy "in the dark" or use of "shotgun" antibiotic treatment is unlikely to prove effective because it is difficult in many cases to predict the organism involved.
- Therapy can be started with gentamicin (6.6 mg/kg IV q24h; Treatment No. 56), and crystalline penicillin (20,000 IU/kg IV or IM every 6 hours; Treatment Nos. 84 and 85) in horses showing signs consistent with acute bacterial pneumonia.
- Procaine penicillin (15,000-20,000 IU/kg [15-20 mg/kg] IM every 12 hours (Treatment No. 83) is also a good choice to follow initial therapy with crystalline penicillin.

KEY POINT

The procaine in procaine penicillin G may take up to 6 weeks to be eliminated in racehorses after administration. This may cause a problem with the regulatory authorities in some countries.

- Other potentially suitable antibiotics include trimethoprim-sulfonamide (15 mg/kg of combined agent orally q12h; Treatment No. 108) and ceftiofur 2.2-4.4 mg/kg IV or IM q12-24h (Treatment No. 18).
- After the return of bacterial culture and sensitivity results, the appropriate antibiotic selections can be made.
- Other therapies for pneumonia may include bronchodilators, mucolytics, nonsteroidal antiinflammatory drugs, reduction of stress, and good nursing care.
- The most commonly used and effective bronchodilators are aminophylline (4-7 mg/kg orally q24h; Treatment No. 5) and clenbuterol HCl (0.8 μg/kg IV or orally q12h; Treatment No. 27). These agents appear to reduce the respiratory effort required by many horses with acute bacterial pneumonia and improve the clearance of mucopurulent secretions from the lower airways.
- Nonsteroidal anti-inflammatory drugs (see Chapter 18) may be required in horses showing significant debility as a result of the pneumonia to afford them greater comfort and to increase interest in eating and drinking.

KEY POINT

Anti-inflammatory drugs may mask fever and, therefore, progression of the disease. It is

important not to discontinue antimicrobial therapy too soon or return the horse to work too rapidly if the horse is receiving nonsteroidal anti-inflammatory drugs.

- Withdrawal of the affected animal from a training program during periods when there is clinical evidence for the existence of pneumonia is also vital.
- The ongoing stress of training in the face of bacterial pneumonia is one of the most frequent causes of severe complications such as pleuropneumonia.
- Provision of a high-quality, palatable, digestible diet is important in animals that are debilitated by the systemic effects of bacterial pneumonia.
- Appropriate attention to the fluid and electrolyte needs of the animal is also critical in the successful management of horses with pneumonia.

Chronic Obstructive Pulmonary Disease ("Heaves")

COPD is a condition found mainly in older horses that are kept in box stalls. It is the result of an allergic bronchitis and bronchiolitis from exposure to various allergens including dust in straw and hay. Various molds (e.g., *Aspergillus* spp., *Micropoly spora* spp., and *Faenia* spp.) and endotoxin associated with bedding and feedstuffs have also been implicated in the etiopathogenesis of COPD.

KEY POINT

The condition is common in the northern hemisphere because horses are often kept in barns, many of which have poor ventilation, for prolonged periods.

Because of different housing and management conditions, COPD is rare in the southern hemisphere.

HISTORY AND PRESENTING SIGNS

KEY POINT

Chronic cough, dyspnea, and exercise intolerance in older horses are the most common complaints by owners.

CLINICAL FINDINGS AND DIAGNOSIS

- The most common signs are increased respiratory effort and dyspnea after strenuous exercise and a soft cough, particularly in association with feeding and exercise.
- Clinical examination may reveal a biphasic expiratory effort, and in long-standing cases, there will be a "heave line" along the ventral rib cage.
- In addition to decreased exercise tolerance, horses with COPD may exhibit dyspnea and coughing during exercise.
- Auscultation of the chest reveals wheezing sounds that are most easily heard if a rebreathing bag is used.
- Blood gas analysis will reveal low oxygen partial pressures in arterial blood, with values usually less than 80 mm Hg at rest in horses living near sea level. Some cases will have an increase in arterial carbon dioxide tensions.
- Endoscopy of the respiratory tract may reveal increased amounts of mucopus within the trachea and larger bronchi.
- Clinically affected horses suffer primarily from disease of the small bronchioles and alveoli. Because the offending allergenic mold spores/dust particles are small enough to pass into the lower airways, it has been suggested that BAL can provide a more consistent cytologic profile of airway pathology in horses with COPD than that obtained with transtracheal aspiration. BAL fluid usually will reveal marked neutrophilic inflammation, up to 50 to 70% of the total cell count in advanced cases. Horses with COPD rarely exhibit eosinophilia in BAL fluid, and if present, this finding may indicate other diseases such as eosinophilic pneumonitis or parasitism.
- Hematologic and serum or plasma biochemistry values are usually within the normal ranges.

DIFFERENTIAL DIAGNOSIS

- Inflammatory airway disease
- Viral respiratory disease
- Bacterial pneumonia
- Pleuropneumonia
- Interstitial pneumonia
- Eosinophilic pneumonia
- Other causes of dyspnea/respiratory noise during exercise (e.g., idiopathic laryngeal hemiplegia, epiglottic dislocation, pharyngeal cysts)

TREATMENT

- This is a management-related disease; therefore, the main aspect of treatment is to remove the predisposing cause, that is, remove the horse from the stable (if possible) and turn it out to pasture. Most horses respond relatively rapidly to rest while housed outdoors. Recurrence is common when they are returned to the stable environment. Reduction of dust in bedding and

feed (wetting down food before offering it to the horse or use of pelleted rations and alfalfa cubes) may assist in reducing the severity of signs.

- Bronchodilator therapy using clenbuterol (Treatment No. 27) is often helpful in alleviating signs, although long-term treatment may be required. Alternative methods of administering β_2 agonists include aerosolization using specially designed face masks (Equine Aero-mask) and handheld metered dose inhalers that have been adapted to use with conventional human "asthma puffers." Drugs administered via these means include pirbuterol and albuterol at dose rates of 1 to 2 μg/kg and fenoterol at 2 to 4 μg/kg. Although these drugs can alleviate clinical signs in horses suffering from COPD, their duration of action is often short lived. Other bronchodilator drugs include aminophylline and theophylline; however, they may cause excitation in treated animals at doses required to induce bronchodilation. A commonly used human bronchodilator is ipratropium bromide, which when administered via a nebulizer at a dose of 2.0 to 3.0 |xg/kg produces short-lived bronchodilation. The use of mast cell-stabilizing drugs such as sodium cromoglycolate has also been documented in prevention of COPD signs when administered before allergen exposure at dose rates of 80 mg via a nebulizer.
- Corticosteroids given by mouth (prednisolone; Treatment No. 93) may also provide good relief of clinical signs. Oral administration of corticosteroids is still one of the most commonly used treatment methods, although aerosolized beclomethasone has also been shown to improve lung function in affected horses. Successful local inhalation of such agents may avoid some of the potential side effects associated with systemic administration of bronchodilators and corticosteroids.
- Care should be taken when using these drugs in racing animals, as all agents have withholding periods before racing.

Summer Pasture-Associated Obstructive Pulmonary Disease

A form of COPD exists in the southern states of the United States and in Europe in which allergens in hay and pastures during summer months produce signs similar to those in stabled horses. In these cases, the treatment is to remove the horses from pasture into well-ventilated stables. Concomitant bronchodilator and/or corticosteroid therapy may also be warranted in selected cases.

Exercise-Induced Pulmonary Hemorrhage

Exercise-induced pulmonary hemorrhage (EIPH) is a common disorder, particularly in Thoroughbred and Quarter horse racehorses. It is likely that 100% of racehorses have some degree of pulmonary hemorrhage during high-intensity exercise. Standardbreds probably have a lower incidence, and EIPH is rare in endurance horses.

KEY POINT

From these findings it is apparent that the incidence of EIPH is directly related to the absolute intensity of exercise undertaken.

Hemorrhage originates in the lungs, probably in the thoracophrenic region. This origin for the hemorrhage is supported by necropsy examinations of horses with a known history of EIPH. Although many hypotheses (at least five are currently under discussion) as to the pathogenesis of EIPH have been proposed, no clear mechanism has been defined. Whatever the exact pathogenesis, it is highly likely that EIPH occurs as a result of the high cardiac outputs and vascular pressures required to enable metabolic activity at maximal exercise intensities. To date, the most popular theory is one of stress failure of pulmonary capillaries resulting from abnormal intravascular and extravascular pressure changes that occur during exercise.

HISTORY AND PRESENTING SIGNS

- Horses with EIPH frequently are reported to perform poorly. The effects of EIPH on performance are not clear.
- Respiratory distress and an increased rate of swallowing after exercise can occur. The swallowing is presumably to clear blood ascending from the lower airway.
- On rare occasions, blood is discharged from the nostrils.

CLINICAL FINDINGS AND DIAGNOSIS

- The predominant clinical manifestation of EIPH is blood in the ventral part of the tracheobronchial tree, visible via endoscopy. Only about 1% to 10% of horses with EIPH ever demonstrate epistaxis.
- Coughing also may occur, but this is a nonspecific sign associated with many conditions of the upper and lower respiratory tracts. In severe cases there may be noticeable alterations in the breathing pattern during the postexercise period.
- Diagnosis is usually based on the history and

endoscopic findings. Blood in the trachea will be most obvious 30 to 60 minutes after strenuous exercise.
- Other procedures that may assist in the diagnosis of EIPH are transtracheal lavage and aspiration or BAL to demonstrate the presence of hemosiderophages.
- Hemosiderophages clear slowly from the lungs, and their recovery in respiratory secretions can be anticipated for prolonged periods (more than 1 year) after the hemorrhagic episode.
- Special stains (e.g., Sano's trichome) may increase the likelihood of visualizing hemosiderophages on appropriate smears. Increases in the proportion of neutrophils and eosinophils in lavage fluid also occur in horses with a history of EIPH. Increases in the bronchointerstitial pattern in chest radiographs, particularly in the caudodorsal lung fields, may be visualized in horses with EIPH. However, this finding is not consistent in all horses with EIPH.

DIFFERENTIAL DIAGNOSIS

- Other causes of dyspnea/respiratory embarrassment during exercise (e.g., idiopathic laryngeal hemiplegia, epiglottic dislocation, pharyngeal cysts)
- Viral respiratory disease
- Pharyngeal lymphoid hyperplasia
- Other causes of epistaxis (e.g., guttural pouch disease, nasal tumors, ethmoid hematoma)
- COPD

TREATMENT

- Veterinarians and horse people are particularly inventive individuals. As a result, a plethora of remedies for EIPH have been described. Unfortunately, few if any of these provide good palliation of the condition.

KEY POINT

Furosemide (0.3-0.6 mg/kg IV 3-4 hours before racing; Treatment Nos. 54 and 55) is widely prescribed in the United States, and in approximately 50% of horses, the drug is administered before racing.

- Based on the observations of skilled horse people, there seems little doubt (at least on the basis of a large volume of empirical evidence) that this medication reduces the severity of EIPH.
- Furosemide does not eliminate hemorrhage; it just reduces the severity. The mechanism by which the agent exerts these effects is not clear.

Fungal Pneumonia

Fungal elements are ubiquitous in the environment, and it is common to discover fungal hyphae in pulmonary tract secretions of normal horses as an incidental finding. Primary fungal disease of the respiratory tract is rare. However, infection may occur secondarily to an underlying disease process, resulting in compromise of host immune defense mechanisms. Pathogenic fungi include *Coccidioides* spp. and *Histoplasma* spp., whereas opportunistic fungal pathogens include *Crytptococcus* spp., *Aspergillus* spp., *Candida* spp., *Mucor* spp., and *Rhizopus* spp. Neoplasia; myeloproliferative disorders or diseases resulting in endotoxemia, septicemia, or laminitis; and prolonged antibiotic or corticosteroid therapy may predispose to secondary fungal infections.

PRESENTING SIGNS AND CLINICAL FINDINGS

- Progressive respiratory disease and failure to respond to conventional therapeutic measures should alert the clinician to the possibility of fungal respiratory disease. Concurrent serosanguineous/hemorrhagic nasal discharge and nasal and/or peripheral ulcerative granuloma formation may also be a feature of disseminated fungal disease.
- Endoscopic examination of the respiratory tract, transtracheal aspiration, BAL, thoracic radiography, and percutaneous lung biopsy may be required to achieve a diagnosis.

TREATMENT

- Treatment of fungal pneumonias carries a poor to guarded prognosis for success. Various antifungal drugs are available; however, expense and potential side effects limit their use. Examples include ketaconazole, itraconazole, amphoteracin B, and fluconazole.

Inflammatory Airway Disease

Perhaps better referred to as a syndrome rather than a disease process, inflammatory airway disease represents a persistent inflammation of the lower respiratory tract or small airways. It has been suggested that up to 50% of racing Thoroughbred and Standardbred horses are affected by this condition.

HISTORY AND PRESENTING SIGNS

- Horses will often be presented with a history of poor performance, chronic cough, and in some

cases suspected previous respiratory viral infection.

KEY POINT

Affected horses are generally younger than those suffering from COPD, the clinical signs are generally milder and may be subclinical in nature or manifest during more intense exercise, and the severity of neutrophilia in BAL fluid is not as great as noted in horses with COPD.

DIAGNOSIS AND CLINICAL FINDINGS

- Endoscopic examination of afflicted horses may reveal mucopurulent exudate involving the nasal passages, pharynx, and trachea. Reduction in arterial partial pressure of oxygen during exercise is common.
- Diagnosis is confirmed based on results of BAL, with most horses having a cytologic profile representative of inflammation consisting of lymphocytosis, monocytosis, and mild neutrophilia. Some horses have a predominace of mast cells or eosinophils.
- Possible etiologic factors include persistent respiratory viral infection, inhalation of particulate matter or irritants (e.g., ozone), persistent *Streptococcus pneumoniae* infection, pulmonary stress associated with exercise, EIPH, poor stable ventilation, and hypersensitivity reactions.

DIFFERENTIAL DIAGNOSIS

- COPD
- Viral respiratory infections
- Eosinophilic pneumonitis
- Lungworm

TREATMENT

- Therapeutic measures depend to some extent on the history, individual case, and results of BAL. Horses with a mixed cytologic profile on BAL have responded favorably to interferon therapy. Natural human interferon alpha administered by mouth to horses suffering from inflammatory airway disease at a dose rate of 0.1 μg/kg body weight daily for 5 days resulted in reduction of respiratory exudate, decrease in total BAL cell counts, and reversion of the differential BAL cell count to a noninflammatory profile. Noncellular indicators of airway inflammation including total protein, albumin, IgG, and IgA and procoagulant activity of airway secretions were also reduced in horses treated with interferon. The mechanism of action of interferon in equine respiratory disease is thought to be related to its immunomodulatory and antiviral effects. However, this is yet to be substantiated.
- More conventional therapeutic agents including bronchodilators, mast cell stabilizers, and corticosteroids may afford some relief in cases in which BAL results show a predominence of eosinophils or mast cells, suggesting the existence of an underlying allergic component to the disorder. The dose regimens are similar to those used in the treatment of COPD. In general, 6 to 8 weeks of medication and discontinuation of exercise will yield favorable results, although response to therapy may be slower in horses exhibiting eosinophilia in BAL fluid.

Interstitial Pneumonia

Interstitial pneumonia in horses is a relatively rare condition caused by infectious and toxic agents and immune-mediated processes. Specific etiologic agents include viruses (e.g., Equine Morbillivirus [EMV], or Hendra virus), bacteria (e.g., *R. equi),* protozoa (e.g., *Pneumocystis carinii),* toxic plants (e.g., crofton weed), inhaled chemicals (e.g., smoke, pesticides), silicosis, and hypersensitivity reactions (type III and IV reactions). Often the disease process is chronic, and in many cases a definitive diagnosis is not obtained.

HISTORY AND PRESENTING SIGNS

- Interstitial pneumonia has been reported in foals and adults. Often, affected horses will have a history of chronic weight loss, exercise intolerance, and respiratory distress unresponsive to antimicrobial and anti-inflammatory therapy.

CLINICAL FINDINGS AND DIAGNOSIS

- Affected horses will usually have a chronic cough, nasal discharge, tachypnea, tachycardia, fever, and often severe respiratory distress.
- Laboratory evaluation may show a neutrophilic leukocytosis, hyperfibrinogenemia, and hypoxemia. Thoracic auscultation may reveal harsh lung sounds with crackles and wheezes. Endoscopic evaluation of the respiratory tract may reveal increased amounts of mucopus in the larger airways.
- Analysis of tracheobronchial secretions may show increased numbers of neutrophils and macrophages.
- Thoracic radiography is more useful in foals; nonetheless, it may reveal a classic interstitial pattern in chronically affected adult horses.

- Transthoracic percutaneous lung biopsy will often provide a diagnosis of interstitial pneumonia yet rarely allows identification of the etiologic agent.
- Histopathologic findings include alveolar necrosis and inflammation with progressive thickening of the interlobular septa and pulmonary fibrosis.

DIFFERENTIAL DIAGNOSIS

- COPD
- Pneumonia/pleuropneumonia
- Airway obstruction
- Congestive heart failure

TREATMENT

- In most instances, treatment is difficult and often unrewarding, largely owing to an inability to definitively determine the etiologic agent in most cases. If a primary agent can be identified, then treatment regimens should be specific in their nature. Most treatment regimens include appropriate anti-inflammatory therapy consisting of nonsteroidal drugs or corticosteroids. Antimicrobial therapy may be indicated to treat primary agents or severe secondary opportunist invaders. Oxygen therapy may be required in some horses. Bronchodilators, antihistamines, and some of the newer immunomodulating drugs may be useful. Congestive heart failure may be a secondary complicating factor associated with pulmonary hypertension. Horses suffering from interstitial pneumonia often have a poor prognosis for recovery because of the progressive nature of the disease.

Lungworm

Dictyocaulus arnfeldi, or lungworm, may be a cause of chronic coughing in the horse. The donkey and donkey crosses (mule and ass) are the natural hosts for lungworm. Natural hosts show few, if any, clinical signs of infestation. The condition in horses is usually found in those that have been in the company of donkeys, mules, or asses. Clinical signs become apparent in horses in the fall or winter after exposure to primary hosts in the summer months. Infective larvae are sensitive to low ambient temperatures, and the cold temperatures common to the frost belt of North America and northern England and Europe kill many of the infective larvae on pasture. As a result, new infections and clinical signs in horses are often noticed after the warmer months of the year.

KEY POINT

Patent lungworm infections are quite rare in horses.

HISTORY AND PRESENTING SIGNS

- Chronic cough, particularly in the late summer, fall or early winter
- A history of the horse living with donkeys

CLINICAL FINDINGS AND DIAGNOSIS

- Coughing, often for several weeks, and possibly signs referable to a condition producing lower respiratory tract obstruction in the late summer or fall are the most common indicators of clinically significant disease.
- Because patent infections are rare, examination of feces for eggs by the Baermann flotation procedure may be unrewarding. However, use of this technique in donkey or donkey crosses that have been in contact with the horse suspected of having the disease may provide evidence of patent infection. This may be sufficient evidence to strongly suspect *D. arnfeldi* infestation in the horse in question.
- Transtracheal aspiration can be helpful in diagnosis in some cases in which a large number of eosinophils is observed in smears of the aspirate. On occasion, transtracheal aspirate samples may contain adult worms, larvae, or eggs.

DIFFERENTIAL DIAGNOSIS

- COPD
- Viral respiratory infection
- Bacterial pneumonia

TREATMENT

- Ivermectin at a dose rate of 200 μg/kg orally (Treatment No. 62) or moxidectin at a dose rate of 0.4 mg/kg orally (Treatment No. 78) appear to be the drugs of choice for treatment of lungworm infection in horses.

Pleuropneumonia (Infectious Pleural Effusion, Pleuritis)

KEY POINT

Pleuritis or pleural effusion is a condition that results in the production of large volumes of fluid in the thoracic cavity, usually secondary to a bacterial pneumonia.

Stress (e.g., transport, racing, surgery) is often an integral part of the history of horses with

pleuritis/pleuropneumonia. A less common cause of infectious pleural effusion is blunt trauma to the thoracic cavity (with or without foreign-body penetration). In many cases in which fluid is present in the chest, careful clinical evaluation is required to determine its presence. Aggressive therapy is required to limit the progression of this serious disease and its complications, which can include laminitis.

HISTORY AND PRESENTING SIGNS

- Signs of depression, inappetence, and a prior history of stress (e.g., transport) is common.
- A history of what appeared to be a viral respiratory tract infection is common.
- Reluctance to move, sweating, and apparent anxiety may be reported.
- In chronic cases, weight loss and ventral edema may be noted.

CLINICAL FINDINGS AND DIAGNOSIS

- Horses may have presenting signs similar to those described for pneumonia.
- At rest, a variable degree of dyspnea may be present, and the respirations are often shallow and rapid.
- Respiratory excursions often appear to be painful, and affected horses may "grunt" when required to move.
- The gait is often stiff owing to pain (pleurodynia), because the parietal pleura are well endowed with pain-sensing fibers. Care must be exercised not to mistake these signs as being indicative of laminitis, myopathy, or colic.
- There is usually an elevated heart rate and increased rectal temperature.
- A soft cough, mucopurulent nasal discharge, and fetid breath are common.
- On auscultation of the chest, there may be abnormal lung sounds in the dorsal regions, such as wheezes, crackles, rales, and harshness, with muffled or absent sounds in the ventral regions.
- Auscultation with a rebreathing bag often is useful in helping define abnormal lung sounds but should not be used in more severe cases, as the rebreathing can produce severe pain. Pleural friction rubs may be detectable. In addition to auscultation, percussion of the chest can be a helpful and inexpensive means of establishing the presence of pleural effusion. Other, more sophisticated techniques include thoracic ultrasonography (the technique of choice) and radiography.

KEY POINT

In any horse suspected of having a pleural effusion, thoracocentesis always should he performed (on both sides of the chest). Ultrasound examination will greatly assist in appropriate placement of the needle or cannula. Cytologic and bacteriologic examination of this fluid is imperative.

- If pleuritis and pleural effusion have been present for more than 1 to 2 weeks, there may be evidence of weight loss, ventral edema (particularly in the pectoral region), and a history of intermittent pyrexia.

KEY POINT

Inappetence is not necessarily a common feature in horses with pleuropneumonia.

- Analysis of peripheral blood samples usually reveals anemia, leukocytosis, neutrophilia, profound hyperfibrinogenemia, and hypoalbuminemia.

DIFFERENTIAL DIAGNOSIS

- Viral or bacterial pneumonia
- COPD
- Thoracic neoplasia with effusion
- Conditions causing signs of generalized pain (e.g., laminitis, colic, myopathy)
- Thoracic foreign body
- Severe EIPH
- Pulmonary edema
- Cardiac failure (e.g., pericardial disease, myocarditis)

TREATMENT

- Thoracic drainage to remove fluid and necrotic material from the chest is usually necessary, particularly if more than a few liters of fluid are present in the thoracic cavity. This may be done with a catheter, teat cannula, or small-gauge chest tube if the volume of fluid is small and the fluid contains little fibrinous material.
- When there is a large volume of flocculent fluid, it should be drained with a relatively large-bore chest tube (20-28F blunt-tipped chest tube).

KEY POINT

Fluid is allowed to drain via gravity (suction is contraindicated because it aspirates flocculent material into the tube, thereby blocking the lumen). Tubes may be fixed in place with a pursestring suture, and a one-

way valve can be made from a latex condom with the tip cut off that is taped to the end of the tube.

- If the volume of fluid is not thought to be too great, intermittent drainage every day or every other day may be considered.
- Parenteral antibiotics are essential for the effective treatment of these horses. It is important that a bactericidal rather than a bacteriostatic antibiotic be used (see Pneumonia).

KEY POINT

From a bacteriologic point of view, pleuropneumonia does not usually constitute a medical emergency.

- As a result, waiting 1 to 2 hours until appropriate samples for bacterial culture and sensitivity are obtained is a far superior practice than commencing "shotgun" antibiotic therapy before collection of appropriate samples for bacteriologic analysis.
- Once samples have been collected (ideally this will include fluid from both thoracic cavities and from a transtracheal aspirate), empirical antimicrobial therapy should be commenced and continued until culture and sensitivity results are available. Penicillin and aminoglycoside antibiotics (see Chapter 18) are usually the drugs of choice in pleuropneumonia because they are active against most organisms encountered in this disease, are bactericidal, provide good plasma and tissue concentrations, have a reasonably broad therapeutic index, and are relatively cost-effective. Ceftiofur (Treatment No. 18), *2.2-4.4* mg/kg q12h IM or IV, also is a good choice.
- Because many cases will have anaerobic bacteria involved, the use of metronidazole (10-15 mg/kg orally q6h; Treatment No. 75) should be strongly considered if the culture results indicate penicillin-resistant *B. fragilis* (see Chapter 16). The most commonly isolated anaerobic organisms are *Bacteroides* spp. and *Clostridium* spp. Mixed aerobic and anaerobic infections are common, and the presence of anaerobes does not necessarily indicate a poorer prognosis for survival.
- Anti-inflammatory therapy may be considered in an attempt to limit the degree of debility inflicted by the disease. A number of agents are readily available. Examples include flunixin meglumine (Treatment No. 52), phenylbutazone (Treatment No. 89), and ketoprofen (Treatment No. 66). Care should be exercised in horses with associated dehydration because the nephrotoxic potential of these agents is increased. It must also be noted that anti-inflammatory drugs may mask fever, thereby decreasing the clinician's ability to monitor progress.
- In cases in which significant systemic manifestations of the disease exist (e.g., toxemia, dehydration), fluid therapy is also indicated (see Chapter 18). The degree of volume contraction is reflected by physical findings (i.e., skin turgor, mucous membrane color, and capillary refill time) and appropriate clinicopathologic measurements (e.g., packed cell volume [PCV], total plasma protein [TPP], urea nitrogen, creatinine).
- After initial replacement of deficits, oral fluid supplementation will usually cover ongoing losses.
- Good nursing care is vital in the management of pleuropneumonia. This includes limitation of stress, provision of a highly palatable and digestible diet, and constant surveillance for such complications as lung abscessation, anterior thoracic masses/abscesses, pulmonary infarction, bronchopleural fistulas, pericardial effusion, pneumothorax, and laminitis.
- In acute cases with limited effusion, the prognosis for full recovery is likely to be reasonable if appropriate therapy is undertaken. Survival rates of horses suffering from pleuropneumonia range from approximately 60 to >90%, after appropriate aggressive treatment. However, in long-standing or complicated cases that have significant sequelae, the degree of damage to the contents of the thoracic cavity is often quite severe. Although salvage in many cases is possible if aggressive therapy is maintained for appropriate periods, prognosis for return to a successful athletic career must remain more guarded.
- The techniques for standing thoracotomy/rib resection have been described and advocated as a treatment for chronic cases of pleuropneumonia that have proven unresponsive to antimicrobial therapy and pleural drainage. Open thoracic drainage can also be attempted in cases of pulmonary abscessation and bronchopleural fistulas.
- Thoracic ultrasonography should be an integral part of the management of horses with pleuropneumonia. It is a readily accessible noninvasive diagnostic tool that can provide valuable information, allowing assessment of response to treatment and development of sequelae.

Rhodococcus equi Infection ("Rattles")

This is a debilitating disease, with predominantly respiratory signs, of foals. There are two forms of

pneumonia: *subacute,* with diffuse miliary pyogranulomatous pneumonia that usually has a fatal outcome, and *chronic,* in which foals have pneumonia and are unthrifty for relatively protracted periods. The disorder is much more prevalent in foals kept in large populations. Morbidity worldwide is about 10%, with mortality rates greater than 50%. Because this disease is restricted almost entirely to foals 1 to 6 months of age, the disease process is discussed in greater detail in Chapter 9.

Streptococcus equi var. *equi* Infection ("Strangles")

"Strangles" is a highly infectious bacterial respiratory disease in horses caused by *S. equi* var. *equi.* Infection is by inhalation or ingestion of the organism with subsequent localization in the mandibular and pharyngeal lymph nodes. The disease is most common in young horses (1-5 years old). The organism is spread in nasal discharges or by contaminated grooming utensils, rugs, feed bins/utensils, or humans (e.g., on hands, clothes, stomach tubes). Animals incubating or recovering from the disease are the usual source of introduction of the organism to a naive population. Although the reason for sudden outbreaks of the disease is often unknown, it is believed that horses chronically shedding the organism are rare. However, recent research has indicated that chronic carriers of the organism are possible and these carriers may be responsible for some outbreaks of the problem. Periodic outbreaks of strangles, characterized by a high incidence in young horses, occur on stud farms and race training complexes.

The incubation period is 3 to 10 days, with morbidity being variable depending on the age, immune status, and management of the herd. At times, morbidity may approach 100% in some susceptible populations. Mortality in well-managed outbreaks is usually less than 5% and often related to secondary pneumonia and upper respiratory tract obstruction.

HISTORY AND PRESENTING SIGNS

- Acute onset of signs of depression, inappetence, and nasal discharge, often in a number of younger horses in a population.
- Outbreaks may occur.
- In more advanced cases, swelling in the submandibular region may be noted.

KEY POINT

Submandibular swelling together with inappetence is often the first sign of the disease noticed by the owner.

CLINICAL FINDINGS AND DIAGNOSIS

KEY POINT

Initial clinical signs in addition to inappetence, signs of depression, and serous nasal discharge include fever (>40°C), pain, and swelling in the pharyngeal region.

- Dysphagia or dyspnea may occur as a result of swelling in the "throatlatch" (hence the name "strangles"). Coughing is a feature in some cases.
- Clinical signs may persist for days to months. In most cases, once affected nodes have abscessed and drained, recovery is uneventful.
- Major clinicopathologic changes may include evidence of dehydration in the early phases of the disease, leukocytosis, and an increase in serum fibrinogen concentration.
- Abscessation in a variety of body sites is possible, including the periorbital area, retropharyngeal lymph nodes, guttural pouches, lungs/thorax, abdominal cavity (e.g., mesentery, liver, spleen, and kidney), brain, and joints. These metastases are referred to as "bastard strangles."
- Horses with metastatic abscesses often show chronic weight loss.
- Affected lactating mares may have significant reductions in milk production.
- Purpura hemorrhagica is a relatively rare but potentially fatal complication. Signs include fever, peripheral edema, signs of depression, and possibly petechial hemorrhages evident on the mucosae.

KEY POINT

Diagnosis is best made on the basis of clinical signs (e.g., signs of depression, fever, lymphadenopathy) and isolation of S. equi *var.* equi. *Swabs from the nose, purulent discharges, and direct aspiration of material from abscesses that have not yet discharged (the optimal site for collection of samples) are suitable for isolation of the organism.*

- Diagnostic success is increased if samples from a variety of sites are cultured (e.g., swabs from draining lymph nodes and the nasal or pharyngeal mucosa). Use of an appropriate transport medium (Streptswab, Medical Wire and Equipment Co., Cleveland, OH) improves the recovery rate of organisms from samples, particularly those that are likely to be delayed in transit to the laboratory. Recent use of a polymerase chain reaction method enables more rapid diagnosis

(reportedly within 6 hours). However, at present this technique is only available in the United States.

- Bastard strangles may be suspected on the basis of physical examination findings (e.g., rectal examination, endoscopy) and the use of other diagnostic aids (e.g., radiography, ultrasonography, abdominal or thoracic paracentesis, joint-fluid analysis). Anemia of chronic disease, persistent leukocytosis, and hyperfibrinogenemia are common features of this disease.

DIFFERENTIAL DIAGNOSIS

- Viral respiratory tract disease
- Bacterial pneumonia
- Guttural pouch empyema
- Abscessation due to bacteria other than *S. equi*

TREATMENT

KEY POINT

Affected horses should be quarantined to prevent exposure to naive horses. Shedding of the organism is likely to be greatest for 2 to 3 weeks after the onset of clinical signs.

- The presence of clinical signs may have no relationship to the potential a horse has for spreading the disease. Previously affected horses (approximately 20%) may still spread the disease for 1 to 2 months after disappearance of clinical signs. In a small percentage of cases, shedding of the organism may continue for 12 to more than 36 months. It is likely that the bacteria reside within the guttural pouches so repeated guttural pouch washes in conjunction with nasal swabs may increase the chance of detecting a carrier animal. As a general protocol, horses should remain isolated for 4 to 6 weeks after disappearance of signs. There have been reports of the bacteria surviving on fomites for approximately 40 to 60 days, depending on environmental conditions.
- Negative results from three culture swabs taken at 7- to 10-day intervals are recommended before a horse is assumed to have cleared the infection. This seems to be true for approximately 85% of recovered horses.
- Endoscopic examination of the nasopharynx and guttural pouches should be considered in horses suspected of being chronic carriers of the organism.
- Personal hygiene is important for those handling horses. For example, handlers of infected horses should not, if at all possible, be in contact with uninfected horses. Scrupulous hand and boot washing and the use of disposable overclothing are recommended. Disinfection of food and grooming materials and reusable veterinary equipment is imperative.

KEY POINT

Penicillin G is the drug of choice.

- Appropriate doses must be used (15,000-20,000 IU/kg [15-20 mg/kg] procaine penicillin IM q12h or crystalline penicillin 20,000 IU/kg IV q6h; Treatment Nos. 83-86). Administration of the agent must be continued for 5 to 7 days after clinical signs have resolved. Failure to do so may result in recrudescence of the disease. Other drugs that have been used are the semisynthetic penicillins (ampicillin, amoxycillin), ceftiofur, trimethoprim-sulfonamide combinations, and oxytetracycline. All have activity against *S. equi* but are much less effective and more costly than penicillin G.
- Routine monitoring of animals at risk by measurement of rectal temperature (once or twice a day) and prompt administration of penicillin to those showing evidence of fever appear to reduce the severity of disease.
- Most mild cases of strangles probably do not require antibiotic therapy and resolve without incident.
- In some cases, drainage of purulent material from localized lymph node infections may be required. Exudate should be disposed of appropriately.
- Bastard strangles often requires protracted antimicrobial therapy (4-6 weeks) and specific local therapy (e.g., abscess drainage) and possibly anti-inflammatory therapy.
- Purpura hemorrhagica requires aggressive long-term antibiotic and anti-inflammatory therapy. Initial treatment with penicillin (Treatment Nos. 83-86) and dexamethasone (0.06-0.1 mg/kg q24h; Treatment Nos. 29 and 30) is indicated. Reduction of the steroid dose as soon as possible after clinical improvement occurs is advised. Dexamethasone appears to be more effective than prednisolone. Phenylbutazone (Treatment No. 89) or flunixin meglumine (Treatment No. 52) also can be useful.

PROPHYLAXIS

- The chance of prevention of strangles is improved if stringent quarantine measures are used. Newly introduced horses should be isolated for 2 to 3 weeks and have regular re-

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cordings made of their rectal temperatures. Repeated nasopharyngeal/guttural pouch cultures can be attempted to identify likely carriers.

There is a report in which the morbidity due to strangles in foals in the face of an outbreak was decreased when benzathine penicillin was administered (5000 IU/kg IM q48h) for 3 weeks (Treatment No. 83). The use of prophylactic antibiotics is questionable because it may limit development of protective immunity such that infection could still occur after discontinuation of antimicrobial therapy.

Several vaccines are available. One vaccine contains the capsular M-protein to *S. equi,* whereas the other contains purified enzyme extract of *S. equi.*

Vaccination has been shown to reduce morbidity and severity of clinical signs but *does not* prevent the disease. Best immunity appears to be afforded by a course of vaccinations, although serum antibody concentrations will often be minimal within 6 months of vacination. This has led to the suggestion of twice-yearly booster administration versus annual vaccination.

It is apparent that immunity after natural exposure is conferred by mucosal IgG, IgA, and serum IgG. The most important antigenic stimulus is likely to be the M-protein. Protective secretory IgG and IgA are produced in milk from mares recovered from the disease and will apparently afford protection to foals. Although intramuscular vaccination with either a bacterin or M-protein extract will stimulate serum antibodies, it is apparently a poor stimulus for mucosal antibody, which appears to be more important in preventing infection.

Swelling at the site of injection and some mild systemic signs (e.g., depression, inappetence) are relatively common following vaccination.

Thoracic Neoplasia

Neoplasia in the thoracic cavity is rare in the horse. Most commonly, thoracic neoplasia involves metastasis from another site. Reported neoplasms include squamous cell carcinoma, lymphosarcoma, adenocarcinoma, and hemangiosarcoma.

HISTORY AND PRESENTING SIGNS

- Weight loss is commonly reported.
- Older horses are often affected.
- Respiratory distress, particularly in response to exercise, may be a feature.

CLINICAL FINDINGS AND DIAGNOSIS

KEY POINT

Affected horses may have evidence of weight loss, dyspnea, tachypnea, cough, nasal discharge, and epistaxis.

- Diagnosis is based on clinical signs and the results of specific diagnostic tests, including endoscopy, radiography, thoracocentesis, and possibly biopsy. Examination of other body systems may provide evidence of generalized systemic involvement of the neoplasm. Results of blood analyses frequently show nonspecific evidence of disease (e.g., anemia of chronic disease).
- Often, diagnosis is not confirmed until a postmortem examination is performed. Discovery of a thoracic neoplastic process may be an incidental finding during a routine necropsy.

TREATMENT

- In general, thoracic neoplasia is untreatable.

Viral Respiratory Disease

Viral respiratory disease is common in the horse and provides a frequent reason for horse owners to seek veterinary attention for affected animals. A number of viral agents have been implicated, with influenza (A/equine 2) and herpesvirus (rhinopneumonitis, equine herpesvirus 4) being the most prominent causes of disease. Other viral agents implicated in equine respiratory disease include other strains of influenza virus, equine herpesviruses 2 and 5, equine viral arteritis, adenoviruses, rhinoviruses, and parainfluenza viruses. The significance of the last two virus groups in the pathogenesis of respiratory disease in horses is limited, although some recent research has indicated that rhinovirus infections may be more important than was previously realized. Respiratory viral infections generally occur in young animals that are stabled together, with spread between animals the result of direct contact, aerosols, fomites, and, in some cases (e.g., equine viral arteritis), via venereal spread.

KEY POINT

Outbreaks of viral respiratory disease is one of the major causes of wastage and poor performance in the racing industry. Valuable training time is lost while horses recover from infection.

Vaccines are now available for equine herpesviruses 1 and 4 (EHV-1 and EHV-4), the A/equine 1 and 2 strains of influenza virus, and equine viral arteritis virus, although the efficacy of vaccination programs remains open to question.

Adenovirus is a normal inhabitant of the upper respiratory tract and generally only causes infection in immunocompromised foals, such as Arabians with combined immunodeficiency (CID) or other horses that have been under significant stress.

HISTORY AND PRESENTING SIGNS

- Inappetence and signs of depression are two of the most commonly reported presenting signs.
- Outbreaks of the disease may occur, particularly in young horses or those housed under intensive conditions (e.g., racing and training facilities).
- Viral respiratory disease frequently causes poor performance.

CLINICAL FINDINGS AND DIAGNOSIS

- Viral respiratory infections cause a variety of clinical signs that vary depending on the type of virus involved and the age and susceptibility of the host.

KEY POINT

The most common signs include inappetence, fever, signs of depression, and a slight nasal discharge in the early stages of infection.

- Within 24 to 48 hours, the nasal discharge often is more copious, and a cough, due to pharyngitis and/or laryngitis, may be detected.

Equine Influenza

- The most debilitating effects of viral respiratory infections are commonly associated with equine influenza virus. Two subtypes of the virus exist, namely *A/equine/1/Prague 56* and *A/equine/2/Miami 63*, with the second subtype resulting in a more severe clinical syndrome.

KEY POINT

Spread is rapid, and morbidity is often high, with horses in training and those amassed in large populations (e.g., racetracks, training stables) being most at risk. Younger animals (2-3 years old) appear to be most susceptible.

- Signs include fever (up to 41°C), inappetence, nasal discharge, signs of depression, coughing, and a reduced willingness to move around. Harsh lung sounds may be detected.
- Uncomplicated infections tend to show improvement in clinical signs in 4 to 7 days, although a dry cough may persist for several weeks. Complications, including secondary bacterial invasion, cardiomyopathy, and persistently poor performance, also may occur.
- Horses exposed to continued stress or immune compromise may experience secondary complications (e.g., bacterial pneumonia or pleuropneumonia).

Equine Herpesvirus 4

- EHV-4 is the major cause of acute respiratory disease in horses throughout the world, with most being affected during the first 2 years of life.

KEY POINT

The clinical manifestations tend to be less severe and morbidity lower than those resulting from infection with influenza virus.

- In young foals, clinical signs are often relatively restricted and transient. However, in older foals that are exposed to high levels of stress (e.g., weaning) and where maternal antibody titers have waned clinical signs may be more severe, including fever, serous nasal discharge, pharyngeal lymphoid hyperplasia, lymphadenopathy, and an increase in the intensity of respiratory sounds. Herpetic lesions involve the mucous membranes of the upper and lower respiratory tract to varying extents, resulting in subclinical disease, sometimes progressing to acute bronchopneumonia.
- Secondary bacterial complications are common in foals exposed to continued stress throughout the course of the viral infection and may contribute significantly to morbidity.
- Latent infection with both EHV-1 and EHV-4 is apparently possible, and reactivation may occur after periods of stress or corticosteroid administration.

Equine Herpesvirus 1

- EHV-1 also is capable of causing respiratory disease similar to that caused by EHV-4. Recently isolation of EHV-1 from foals as young as 60 days of age has provided some indication of this important reservoir for spread of the disease. The most important economic losses due to EHV-1 are because of intermittent outbreaks of abortion, which in some years can have devastating effects on stud farms.

KEY POINT

Infection of pregnant mares with EHV-1 in the last trimester may cause abortion or the birth of weak foals that die soon after birth with degenerative lesions in the respiratory tract, liver, and lymphoid tissue.

- Neurologic dysfunction, specifically ataxia, has been reported in horses subsequent to infection with EHV-1 as a result of viral myeloencephalitis.

Equine Herpesvirus 2

- Fever, nasal discharge, lymphadenopathy, inappetence, pharyngeal lymphoid hyperplasia, and failure to thrive have been reported in foals in response to infection with EHV-2. Recent studies have demonstrated a greater prevalence of EHV-2 in the tracheal secretions of foals with respiratory disease compared with clinically unaffected foals. However, it has also been determined that most foals develop high serum neutralizing antibody titers by 7 months of age, indicating exposure and/or recovery from infection. A definite causal relationship between EHV-2 and respiratory disease is yet to be confirmed. Isolation of EHV-2 from leukocytes has led to an assumption of latency and/or subclinical infection and a possible role in reactivation of EHV-1 or EHV-4 infection.
- Keratoconjunctivitis also has been associated with infections with this strain of equine herpesvirus.

Equine Herpesvirus 5

- An association between EHV-5 and respiratory disease in horses is yet to be proved. However, its similarity to EHV-2 has led to its incrimination in vague respiratory syndromes after viral isolation from horses suffering from clinical respiratory disease.

Equine Rhinoviruses

- Three equine rhinoviruses have been identified and isolated from the upper respiratory tract of horses: equine rhinovirus type 1 (ERhV-1), type 2 (ERhV-2), and type 3 (ERhV-3). The three equine rhinovirus types were assumed to be members of the Picorniviridae. However, new evidence suggests that ERhV-1 may be more closely related to Apthoviridae, of which foot and mouth disease virus is a member.
- Equine rhinoviruses have been incriminated in causing respiratory disease ranging from severe pharyngitis and pyrexia to anorexia, mild submandibular lymph node enlargement, and a seromucoid nasal discharge.
- Serologic data have confirmed a high incidence of seroconversion to ERhV-1 and ERhV-2 in young horse populations around the world, often without overt respiratory disease. However, both ERhV-1 and ERhV-2 have been associated with natural and experimental induction of respiratory disease in horses providing further evidence for their involvement in equine respiratory disease.

Equine Viral Arteritis

- The clinical signs of infection with this virus are variable, with respiratory signs occurring on occasion.
- The most common indications of infection include fever, inappetence, peripheral edema, conjunctivitis, nasal discharge, possibly abortion, diarrhea, and frailty. Interstitial pneumonia has been reported infrequently in foals.

Equine Morbillivirus (Hendra Virus)

- EMV, recently renamed Hendra virus (HV), a newly discovered member of the paramyxoviridae family, is capable of causing a severe, acute, fatal interstitial pneumonia in horses. EMV (HV) is also a zoonotic infection capable of resulting in a fatal encephalitis and pneumonia in humans. To date, two separate outbreaks affecting both horses and humans have been described in Australia in 1994. Based on serologic evidence obtained from flying foxes, it is possible that these animals act as reservoir hosts. However, it is not clear how transmission occurs between host, horse, and humans.
- The incubation period ranges from 8 to 16 days, with affected horses showing signs consisting of pyrexia, tachypnea, dyspnea, and tachycardia. Progression to acute fulminating pulmonary edema occurs followed by recumbency and death usually within 3 days. Large volumes of blood-tinged stable foam can be found within the lung airways at necropsy. To date, the disease appears to have a high mortality rate in clinically affected horses.
- Diagnosis can be achieved using an indirect immunofluorescence test and polymerase chain reaction test. Serologic surveys using serum neutralizing antibody tests have not identified any other serologically positive horses in Australia.
- Because of the zoonotic potential of EMV (HV), universal precautions should be followed when treating animals suspected to be suffering from EMV (HV). Appropriate hygiene and quarantine procedures should be applied to all in-contact humans and horses.

DIAGNOSIS

- Diagnostic procedures for identification of the etiologic agent in horses with respiratory disease of suspected viral origin often are not performed because of the self-limiting nature of the disease in most cases.
- However, when an outbreak of severe respiratory disease occurs, the attending clinician may wish to pursue a specific diagnosis, and the index of suspicion may be increased by the utilization of virus isolation (from nasal mucus, nasal/nasopharyngeal, and conjunctival swabs and scrapings), immunofluorescence, electron microscopic, serologic, and histopathologic techniques.
- Serologic tests are used most commonly for confirmation of respiratory viral infections and require the collection of acute and convalescent serum samples at an interval of about 3 weeks. Serologic diagnosis of EHV-1 and EHV-4 exposure and/or infection can be achieved through type-specific ELISA kits. Recent retrospective and prospective serologic surveys indicate that approximately 100% and 28% of horses aged 2 years or more are serologically positive to EHV-4 and EHV-1, respectively.
- Polymerase chain reaction (PCR) technology is also available at some laboratories to enable identification of EHV-1 and EHV-4 genome in nasopharyngeal swabs. PCR tests are also available for EHV-2, EHV-5, ERhV, and EVA.
- If specific procedures are to be undertaken, the clinician should consult with a suitable diagnostic laboratory to determine the methods most applicable for diagnosis of the suspected viral agent and appropriate methods for handling samples.

TREATMENT

KEY POINT

Most uncomplicated viral infections in racehorses run a natural course of 7 to 14 days, with spontaneous resolution.

- In some cases, secondary bacterial infection may give rise to more severe clinical signs of lower respiratory disease.
- There is no specific effective treatment for viral respiratory disease.

KEY POINT

Good nursing care, reduction of stress, decrease or cessation of training, and minimization of potential complications should be the strategy used in horses with suspected viral respiratory disease.

- Infected horses can shed large amounts of virus in nasal secretions and can provide a significant reservoir of infection for other horses. Isolation of affected horses is indicated and should be encouraged.
- Some practitioners suggest that antibiotics should be administered to horses with suspected viral respiratory tract infections to prevent secondary complications. This practice is controversial because viruses are not sensitive to antibiotics and most viral respiratory tract infections are self-limiting. In addition, indiscriminate use of antibiotics encourages the selection of resistant strains of bacteria and can result in untoward side effects in horses being treated (e.g., diarrhea). As a result, we rarely use antibiotics in horses with suspected viral respiratory tract infections. Good nursing care and reduction of stress are likely to provide a better clinical response than antibiotics.
- Additional therapeutic considerations may involve the use of bronchodilator drugs (e.g., clenbuterol, aminophylline, and terbutaline—respectively; Treatment Nos. 27, 5, and 103).
- Pulmonary immunomodulation has been attempted in cases of infectious (viral) and noninfectious respiratory disease. Nonspecific immunostimulants in the form of inactivated *Propionibacterium acnes* (Equistim) and purified mycobacterial cell wall extract (Equimmune) are examples. Intravenous administration of these products has apparently resulted in clinical improvement in cases of undifferentiated infectious respiratory disease. Proposed mechanisms of action include stimulation of pulmonary macrophages, lymphocyte function, and cytokine production.
- Potential complications associated with administration of these products include fever, anorexia, and lethargy. Granulomatous pneumonitis has been reported in horses after intravenous injections of purified mycobacterial cell wall extract.

PROPHYLAXIS

- Isolation of infected animals (if possible), reduction of stress, and a clean well-ventilated environment probably provide the best mechanism for reducing the incidence and severity of viral respiratory tract infections.
- Killed and modified live virus vaccines for EHV-1 and EHV-4 are currently available in the United States (Treatment No. 110). A combined bivalent inactivated vaccine is now available in

Australia and Europe and has been shown to induce high serum antibody concentrations in vaccinated horses (Duvaxyn EHV 1,4 Vaccine, Duphar). The efficacy of these vaccines in preventing rhinopneumonitis has been questioned, but it does appear that they decrease the severity of clinical signs and the duration of viral shedding. One of the benefits of vaccination may be a reduction in the spread of the disease by limiting the amount of virus being liberated into the environment by infected horses.

- Recommended vaccination schedules using the combined inactivated vaccine (Duvaxyn EHV 1,4) include initial vaccination at 5 to 6 months of age followed by a second dose 4 to 6 weeks later and subsequent booster vaccinations at six monthly intervals.
- Vaccination of pregnant mares with EHV-1 antigens at 5, 7, and 9 months of gestation reduces the incidence of abortions due to infection with this virus.
- Inactivated virus vaccines for equine influenza A/equine 1 and A/equine 2 strains are available. Two primary doses are usually given several weeks apart when the horse is 4 to 8 months old. Booster injections are prescribed subsequently. The recommendation by manufacturers is for annual or semiannual boosters; however, many veterinarians prescribe 3 to 4 monthly boosters. Inactivated subunit vaccines are available and are said to stimulate higher serum antibody titres compared with conventional adjuvant inactivated vaccines.
- The efficacy of vaccination schedules for reducing the incidence or severity of equine influenza has been clearly established and regular revaccination should be encouraged so as to maintain high concentrations of neutralizing antibody.
- A modified live virus vaccine for protection against equine viral arteritis is available. The vaccine is effective in reducing the severity of signs, degree of viral shedding, and extent of spread of the disease. An inactivated vaccine (Artervac) is also available. However, it is uncertain if immunity is protective or long lived.

KEY POINT

Mild fever, inappetence, and local pain and swelling may occur after vaccination with any of the respiratory virus vaccines. This may reduce owner and trainer enthusiasm for compliance when veterinarians prescribe this form of prophylaxis.

Equine Respiratory Immunology—New Directions

The questionable efficacy of many respiratory viral vaccines has lead to further investigation of equine immunopathology. The stress of exercise and new environmental conditions have long been suspected as resulting in compromised host defense mechanisms. Recent studies have suggested that mechanisms such as cell mediated immunity and mucosal immunity play important roles in protective immunology against viruses like EHV-1 and equine influenza. Traditional killed vaccines result in primary stimulation of humoral IgG immunity, which is not necessarily protective. However, in the case of equine influenza, it reduces viral shedding after infection. Immunity after natural exposure to equine influenza may persist for 12 months or more even though serum neutralizing antibody titers are minimal, indicating the importance of other immune mechanisms. The use of attenuated vaccines allows immunostimulation through pathways similar to naturally occurring infection. However, they carry risk of recrudescence of virulence and subsequent disease.

The development of technologies including new vaccine adjuvants, recombinant DNA vaccines, and naked DNA vaccines may provide superior methods of stimulating specific (i.e., mucosal IgA) or complementary host defense mechanisms, thus inducing a longer-lasting and more effective immunity. The search for superior vaccines against the major equine respiratory viral pathogens continues, with encouraging results.

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CHAPTER 6

Cardiovascular System

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Diseases of the cardiovascular system are rare in horses compared with other species. However, in athletic horses required to perform at their peak, minor disturbances of cardiovascular function can result in significant decreases in exercise capacity.

KEY POINT

It is natural for clinicians to focus on the heart; therefore, auscultation forms one of the key parts of the clinical examination. However, evaluation of vessel function also should be a key part of cardiovascular assessment.

Over the last 20 years, we have been involved in the assessment of a variety of competitive horses (Thoroughbred and Standardbred racehorses, endurance and eventing horses) for reduced performance. We became aware that although cardiovascular assessment at rest was valuable, it often was difficult to determine the clinical significance of minor degrees of cardiovascular dysfunction. The availability of treadmills has resulted in the capacity to monitor various aspects of cardiovascular function at different exercise loads up to intensities similar to those of racing. Under these circumstances, it is sometimes possible to evaluate the significance of a low-grade heart murmur and to diagnose abnormalities that may not be apparent at rest. The advent of real-time two-dimensional ultrasound in conjunction with Doppler ultrasound techniques, such as pulsed-wave and color-flow Doppler, also has provided a greater insight into the effects of cardiovascular disease on cardiac output. Some aspects of cardiovascular function during exercise are discussed later in this chapter.

The cardiac reserve in the horse is notable. Although the heart rate at rest is usually in the range of 30 to 40 beats/minute, during exercise the maximum heart rate (HR_{max}) increases to up to 240 beats/minute. There is little increase in stroke volume during exercise, and in an average 450-kg Thoroughbred racehorse, the stroke volume will average 1 to 1.5 L/beat. Thus, the cardiac output will increase from around 40 L/min at rest to 240 to 350 L/min during maximal exercise. Because of this reserve, clinical signs of heart disease that commonly are found in other species may not be obvious in the horse until the end stage of disease. Considerable rigor must be exercised when evaluating the cardiovascular system and when providing assessments of abnormal findings, which may be subtle but have significant effects on exercise capacity.

Apart from primary cardiovascular disease, of greater significance in daily clinical practice is secondary cardiovascular dysfunction.

KEY POINT

Problems such as fluid and electrolyte disturbances, colic, endotoxemia, and grain overload can result in major disturbances to cardiovascular function.

Generally this is due to disturbances to fluid balance within the different fluid compartments.

CARDIOVASCULAR EXAMINATION

Evaluation of the cardiovascular system is an essential part of every physical examination. Cardiovascular examination is also an integral part of an examination for insurance purposes, prepurchase examination, and examination for performance-related problems. A detailed cardiovascular examination takes time, although a brief examination

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may be acceptable in horses presenting with a problem that is not likely to involve the cardiovascular system.

KEY POINT

One advantage of starting at the front and working to the rear of the horse is that different aspects of the cardiovascular system are examined: mucous membranes, peripheral perfusion, pulse quality and regularity (head), jugular venous distention (neck), and heart sounds (chest).

Signalment

The signalment may help to determine the likelihood of certain cardiac problems in horses. The most important aspect of signalment is the age of the horse, because a young horse (<3 years old) presenting with signs of cardiovascular disease is more likely to have a congenital cardiac problem, whereas an older horse (>3 years old) is likely to have valvular or conduction disturbances. The sex of the horse might lead to a tentative diagnosis such as aortic ring rupture in older breeding stallions. Breed predilections are not well documented in horses, but from previous reports one might consider certain problems in special breeds such as Arabian horses or Standardbreds. The use of the animal is important, because heart disease will have more impact in racehorses than in pleasure horses.

History

A detailed and accurate history is important in any clinical examination because it reveals valuable information and avoids unnecessary investigations. Information about the horse's stabling conditions and general environment, present and past performance, previous diseases, vaccinations, deworming history, appetite, water consumption, urination and defecation, and the occurrence of similar problems in other animals that have been in contact with the horse should be sought. It also may help to determine whether the problem relates to a decrease in performance or rather the horse has a long-standing problem of reduced exercise capacity. If specific documentation such as race or competition times is available, these may be used to further evaluate the severity of the presenting condition. History related to training schedules and duration of training is helpful in establishing whether the owner or trainer considers the horse to be at peak fitness. In our experience, such details are difficult to obtain from many horse trainers.

The presenting complaint should be characterized according to onset, duration, and progress, and if case treatment has already been instituted, the response should be evaluated. In regard to previous medical problems, it should be kept in mind that the cardiovascular problem might only be secondary to respiratory disease, colic, fluid and electrolyte imbalances, muscular problems, or thermoregulatory abnormalities.

KEY POINT

Of particular importance in the history is whether the problem for which the horse is presented is a progressive disorder, a constant problem, or an isolated event in the midst of apparent normality.

Isolated problems, with sudden collapse, unsteadiness on the legs, and abrupt decrease in performance, are more difficult to diagnose than problems that have a consistent and progressive history.

General Inspection

Observation of the horse should be performed from a reasonable distance so as not to influence behavioral and psychological patterns. The condition, general attitude, and movement in a stall or paddock should be noted.

KEY POINT

Of particular interest for the cardiovascular examination is the presence or absence of abdominal distention, ventral edema, venous engorgement, and the respiratory rate, effort, and pattern.

A prominent jugular pulse with distention of this vein should alert the clinician to the possibility of congestive heart failure.

Detailed Examination

VENOUS CIRCULATION

Evaluation of the venous circulation is difficult because of its low pressure. Of most significance is the jugular pulse, which reflects right atrial and thoracic pressure changes. Because the average right atrial pressure is around 4 mm Hg, the pressure wave does not progress far up the jugular vein. Normally, the jugular pulse is only visible around the thoracic inlet and for up to 10 cm rostrally. The jugular pulse is most evident in the normal horse toward the end of diastole, when

the stage of rapid ventricular filling is complete. However, if the head is lowered below the level of the right atrium or if there is an increase in right atrial pressure, which may be the situation in right-sided congestive heart failure, the pulse in the jugular vein becomes more prominent, together with jugular venous distention.

KEY POINT

In racehorses, because many medications are given via the jugular veins, it is important to check for the presence of thrombophlebitis by palpating along the length of the vein.

In some cases, thrombophlebitis can progress to occlude the affected jugular vein. Facial vein distention may be found in horses with thrombophlebitis of the jugular vein, particularly after exercise. Saphenous vein distention is normally found in horses at rest and after exercise. In cases of iliac thrombosis, a lack of saphenous vein distention has been noted on the affected side immediately after exercise.

MUCOUS MEMBRANE COLOR

The mucous membrane color gives an approximate guide to tissue oxygenation and perfusion of the capillary bed in the assessed area. Depending on the area examined, the mucous membranes of the horse are normally pale pink (mouth and eye) to pink (nasal septum) and should feel moist to the palpating finger.

KEY POINT

Only severe disturbances to peripheral perfusion and/or gas exchange will induce a change in mucous membrane color. With hypooxygenation of the blood, the color will turn bluish or cyanotic, whereas dehydration and endotoxemia will lead to a dark-red color, usually described as "injected. "

In very severe cases, anemia will result in pale mucous membranes. However, many horses will have mucous membranes that appear pale but have normal hemograms.

CAPILLARY REFILL TIME

Capillary refill time is a helpful clinical tool for assessing the peripheral circulation. It usually is evaluated by pressing firmly with the index finger on the mucous membrane of the gum above a corner incisor tooth. This will result in blanching of the mucous membrane, after which blood will refill the area over 1 to 2 seconds. If the test is repeated in the same area several times, there will be a decrease in the capillary refill time.

KEY POINT

Prolongation of capillary refill time may indicate a decrease in peripheral perfusion. However, care must be taken in interpretation of capillary refill time.

In septic or toxemic states, there may be dilatation of the arterioles, and despite a reduction in peripheral perfusion, the capillary refill time will be normal. It is also disturbing to note that on examination of the capillary refill time in several horses after death, we found that the refill time was excellent.

PERIPHERAL PULSE

The character of the pulse provides an important subjective guide to the state of the cardiovascular system. Pulse character depends on vessel size, distance away from the heart, and difference between systolic and diastolic pressures. Because there can be heightening of the pulse wave as it moves peripherally, the pulse may be more easily detectable in the digital artery than in the carotid artery.

KEY POINT

Assessment of the pulse character in the digital artery is part of the normal evaluation of the limbs, and digital pulses usually are more prominent when there is an inflammatory disease in the distal limb.

Increased digital pulse often is used as an indication of early laminitis. An exaggerated pulse in the central circulation is common in association with aortic insufficiency. A decrease in pulse pressure (hypokinetic pulse) is often the result of shock and hypovolemia.

The pulse is most easily detected in the facial artery as it rounds the mandible, although the transverse facial artery, near the lateral canthus of the eye, is also a good site for palpation. The different sites for detecting the peripheral pulse are shown in Chapter 1. The normal pulse character is a prolonged full wave that is easily palpated with mild digital pressure.

KEY POINT

Of greatest clinical significance is a decreased pulse pressure, which is detected as a weak peripheral pulse and usually indicates decreased systemic arterial pressure and shock.

Pulse rhythm is important to assess when the character of the pulse is being evaluated because it gives an indication of the presence of cardiac arrhythmias and should always be compared to heart rate and rhythm. Abnormalities of pulse rhythm, although not always easy to detect, will alert the clinician to the possibility of a primary cardiac disturbance.

PALPATION

Palpation is, compared with visual inspection, a more sensitive way of assessing ventral edema in horses, especially if they have a hairy coat or are heavily muscled horses. It is important to palpate all four legs and the ventral chest and abdomen for any signs of swelling.

KEY POINT

Before the commencement of cardiac auscultation, palpation of the apex beat gives an indication of the location of the heart in the thorax because it is the more apical region of the heart that usually will strike the chest wall.

Most commonly, the apex beat will be felt on the left ventral chest wall about 10 cm dorsal to the sternum, in the fifth or sixth intercostal spaces. In lean, narrow-chested, fit racehorses, it usually is easier to detect an apex beat than in broad-chested, fat, or heavily muscled horses. Uncommonly, an apex beat also can be felt on the right-hand side.

KEY POINT

If a thrill is detected on cardiac palpation, this indicates a severe flow disorder in the heart or in one of the great vessels.

Detection of such thrills, while uncommon, is important because it usually indicates severe cardiovascular disturbance. However, many clinically significant heart murmurs will not be accompanied by a thrill, particularly in a horse that is heavily muscled. We have noted that a thrill usually is found more often in young horses with congenital cardiac disease.

PERCUSSION

Percussion is a useful tool in the differentiation of the amount of gas and fluid in certain body tissues. This easily can be appreciated in the difference of sounds emanating from the percussion of the lung fields and muscled body areas. Whereas it is a well-regarded tool for diagnosis of lung disorders such as pleuritis and pneumonia, percussion has its limitations in the diagnosis of heart problems because it will only differentiate a very small or very large heart field. Although enlargement of the heart field is indicative of cardiac enlargement, a reduction of the heart field might be either attributable to an increase in lung size or other reasons that will move the heart away from the left chest wall, such as mediastinal masses or pericardial tamponade.

AUSCULTATION

Once the more peripheral parts of the circulation have been examined, careful auscultation of the heart should be performed on both sides of the chest. This should be carried out in a quiet area away from traffic or wind noise and, hopefully, without the owner or trainer talking to you. We have noted a remarkable tendency for owners to want to carry out a conversation as soon as you place the earpieces of the stethoscope in your ears.

Initial Auscultation

Auscultation should be undertaken using a good-quality stethoscope (i.e., Tycos The Harvey Elite or Littmann Cardiology II) with a diaphragm and a bell and tubing not longer than 40 cm because the heart sounds will be attenuated.

KEY POINT

It is important to use both the diaphragm and the bell of the stethoscope during cardiac auscultation.

The diaphragm will accentuate the higher frequency sounds (e.g., second heart sound) when applied firmly and the bell will enhance the lower frequency sounds (e.g., third heart sound) when applied lightly so as to just achieve an airtight seal.

The examination usually begins over the area of the apex beat, caudal to the triceps muscles

(usually the fifth intercostal space) and about 10 cm ventral to the level of the point of the shoulder. Over 60 to 90 seconds, a general impression of the heart sounds and rhythm should be gained. In many horses the heart rate will be elevated initially but will settle to a true resting rate of 25 to 40 beats per minute over 30 to 60 seconds. Abnormal heart sounds or murmurs will be detectable, as will gross disturbances to rhythm. However, a longer period of auscultation is necessary for more subtle findings.

Detailed Auscultation

The stethoscope should be placed over the point where the cardiac impulse can be felt most strongly on the left hemithorax and auscultation commenced with the diaphragm portion of the stethoscope placed firmly on the skin. Particular attention should be paid to the first heart sound in this area. This is the region where abnormal sounds associated with the mitral valve are best heard and where the first and third heart sounds are loudest. The stethoscope then is moved cranial and dorsal to the region over the aortic valve, where the second heart sound is most prominent. From there the stethoscope should be moved straight dorsally to follow the ascending aorta. From the area of the ascending aorta, the stethoscope is moved cranially to an area under the triceps muscle and then ventrally where the pulmonary valve component of the second heart sound becomes most prominent. This is also where the fourth heart sound (atrial contraction) is best heard. The stethoscope is then moved further ventrally and routinely pushed forward to the second intercostal space well under the triceps muscle.

KEY POINT

During the whole auscultation process the diaphragm portion should be used alternately with the bell portion of the stethoscope, especially around the valve areas, but generally the diaphragm portion is used for a longer period of time.

The examination is then continued on the right-hand side. In the case of absence of any palpable apex beat, auscultation is commenced in the fifth intercostal space about a hand-width dorsal to the sternum. The stethoscope should then be moved in a fashion similar to the left side, with the only landmark being the area of the tricuspid valve where the first heart sound can be heard best. Apart from the tricuspid valve area, particular attention should paid to the area just above the sternum, where ventricular septal defects are best heard and well forward in the second intercostal space to find radiating murmurs from the left side.

Normal Heart Sounds

Heart sounds are brief high-frequency noises that generally mark the beginning or end phases of the cardiac cycle.

KEY POINT

Heart sounds that can be heard easily are restricted to the first and second sounds, although in many horses three sounds can be heard (S_4, S_1 S_2), and in some, the soft ventricular inflow heart sound (S_3) can be detected over the mitral valve area.

FIRST HEART SOUND (S_1)

The first heart sound results from the noises generated by the closure of the left (mitral) and right (tricuspid) atrioventricular valves. Its intensity depends on the ability of these two valves to halt ventricular flow, on the mobility of the valve cups, and on the position of the cups and the rate of ventricular contraction. Although usually perceived as one sound, splitting of the first heart sound can occur in the heart of normal horses owing to the earlier rise of ventricular pressure in the left ventricle. Splitting of the first heart sound in horses is most easily detected in cases of atrial fibrillation, because the fourth heart sound is absent and closer attention can be paid to the first heart sound. In many cases where splitting of heart sounds are described in horses, it is actually a pseudosplitting caused by a very close time association of the fourth and first heart sounds.

SECOND HEART SOUND (S_2)

The second heart sound is generated by the closure of the aortic and pulmonary valves occurring after the blood flow has been reversed from antegrade to retrograde at the conclusion of systole. Splitting of the second heart sound may occur quite frequently in horses because the aortic and pulmonary valves do not close simultaneously. This may be due to a variety of reasons, some of which are pathologic.

THIRD HEART SOUND (S_3)

The third heart sound is a low-frequency sound produced by the distention and vibration of the ventricular wall, papillary muscles, and chordae

tendinae in diastole during the termination of rapid ventricular filling.

FOURTH HEART SOUND (S_4)

The fourth heart sound is a low-frequency sound generated by blood flow into the ventricles during atrial contraction. It is quite common in the horse, and its absence can lead to a preliminary diagnosis of failing atrial contraction, for example, as found in atrial fibrillation. Because of its close association with the first heart sound, S_4 has often been described as the first part of a split first heart sound, an assumption that was corrected after the introduction of phonocardiography.

EJECTION SOUNDS

In the veterinary literature, terminology of murmurs sometimes can appear confusing. The terms *ejection sounds, physiologic systolic, innocent systolic, functional systolic,* and *ejection-type murmurs* have been used to describe the same type of murmur occurring during systole in the absence of organic heart disease. To avoid confusion among readers, these terms should all be substituted by the term *functional systolic murmur,* indicating a nonpathologic heart murmur.

Heart Murmurs

Murmurs are audible successive sounds with distinct duration as opposed to heart sounds, which are short and transient. Murmurs are also defined as prolonged audible vibrations occurring during a normally silent period of the cardiac cycle. The exact mechanisms that lead to audible heart murmurs are still unknown, but the currently accepted theory is that they are a result of turbulent blood flow. A critical level of turbulence must be reached to produce a clinically audible murmur. Its characteristics depend on the velocity of blood flow and the surrounding structures that are caused to vibrate.

A critical level for turbulence associated with murmur production has been defined by a Reynold's critical number of 2000 or greater, and common causes for heart murmurs have been listed. These include flow in large-diameter vessels, valvular heart disease, and intracardiac shunts. However, a number of other problems can cause the abnormal sounds. For example, an increase in blood velocity or a decrease in blood viscosity can cause a heart murmur.

KEY POINT

A variety of problems can result in heart murmurs, not all pathologic.

Some problems resulting in heart murmurs are

- Decreased viscosity (most commonly anemia and/or hypoproteinemia)
- Conditions that produce increases in cardiac output (e.g., excitement, exercise, high temperature)
- Abnormal blood flow as typically occurs with valvular incompetence
- We also have noted a temporary heart murmur in some horses with colic. The reason for this transient murmur is not clear.

Murmurs are classified according to timing, intensity, point of maximal intensity, radiation, and pitch and quality.

FUNCTIONAL MURMURS

Functional murmurs occur in the absence of organic heart disease. They are usually subdivided into systolic and diastolic murmurs.

Functional Systolic Murmurs

Functional systolic murmurs are murmurs that typically last from early to midsystole and are crescendo-decrescendo in character. They are more commonly found in fit racehorses and are thought to be a result of superior cardiac contractility and lower heart rates, combined with an increase in aortic diameter.

Functional Diastolic Murmurs

A variety of functional diastolic murmurs have been described in equine cardiology. These murmurs are usually of short duration, beginning with or immediately after the second heart sound and extending to or just beyond the third heart sound. Presystolic murmurs occurring between the fourth and the first heart sound also have been reported in clinically healthy horses. Note should be made of a peculiar murmur that is commonly found in young Thoroughbred horses in training. It has been called the "2-year-old squeak" and is a faint high-pitched sound that occurs just after the second heart sound during the rapid filling phase of the left ventricle.

PATHOLOGIC MURMURS

Five main pathologic murmurs caused by organic heart disease are recognized in the horse: murmurs caused by ventricular septal defects, mitral regurgitation, tricuspid regurgitation, aortic regurgitation, and patent ductus arteriosus.

Ejection Murmurs

Ejection murmurs occur with valvular stenosis, increased flow through the aortic and pulmonary

valves, or valvular damage without stenosis or dilation of the vessel beyond the valve. However, pulmonary stenosis has rarely been reported in horses, and the occurrence of aortic stenosis in horses is still controversial. Congenital stenosis of the aortic valve is considered rare. Acquired stenosis may occur in cases of endocarditis. In a postmortem study of 1557 horses, the aortic valve was the site of lesions, ranging from nodules to fibrous bands or fenestrations of varying size in 331 horses (20%). However, only 11% of these horses were thought to have aortic stenosis. Apart from these reports, aortic stenosis only has been mentioned in two case reports.

Regurgitant Murmurs

Systolic regurgitant murmurs are caused by abnormal blood flow throughout systole from a chamber or vessel with a higher pressure to a chamber or vessel with lower pressure. Because this pressure gradient usually persists throughout systole, these murmurs tend to have an even or plateau configuration. Systolic regurgitant murmurs result from mitral or tricuspid insufficiency and ventricular septal defects.

KEY POINT

Regurgitant murmurs are holosystolic and have a plateau character.

Diastolic Murmurs

Diastolic murmurs are divided into three categories: presystolic murmurs, ventricular filling murmurs, and regurgitant murmurs from incompetent aortic and pulmonary valves. Presystolic and ventricular filling murmurs have been described above. Ventricular filling murmurs can also occur with severe mitral regurgitation. Mitral and tricuspid stenosis are recognized causes of diastolic murmurs in humans but rarely occur in horses, although they have been reported in foals as part of complex congenital disorders.

Aortic valve insufficiency is a common condition in horses, and its incidence has been reported to increase with age. Pulmonary valve insufficiency can occur in horses but is rarely associated with a distinctive murmur.

MURMUR CLASSIFICATION

Cardiac murmurs should be described based on their timing, intensity, point of maximal intensity, radiation, pitch, and quality. This classification will aid in their identification and is a useful reference for subsequent examinations. Systolic murmurs especially may be difficult to differentiate in horses because they are the most common type of murmur and can arise from a number of sites.

Timing

Murmurs should be identified from their timing within the cardiac cycle as systolic or diastolic. The duration of systole and diastole can be subdivided into the following categories:

Holosystolic—Beginning with the first heart sound and ending with the second heart sound, which is defined as the aortic component

Holodiastolic—Beginning with the second heart sound and ending just before the first heart sound

Presystolic—Between the fourth and first heart sound

In light of these different approaches to cardiac auscultation, it would seem wise to institute a universally accepted standardized system.

Intensity and Radiation

The intensity of the murmur may help in its differentiation. Systolic ejection murmurs often are loudest in early systole and then decrease, whereas regurgitant murmurs tend to have a more constant intensity.

KEY POINT

The intensity of murmurs generally is graded using a grading system from 1 to 6, with grade 1 being barely detectable and grade 6 indicating that the murmur can be heard even when the stethoscope is removed from the chest wall.

The significance of the intensity of a murmur is still controversial, but generally the louder a murmur is and the wider it radiates across the auscultatory field, the more likely it is to be clinically significant. The point of maximum intensity of murmurs traditionally has been linked to the anatomic position of the valves, but exceptions have been noted. For example, systolic sounds, associated with the atrioventricular valves, are best heard toward the apex beat area because they are transmitted by the stiff ventricular wall to the point at which it touches the chest wall. However, external anatomic landmarks are still a relatively good guide to the location of underlying cardiac structures and therefore are helpful for cardiac auscultation. A detailed description of the grading of each of the murmurs found is give below:

Grade 1—A quiet murmur that is just detectable after careful and prolonged auscultation, found over a localized area

Grade 2—A quiet localized murmur that is heard immediately once the stethoscope is placed over its point of maximum intensity

Grade 3—A moderately loud murmur

Grade 4—A loud murmur heard over a widespread area with no palpable thrill

Grade 5—A loud murmur with an associated precordial thrill

Grade 6—A murmur sufficiently loud that it can be heard with the stethoscope just off the skin surface

Pitch and Quality

The pitch and quality (character) of a murmur is a subjective quality that might be perceived differently by individual examiners. For example, murmurs associated with an insufficiency of the atrioventricular valves have a plateau or band-shaped character; functional systolic murmurs have a crescendo or crescendo-decrescendo character; murmurs associated with aortic valve incompetence have a decrescendo character; and early diastolic murmurs in young Thoroughbreds have a high-pitched or squeak quality.

Abnormal Rhythms (Dysrhythmias or Arrhythmias)

The terms *dysrhythmia* and *arrhythmia* are often used interchangeably. However, in a strict sense, dysrhythmia means a disturbance of rhythm, whereas arrhythmia indicates an absence of rhythm. In accordance with common usage, we use the term arrhythmia to avoid confusion. Some arrhythmias (e.g., atrial fibrillation) are immediately recognizable within seconds of placing the stethoscope on the chest wall. Others are more subtle and, if intermittent, may be missed on auscultation. For this reason, it is important to auscultate for several minutes to assess rhythm abnormalities. The following details should be noted:

- Ventricular rate (normal, bradycardic, or tachycardic)
- Rhythm of first and second heart sounds (regular or irregular)
- Presence of "dropped" or premature beats

The most common arrhythmias are second-degree atrioventricular block, sinoatrial block, and sinus arrhythmia. These arrhythmias are considered to be within the normal range of findings, but an electocardiogram (ECG) may be required to make the diagnosis.

DIAGNOSTIC AIDS

From the physical examination it should be clear whether the problem involves the central or peripheral circulation. A range of diagnostic aids then can be used depending on the facilities and equipment available. In some cases, it may be clear that the problem is an electrical one, and therefore an ECG is needed. In others, the history may be suggestive of primary cardiac disease, but auscultatory findings are normal and there are no abnormal findings on physical examination. In these cases, it may be necessary to undertake a range of diagnostic procedures, including ultrasound examination and telemetry electrocardiography during exercise, to establish a diagnosis.

Electrocardiography

The most easily accessible of the diagnostic aids for cardiovascular problems is the ECG.

KEY POINT

However, it must be remembered that the ECG has somewhat limited value because it provides information about the electrical activity of the heart rather than its mechanical function. Thus, many horses with high-grade pathologic heart murmurs will have normal ECG findings.

The ECG machine can be thought of as a type of galvanometer that is used in such a way that the potential difference is measured between two electrodes situated on the surface of the body.

RECORDING TECHNIQUE

To record an ECG, one needs a quiet place with minimal interference. A rubber mat for the horse to stand on has been suggested, but in most cases this can be dispensed with, provided that there are no major sources of electrical interference.

KEY POINT

The best place for recording an ECG is the horse's own box stall, if available. This is because the horse is usually calm and relaxed in the box stall and a high-quality recording can be obtained quickly.

Care must be taken to ensure that the site where the horse stands during recording is completely dry. The ECG machine should have filters to avoid interference, particularly from skin movement and electrical activity.

The ECG machine used should be portable and enable recording without an immediate main power supply by using rechargeable batteries. Recordings usually are made at a paper speed of 25 mm/s and a sensitivity of 1 cm = 1 mV.

The configurations of both QRS and T-wave complexes are affected by limb position and heart rate. Therefore, to allow standardized interpretation, the ECG should always be recorded with the left forelimb slightly in front of the right forelimb and the heart rate less than 42 beats/minute.

The recording system used is usually one of two main types:

1. *Metal electrodes that are connected to the legs by rubber straps.* To permit good conductivity, an electrode paste is applied under the electrodes. Usual electrode placement on the forelimb is on the caudal aspect of the distal radius just proximal to the accessory carpal bone, whereas electrodes are applied to the cranial aspect of the distal tibia just above the point of the hock in the hindlimbs. The chest electrode is secured using a rubber girth strap so that the lead lies 5 cm behind the point of the elbow on the left ventral thorax. If the electrodes are applied directly to the skin by alligator clips, this can be performed by attachment to the skin just below the olecranon in the forelimbs and just below the stifle in the hindlimbs. The machine should be able to record leads I, II, III, aVR, aVL, aVF, and V (the exploring electrode). This system is criticized on the basis that for a quadruped, the frontal lead system is inaccurate because it is based on the three recording leads (left foreleg, right foreleg, and left hindleg) being part of an equilateral triangle (Einthoven's triangle), which is obviously not the case. For determination of conduction disturbances, it does not matter if this system or the Y lead described below are used. More complex vectorcardiography has been described, but it appears to provide little extra information to the clinician than the more simple recording systems.

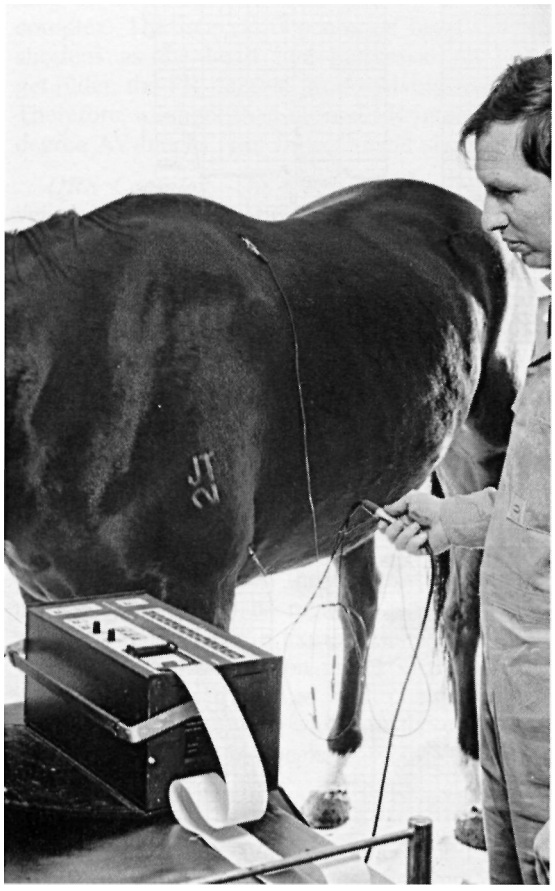


Figure 6-1. ECG recording being taken from a horse using a typical base-apex lead system, the most widely used recording technique for assessing the ECG in practice.

2. *A bipolar lead system using the Y lead.* This is the most commonly used system for general ECG examination, and this or a base-apex system, as is shown in Figure 6-1, may be used. To record the Y lead, the positive lead is attached over the xiphisternum and the negative lead over the manubrium. The ground, or earth, lead (brown) can be attached over the point of the shoulder. Mostly this is performed using alligator clips attached directly to the skin, with ECG paste being applied to the sites to increase conductivity. An alternative to commercial paste is alcohol, but a cheap ECG paste also can be made up by mixing salt with an obstetric lubricant. Some horses object to the alligator clips, and in these horses the metal electrodes and rubber straps will permit a better recording to be made than with the clips applied directly to the skin.

INTERPRETATION OF THE ECG

It is important to obtain several minutes of ECG recording to allow assessment of changes in rhythm and to determine the presence of conduction abnormalities. The most obvious findings will relate to rhythm disturbances. Obvious abnormalities in individual waveforms can be recognized, as can missing components of the ECG, for example, a P wave not followed by a QRS complex or T wave, indicating second-degree atrioventricular (AV) block. Some findings, such as ventricular premature contractions, only may be found as an isolated event on a recording and may be missed unless the tracing is evaluated carefully. The following sequence for ECG investigation is useful:

- Examine the complete tracing to note any abnormal rhythm.
- Determine whether complete P waves, QRS complexes, and T waves are present for each heartbeat throughout the trace.
- Assess individual wave formations, and measure the intervals.

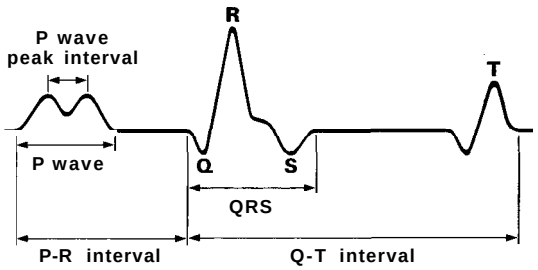


Figure 6-2. Typical lead II recording of an ECG showing the different waveforms and their measurement.

Analysis of the ECG should be performed using an 8 x to 10 x magnifier to assist in accuracy of interval measurement. In the frontal lead system described above, the following intervals are normal for measurements in lead 2:

P-wave duration	<0.17 second
P-wave peak interval	<0.08 second
PR interval	<0.44 second
QRS complex duration	<0.17 second
QT interval	<0.60 second

Measurement of the various intervals is shown in Figure 6-2.

It should be noted that the PR and QT intervals are heart rate dependent, with the intervals shortening considerably when there is an increase in heart rate. A normal ECG tracing using a frontal lead system and recording the various leads is shown in Figure 6-3. A typical tracing using the Y lead is shown in Figure 6-4.

EVALUATION OF ECG WAVEFORMS AND INTERVALS

P Wave. The P wave represents atrial depolarization and will vary in appearance. Most corn-

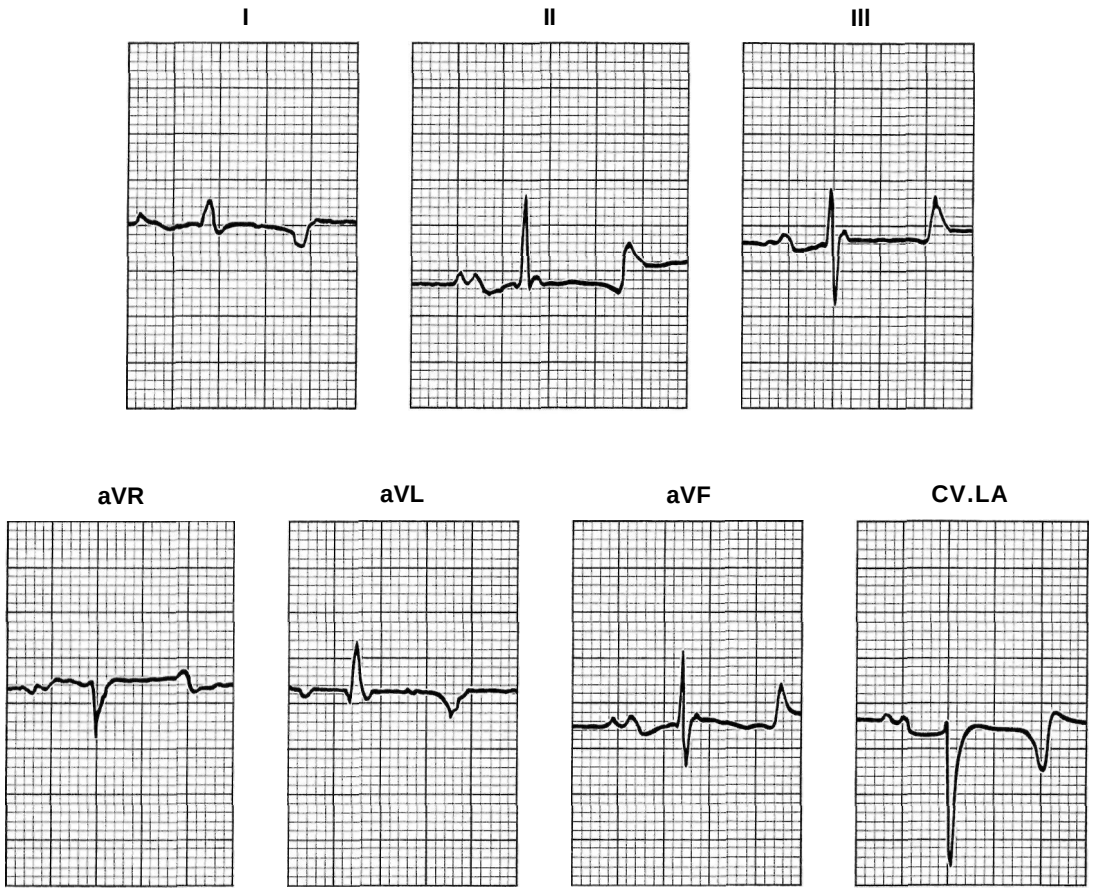


Figure 6-3. Normal frontal-lead ECG recording, from leads I, II, III, aVR, aVL, aVF, and CV.LA (chest lead), showing typical waveforms. Recording was made at 25 mm/s and an amplitude of 1 mV/cm. The chest lead (CV.LA) is recorded with the electrode located approximately 5 cm caudal to the point of the elbow.

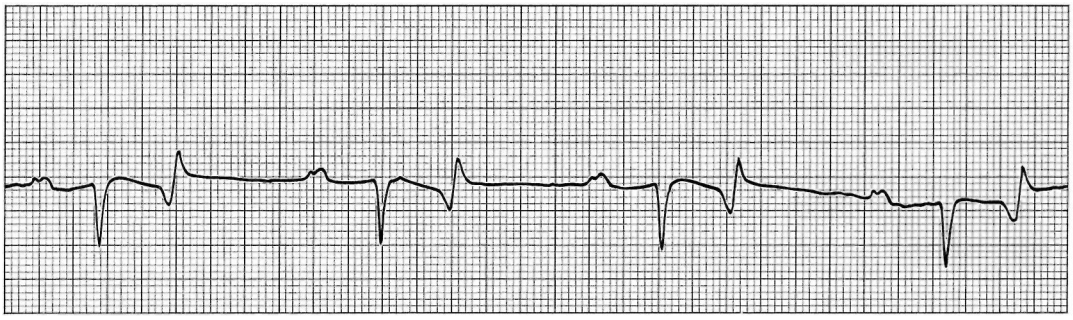


Figure 6-4. Typical recording using the Y lead, with electrodes placed over the manubrium (positive) and xiphisternum (negative).

monly it has an M-shaped appearance, but it can appear diphasic, as shown in Figure 6-5. It is quite common to have a number of different waveforms within the same ECG lead, the appearance varying from beat to beat. This is normal and is referred to as wandering pacemaker.

*PR **Interval.*** The PR interval is from the beginning of the P wave to the beginning of the QRS complex. The interval depends on heart rate and shortens as the heart rate increases. As horses get older, the PR interval progressively lengthens. Therefore, a longer than normal PR interval (first-degree AV block) is of little clinical significance.

***QRS** Complex.* The QRS complex represents ventricular depolarization, and depending on the particular lead, some of the components may not be present. The Q wave is often missing, and the S wave is variable in appearance. Some of the variations in QRS waveforms are shown in Figure 6-6.

T Wave. The T wave represents ventricular repolarization. It is extremely sensitive to changes in heart rate, with changes in both amplitude and polarity. As the heart rate increases, usually there is an increase in both the amplitude of the T wave and a change in direction so that it becomes positive, particularly in the unipolar V lead. Although some clinicians regard changes in the T wave as abnormal, the extreme lability of the T wave makes interpretation rather difficult. There is evidence that T waves will change polarity with training state, with waves in the chest leads becoming positive and peaked (>1 mV). The following T-wave directions have been found to be normal using the frontal lead system:

Lead	T-Wave Direction
Lead I	Negative or diphasic
Lead II	Positive or diphasic
Lead III	Positive or diphasic
Lead aVR	Positive or diphasic
Lead aVL	Negative
Lead aVF	Positive or diphasic
Chest leads	Diphasic

A typical ECG tracing in a horse with "abnormal" T waves in a number of leads, recorded using the frontal system, is shown in Figure 6-7.

*QT **Interval.*** This interval, from the beginning of the QRS complex to the end of the T wave, is of little clinical significance. The interval will shorten with an increase in heart rate.

THE HEART-SCORE CONCEPT

The concept of heart score was developed by Dr. Jim Steel in Australia in the late 1950s. The heart score is an indirect method for assessing heart size and is determined by measuring the QRS complex duration in milliseconds in leads I, II, and **III** and averaging the result. A horse with QRS complex durations of 100, 120, and 110 ms, respectively, in leads I, II, and III has a heart score of 110. Average heart score values in mature Thoroughbred horses are 113 to 116, whereas values over 120 are considered to be indicative of above-average heart size. This is based on the good correlation (r = 0.89) noted by Steel between



Figure 6-5. Range of normal appearances of the P wave in an ECG recording.

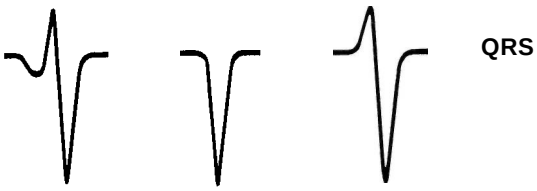


Figure 6-6. Range of normal appearances of the QRS complex in an ECG recording.

heart score and heart weight. There was also an association between heart score and prize money won by Thoroughbred horses, although the correlation coefficient was only 0.44.

From the measurement of heart score, veterinarians have made predictions about performance potential. Other studies have shown correlations with performance in Standardbred pacers and endurance horses.

Although heart-score measurement is used extensively in some countries (Australia, New Zealand, South Africa, and France), various authorities have described the measurement as being unphysiologic and of no value. Apart from the difficulty in measurement of QRS complex duration, Steel's data show that only 80% of the variation in heart score is due to changes in heart weight. Furthermore, no allowance is made for variations in an individual horse's body weight. Therefore, while it can be said that horses with low heart scores (<100) are likely to have lower cardiac capacity and horses with high heart scores (>120) are likely to have higher capacity, it is difficult to be more precise.

KEY POINT

The heart-score concept has been overemphasized by some veterinarians because of the assumption that heart size is the only factor of importance in determining athletic performance.

Heart size is, of course, only one element, and other factors, including respiratory function,

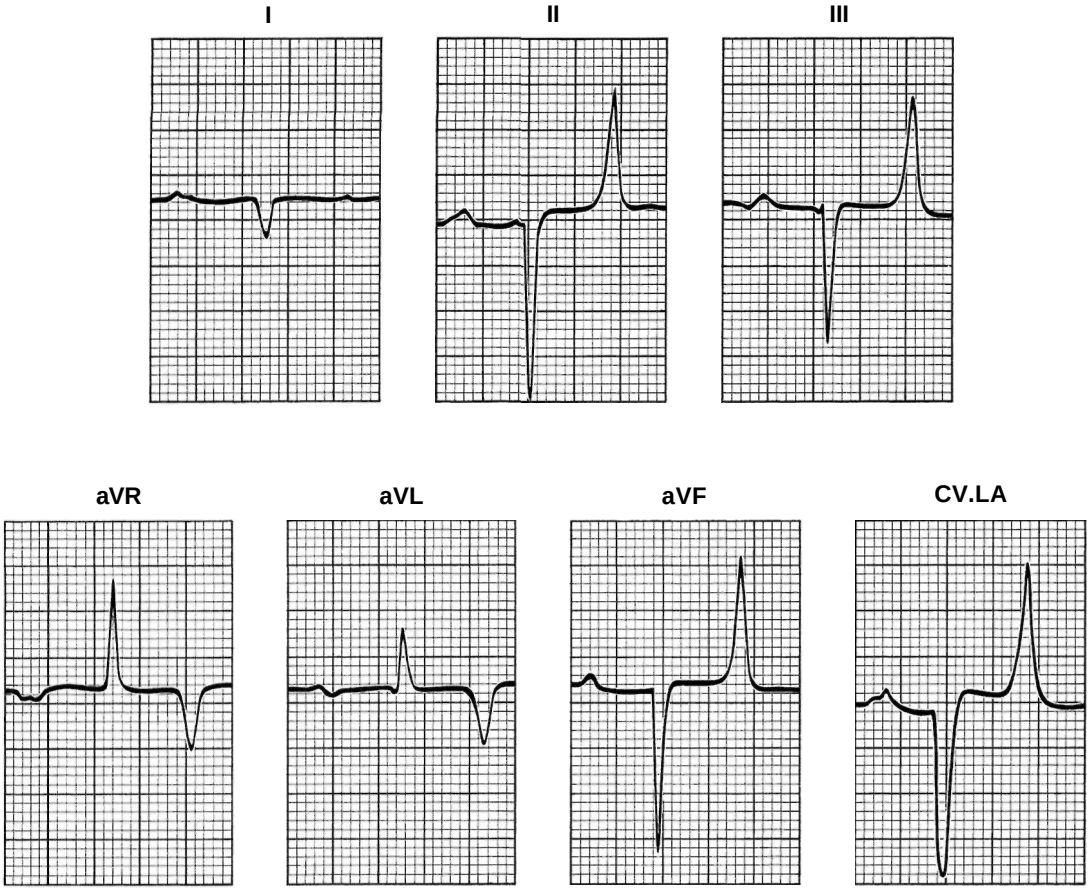


Figure 6-7. ECG tracing from a horse with "abnormal" T waves recorded using the frontal system. Recording was made at 25 mm/s and an amplitude of 1 mV/cm.

muscle function, biomechanics, and oxygen-carrying capacity, all play important roles in performance. The heart score is therefore of some value in providing a broad indication of heart size, but limited significance should be given and care should be taken in providing predictions of performance.

Echocardiography

Since its introduction to equine cardiology by Pipers and Hamlin in the late 1970s, equine echocardiography has proved to be one of the most useful tools for investigation of congenital and acquired heart disease in horses. The technique permits real-time imaging of the heart, allowing quantitation of cardiac dimensions, identification of specific congenital and acquired cardiac lesions, and estimation of the degree of cardiac compensation and myocardial failure. Five techniques are available: M-mode, B-mode or two-dimensional real time, continuous-wave Doppler, pulsed-wave Doppler, and color-flow Doppler. The first two now are routinely used by equine practitioners, making echocardiography a clinically useful technique with widespread application. The latter three techniques are more commonly used in academic institutions and specialized clinics and are used for the evaluation of flow disturbances in cases of valvular dysfunction or congenital heart problems.

For cardiac examination in adult horses, a 2.5- to 3.5-MHz transducer is necessary, and the penetration depth should be at least 24 cm. The most common mode used for echocardiography has, until about 12 years ago, been M-mode, which provides a one-dimensional image of the heart. As in human cardiology, M-mode echocardiography is still used today to measure cardiac dimensions and indices of cardiac function and to assess valve motion. Its superior temporal resolution and convenient graphic display make it the most accurate measurement method for this purpose.

Two-dimensional real-time (B-mode) ultrasound provides a cross-sectional view of the heart in motion and is capable of imaging infinite numbers of planes. Therefore, it has been important to introduce standardized images for comparisons between studies, which has been done over the past 6 years, and the reader is referred to these publications at the end of this chapter. The most commonly used standardized images are shown in the following figures. The importance of these standardized images is that the reader can relate the position of the axial beam to various intracardiac landmarks, thereby making the appropriate measurements more easily comparable.

The following standardized echocardiographic imaging planes are some of the more commonly used for quantitative and qualitative two-dimensional, guided M-mode, and Doppler echocardiography studies in the horse. They are based on standardized images described previously.

RIGHT PARASTERNAL LONG-AXIS VIEWS

These views are taken from the right hemithorax of the horse with the transducer placed in the fifth, fourth, or third intercostal space.

Reference View (Ventricular Inlets)

The reference view (Fig. 6-8) is obtained by placing the transducer in its reference position on the right hemithorax. This usually means the fourth intercostal space, perpendicular to the thoracic wall, 0-degree rotation and 0-degree angulation. In our experience the transducer has to be angled more caudally in Standardbreds than in Thoroughbreds to obtain the reference view. In this plane, the structures imaged are the right ventricle (RV), tricuspid valve (TV), right atrium (RA), interventricular septum (IVS), interatrial septum (IAS), the left ventricle (LV), the mitral valve (MV), the left atrium (LA), and the left ventricular free wall (LVFW). In this particular

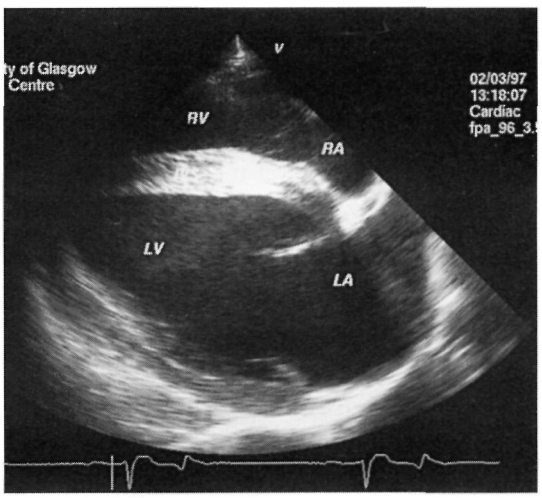


Figure 6-8. Echocardiogram using the right parasternal reference view. This is obtained by placing the transducer is in its reference position on the right hemithorax in the fourth intercostal space, perpendicular to the thoracic wall with 0 degree rotation and 0 degree angulation. In this plane, the structures imaged are the right ventricle (RV), tricuspid valve, right atrium (RA), interventricular septum (IVS), interatrial septum, the left ventricle (LV), the mitral valve, the left atrium (LA), and the left ventricular free wall. A pulmonary vein is seen entering the left atrium.

picture, a pulmonary vein is seen entering the left atrium.

Apical View (Ventricular Inlets)

By sliding the transducer even more ventral from the previous position and having it back in a 0-degree position, the long axis of the heart can be displayed as vertical as possible by angling the transducer dorsally until just before losing contact with the skin surface. The structures imaged (Fig. 6-9) in this plane are indicated in the figure legend.

Long-Axis Aorta

The transducer is placed cranially and rotated 30 degrees clockwise to obtain this image plane (Fig. 6-10).

RIGHT PARASTERNAL SHORT-AXIS VIEWS

To obtain the right parasternal short-axis views in the horse, the transducer has to be rotated 90 degrees couterclockwise from its reference position and then adjusted to a lesser rotation for the different views. This results in the cranial structures being displayed on the left side and the left ventricular lumen being displayed on the right side of the screen. In the three views presented here, care should be taken to get a true short-axis plane transecting the left ventricle by trying to obtain a left ventricular lumen as close to circular as possible.

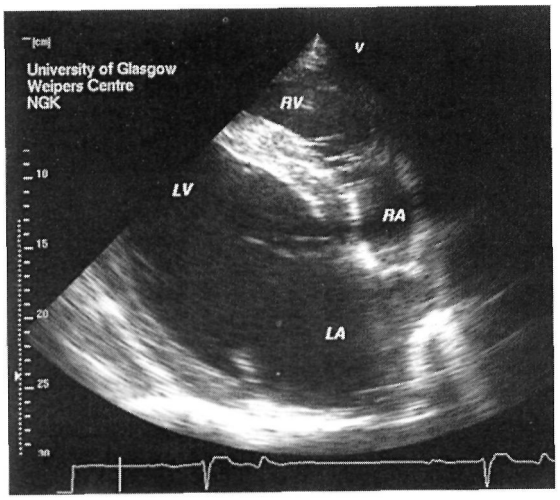


Figure 6-9. Echocardiogram using the right parasternal apical view. The long axis of the heart can be displayed as vertical as possible by angling the transducer dorsally until just before losing contact with the skin surface. The structures imaged in this plane are right atrium (RA), tricuspid valve, right ventricle (RV), interventricular septum, left ventricle (LV), mitral valve, and left atrium (LA).

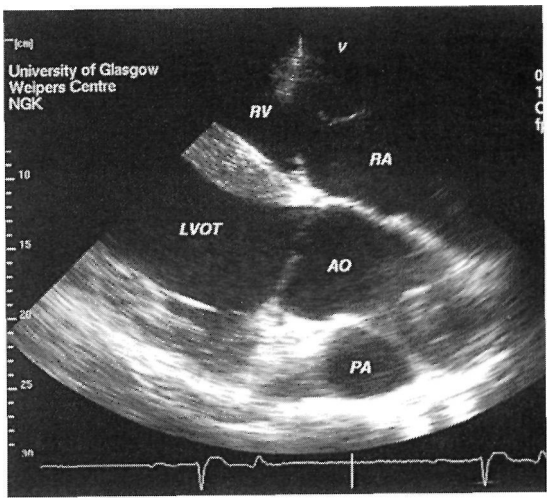


Figure 6-10. Echocardiogram using the right parasternal apical view. This long-axis view of the aorta is obtained by placing the transducer cranially and rotating it 30 degrees clockwise. The structures imaged in this plane are the right ventricle (RV), tricuspid valve, right atrium (RA), interventricular septum, left ventricle (LV), chordae tendineae of the mitral valve, left ventricular free wall, left ventricular outflow tract (LVOT), aortic valve, ascending aorta (AO), and pulmonary artery (PA).

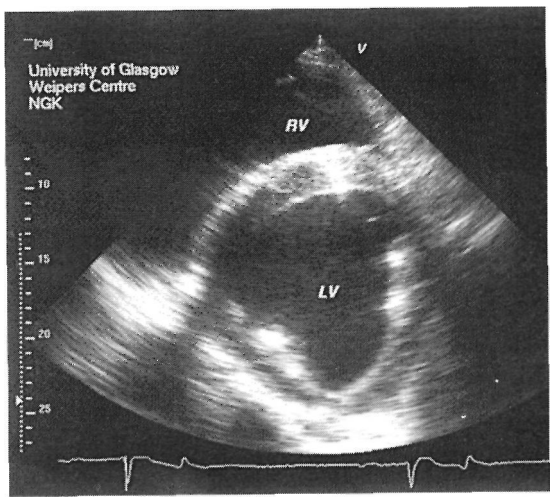


Figure 6-11. Echocardiogram using the right parasternal short-axis view, papillary muscle level. To obtain this view, the transducer is rotated 90 degrees counterclockwise. Both papillary muscles are displayed evenly and no chordae tendinae of the mitral valve are displayed. The right ventricle usually is not shown in its full dimensions but portions of the right ventricular wall usually can be identified. The structures imaged in this plane are right ventricular wall, right ventricle (RV), interventricular septum, papillary muscles, left ventricle (LV), and left ventricular free wall.

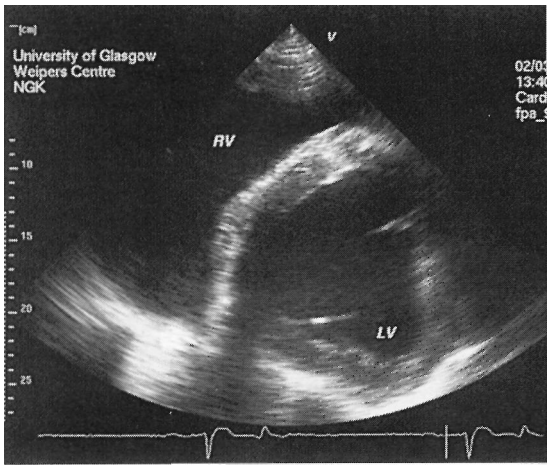


Figure 6-12. Echocardiogram using the right parasternal short-axis view, chordal level. The papillary muscles are no longer visible, and the chordae tendinae are displayed. The structures displayed in this image plane are right ventricle (RV), right ventricular wall, interventricular septum, left ventricle (LV), left ventricular free wall, and chordae tendinae. The axial beam here transects the interventricular septum.

Papillary Muscle Level

The transducer is rotated 90 degrees counterclockwise to obtain this image plane (Fig. 6-11). Usually it stays in a perpendicular position to the chest wall, but minor adjustments are made, if necessary, to achieve a true short-axis transection of the left ventricular lumen. Both papillary muscles are displayed evenly, and no chordae tendinae of the mitral valve are seen. The right ventricle usually is not displayed in its full dimensions, but portions of the right ventricular wall can usually be identified.

Chordal Level

The papillary muscles are no longer visible, and the chordae tendineae are displayed on the screen (Fig. 6-12).

Mitral Valve Level

The transducer is moved further dorsally from the above position until the mitral valve is displayed in its characteristic "fish-mouth" appearance in systole and diastole (Fig. 6-13).

Aortic Valve Level

The transducer has to be angled slightly dorsal and rotated to a 30 degree counterclockwise position relative to the reference position to obtain this image plane (Fig. 6-14).

Pulmonary Artery Level

To obtain a better image of the pulmonary artery, the transducer has to be angled slightly

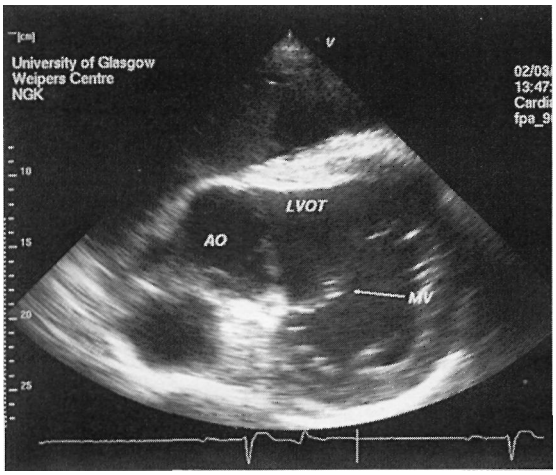


Figure 6-13. Echocardiogram using the right parasternal short-axis view, mitral valve level. The transducer is moved further dorsally from the position described in Figure 6-12 until the mitral valve is displayed in its characteristic "fish-mouth" appearance in systole and diastole. The structures imaged are tricuspid valve, interventricular septum, mitral valve (MV), left ventricular outflow tract (LVOT), and the left ventricular free wall. AO, ascending aorta.

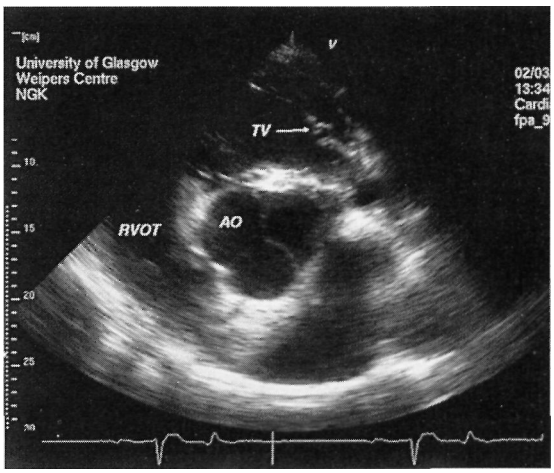


Figure 6-14. Echocardiogram using the right parasternal short-axis view, aortic valve level. To obtain this view, the transducer has to be angled slightly dorsally and rotated to a 30 degree counterclockwise position relative to the reference position. The structures imaged are the right ventricle, tricuspid valve (TV), right atrium, aortic valve (AO), left atrium, and part of the right ventricular outflow tract (RVOT), depending on the degree of rotation.

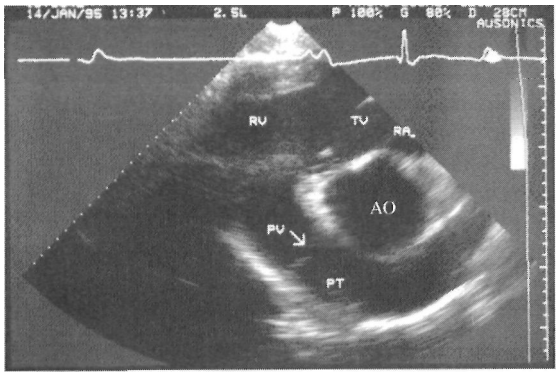


Figure 6-15. Echocardiogram using right parasternal short-axis view, pulmonary artery level. To obtain a better image of the pulmonary artery, the transducer has to be angled slightly dorsally and cranially from the above position. The structures imaged are right ventricle (RV), tricuspid valve (TV), right atrium (RA), aorta (AO), pulmonary valve (PV), and pulmonary trunk (PT).

dorsally and cranially from the above position (Fig. 6-15).

Right Parasternal Angled View

To obtain the right parasternal angled view, it is necessary to move the transducer one intercostal space cranial relative to the reference view. Frequently, the right foreleg has to be abducted slightly and the triceps muscle pushed cranial to make satisfactory transducer positioning possible.

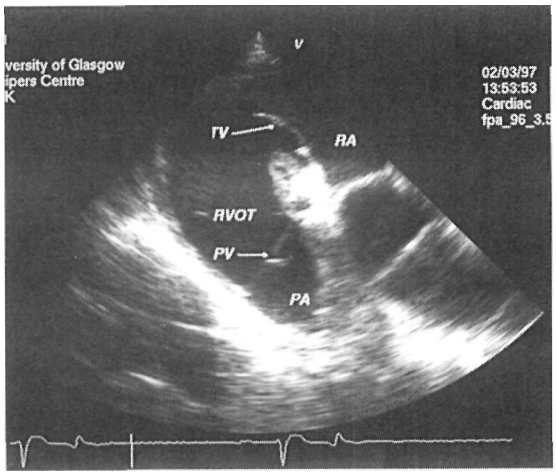
Dorsal Location (Right Ventricular Outflow Tract)

In the abovementioned intercostal space, the transducer has to be moved slightly dorsal and angled cranial while rotating it 30 degrees counterclockwise to obtain an image of the right ventricular outflow tract (Fig. 6-16). In this particular view, an attempt is made to achieve a good image of the pulmonary valve, whereas in the next view (Fig. 6-17) the pulmonary artery is better visualized. Although they are generally the same view, this shows how small changes in angulation and rotation can allow the examiner to highlight adjacent structures.

LEFT PARASTERNAL VIEWS

The left parasternal views are obtained from the left hemithorax of the horses with a defined reference position in long axis being the guide for other views and transducer placement similar to those described on the right hemithorax.

The reference view (left ventricular inlet) is achieved with the transducer placed in the fifth or fourth intercostal space, a 0-degree rotation and, depending on the intercostal space, slight caudal (fourth) or cranial (fifth) angulation.



'Figure B-T6. "Echocardiogram using right parasternal angle view, dorsal location for the right ventricular outflow tract. The transducer is moved slightly dorsally and angled cranially while rotating it 30 degrees counterclockwise to obtain an image of the right ventricular outflow tract. The structures imaged are right ventricle, tricuspid valve (TV), right atrium (RA), right ventricular outflow tract (RVOT), pulmonic valve (PV), pulmonary artery (PA), and right coronary artery.

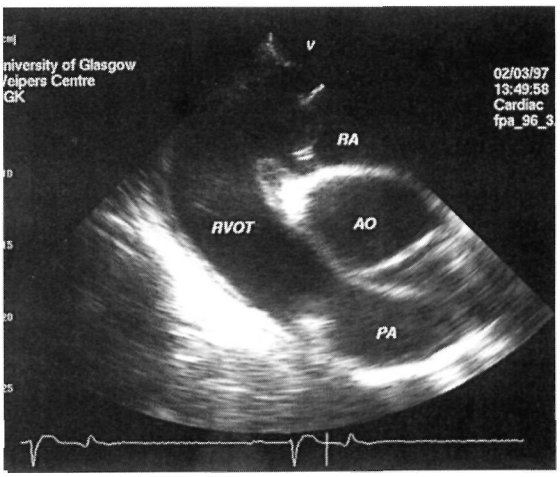


Figure 6-17. Echocardiogram using right parasternal angle view with a slight change in angulation, demonstrating an image of the pulmonary artery. Although the view is similar to that in Figure 6-16, it shows how small changes in angulation and rotation can allow the examiner to highlight adjacent structures. The structures shown include right atrium (RA), aorta (AO), right ventricular outflow tract (RVOT), and pulmonary artery (PA).

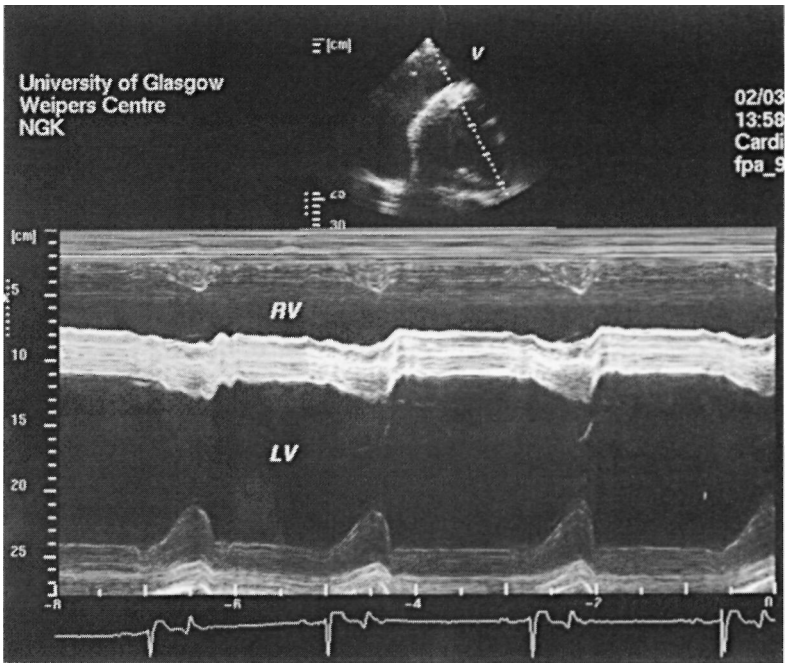


Figure 6-18. Echocardiogram using right parasternal short-axis view at the chordal level, M-mode view. The M-mode cursor is placed in such a fashion that it intersects the interventricular septum and the left ventricular free wall at right angles and transects the left ventricular free wall between the two papillary muscles in diastole and systole. This M-mode view of the left ventricle (LV) is used to obtain measurements of left ventricular dimensions and indices of cardiac function. RV, right ventricle.

Left Parasternal Angled Views

To obtain this view, the transducer has to be in a ventral position relative to the reference position, but frequently placement of the transducer one intercostal space caudal is needed to optimize the image plane. The transducer is angled dorsally and cranially and rotated up to 30 degrees clockwise for this view.

M-MODE ECHOCARDIOGRAMS

M-mode echocardiograms are obtained by using some of the above-described two-dimensional images to guide placement of the axial beam.

Right Parasternal Short-Axis View at the Chordal Level

In this image plane, the transducer is angled cranially to move the left ventricle to the right side of the screen as far as possible. Care is taken to ensure that the plane is horizontal by trying to obtain a near circular appearance of the left ventricular lumen. The M-mode cursor is placed in such a fashion that it intersects the interventricular septum and the left ventricular free wall at right angles and transects the left ventricular free wall between the two papillary muscles in diastole and systole (Fig. 6-18). This M-mode view of the left ventricle is used to obtain measurements of left ventricular dimensions and indices of cardiac function.

Right Parasternal Short-Axis View, Mitral Valve Level

This image plane is used to obtain an M-mode study of the mitral valve (Fig. 6-19). Care is taken that both mitral valve leaflets are visualized at

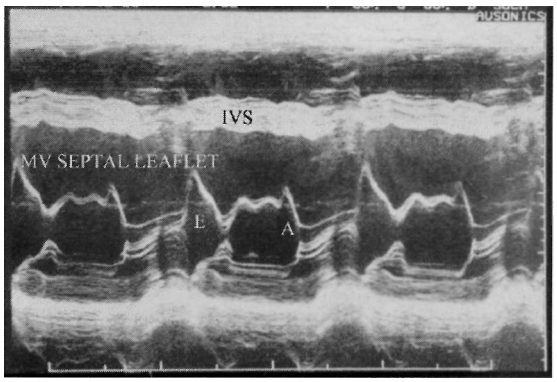


Figure 6-19. Echocardiogram using right parasternal short-axis view, mitral valve level, M-mode. This involves angling the transducer more dorsally than normally needed for achieving the two-dimensional "fish-mouth" appearance of the mitral valve, indicating wide variability when dimensions are measured with the mitral valve as an intracardiac landmark. Structures shown include the interventricular septum (IVS) and the typical motion of the mitral valve leaflets, with the maximum excursion of the mitral valve during early diastole (E) and atrial contraction in late diastole (A) shown.

their maximal excursions during systole and diastole. However, it should be noted that routinely this involves angling the transducer more dorsally than normally needed for achieving the two-dimensional "fish-mouth" appearance of the mitral valve, indicating wide variability when dimensions are measured with the mitral valve as an intracardiac landmark.

Right Parasternal Short-Axis View, Aortic Valve Level

This image plane is used to obtain an M-mode image of one aortic valve cusp that can be clearly seen during systole and diastole (Fig. 6-20). The ensuing M-mode image is used to obtain dimensional and functional information such as the aortic diameter or, as shown in the next picture, the ejection time (Fig. 6-21).

PULSED-WAVE DOPPLER

Pulsed-wave Doppler can be used to obtain hemodynamic information from all four heart valves. It has been shown that accurate measurements of the cardiac output can be made in the standing horse by measuring the velocity time integral of the pulsed-wave Doppler sample of the aortic outflow from the left-hand side and the aortic diameter from the right-hand side. The resulting flow and area information are combined with the heart rate in the formula Q = HR X VTI X A.

For more detailed information, refer to the list of recommended reading. One such measurement of a 5-year-old Thoroughbred gelding is shown in Figures 6-21 and 6-22. The cardiac output measured was ~38 L/min.

Measurements of echocardiograhically displayed structures vary primarily in the manner in which the thick echoes that represent the borders are included within the measurements. The four most commonly applied measurement conventions are the standard convention method, the Penn convention, the American Society of Echocardiography convention, or leading edge method, and the inner edge method.

KEY POINT

Although the leading edge to leading edge method remains the most commonly applied method for M-mode measurements, B-mode measurements are commonly performed using the inner edge method.

Doppler measurement conventions have been described in detail and are widely applied in human cardiology. In horses, Doppler measurements have been done with and without angle correction, and it has been stated that angle correction should not be used because it might lead to overestimation of flow. Velocity time integrals have been measured from the peak velocity lines and the modal velocity lines. The modal velocity has been shown to correlate best with other blood flow measurements.

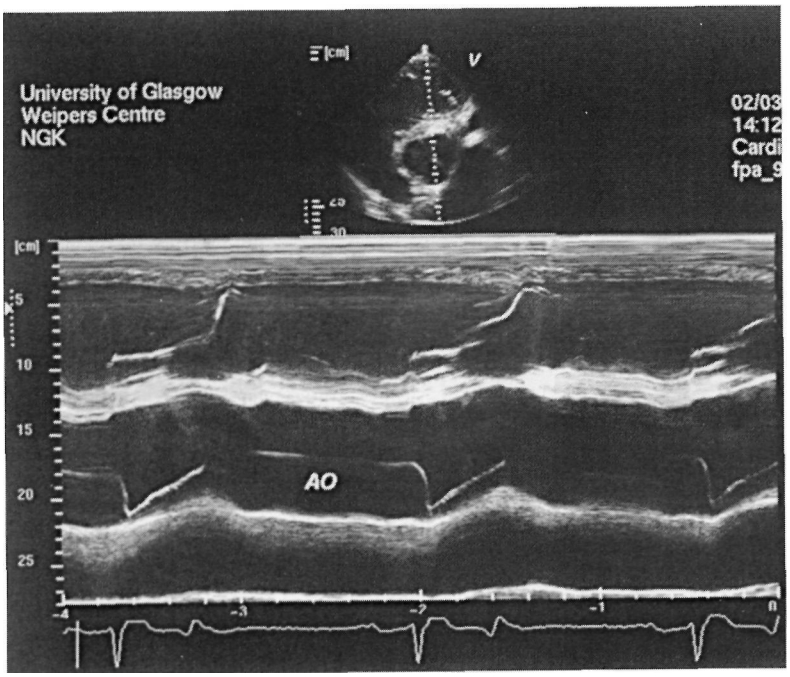


Figure 6-20. Echocardiogram using right parasternal short-axis view, aortic valve level. This image plane is used to obtain an M-mode image of one aortic valve cusp (AO). The image is used to obtain dimensional and functional information such as the aortic diameter or, as shown in Figure 6-21, the ejection time.

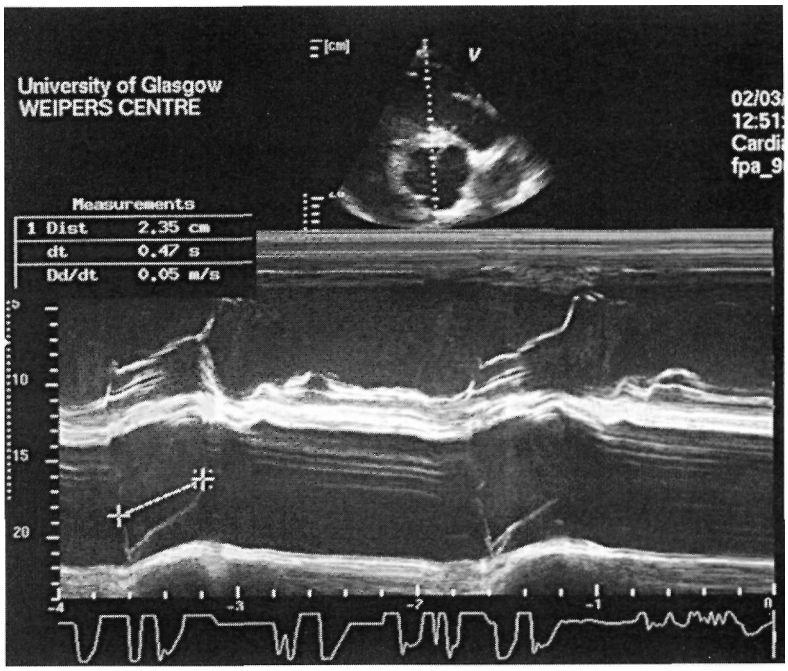


Figure 6-21. Echocardiogram using right parasternal short-axis view, aortic valve level. M-mode view of the aortic valve opening and closing and a measurement of ejection time taken. Above the aortic valve, the tricuspid valve can be seen.

Constant timing of echocardiographic measurements is invaluable to ensure good reproducibility. End-diastolic measurements from M-mode images are taken at the onset of the QRS complex on the overlying ECG. Various definitions for end-systolic measurements have been described, and the method that defines end systole as the point at which the septum reaches its maximum excursion toward the ventricular lumen has been described as the most accurate. The two above-described techniques are the methods used in equine echocardiography today. Timing for B-mode images is usually taken from the ECG or from the frame that contains the relevant information. End diastole is defined as the frame containing the onset of the QRS complex; end systole is defined as either the

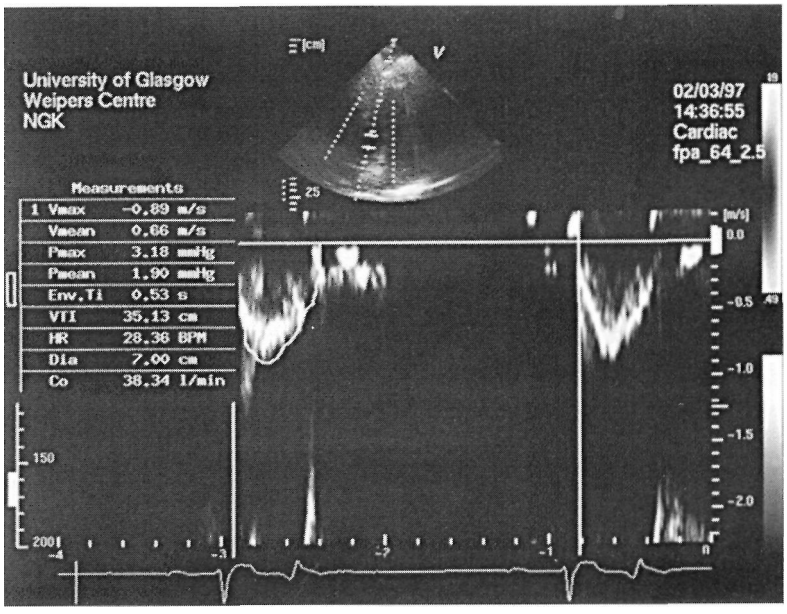


Figure 6-22. Pulsed-wave Doppler picture of aortic outflow with measurements superimposed.

frame with the smallest ventricular lumen or the frame just before opening of the mitral valve. Timing of Doppler measurements is easier to define because one simply follows the flow signal when it is visible above or below the baseline.

Echocardiographic measurements are an accurate way of assessing ventricular dimensions in humans. Studies in horses have shown a good correlation between M-mode echocardiographic measurements at end systole and autopsy measurements. Doppler measurements of the aortic outflow correlate well with measurements obtained with Doppler flow probes and thermodilution. In horses, various authors have shown a good correlation between aortic Doppler measurements and measurements simultaneously obtained with thermodilution. More recent studies suggest that Doppler measurements from transesophageal echocardiography are also useful for monitoring left ventricular systolic performance in anesthetized horses.

Normal cardiac dimensions have been described by a variety of authors, and Table 6-1 lists the normal ranges of the most important measurements in horses weighing about 350 to 550 kg.

Exercise Testing and Telemetry Electrocardiography

In some cases, the significance of resting ECG or auscultatory findings may be difficult to assess. In these cases, exercise or "stress" testing can be of value in determining the prognosis and treatment. Treadmill exercise testing is most valuable because an array of information can be gained under standardized conditions where the workload is precisely known. However, track testing is also useful, particularly in Standardbred trotters and pacers, where systems are commercially available to measure speed and heart rate during exercise (Bauman Speed Puls Equus R, Bauman and Haldi, Switzerland).

We have conducted clinical exercise testing on several hundred Thoroughbred and Standardbred racehorses and on endurance and pleasure horses. The tests are all performed with the treadmill inclined at a slope of 6 degrees (10%), and the horses progress through trotting, cantering, and galloping (or pacing) at speeds up to 12 m/s. It is important that horses are well acclimated to treadmill exercise because apprehension will result in significant elevations in heart rate, which makes interpretation of submaximal heart rate data difficult. We use a protocol of a rapid incremental treadmill test, with a warmup of 3 minutes at 4 m/s, then 90 seconds at 6 m/s, and then 1-minute steps each at 8, 10, 11, and 12 m/s or until the horse cannot maintain pace with the treadmill. Most fit Thoroughbred horses can complete the test up to 11 m/s.

Recording of the ECG by telemetry (Fig. 6-23) is useful to determine whether abnormalities occur and can be most easily visualized in the first 30 to 60 seconds after the horse stops fast exercise. Alternatively, much information can be gained from the horse's heart rate response to exercise,

TABLE 6-1. Measurements of Cardiac Dimensions from Six Studies that Used Similar Standardized Images and Measurement Guidelines

Measurement	Long et al.. 1992	Patteson et al.. 1995a	Slater and Herrtage, 1995	Rewel, 1991	Patteson et al.. 1995b	Authors' Data
IVSd (cm)	3.02 ± 0.39	2.85 ± 0.28	2.8 ± 0.2	3.27 ± 0.4	2.61 ± 0.28	3.19 ± 0.33
IVSs (cm)	4.55 ± 0.55	4.21 ± 0.46	4.6 ± 0.5	4.1 ± 0.6	3.98 ± 0.27	4.32 ± 0.32
LVFWd (cm)	—	2.32 ± 0.38	2.5 ± 0.3	2.77 ± 0.43	2.02 ± 0.2	2.51 ± 0.33
LVFWs (cm)	3.96 ± 0.63	3.85 ± 0.41	3.8 ± 0.3	3.33 ± 0.53	3.7 ± 0.36	3.73 ± 0.51
LVIDd (cm)	11.9 ± 0.71	11.92 ± 0.76	11.2 ± 0.8	11.73 ± 0.8	11.47 ± 0.85	11.76 ± 0.66
LVTDs (cm)	7.35 ± 0.72	7.45 ± 0.62	7.3 ± 0.8	8.33 ± 0.7	7.2 ± 0.73	8.07 ± 0.69
Aod (cm)	8.5 ± 0.51	7.95 ± 0.53	7.5 ± 0.6	7.56 ± 0.7	—	7.69 ± 0.28
HR (bpm)	—	—	—	—	36.5 ± 4.8	35.77 ± 2.45

IVSd, Interventricular septum (diastole); IVSs, interventricular septum (systole); LVFWd, left ventricular free wall (diastole); LVFWs, left ventricular free wall (systole), LVIDd, left ventricular internal diameter (diastole);, LVIDs, left ventricular internal diameter (systole); Aod, aortic diameter; HR, heart rate.

Long, K.J., Bonagura, J.D., and Darke, P.G.: Standardized imaging technique for guided M-mode and Doppler echocardiography in the horse. *Equine Vet J* 24:226, 1992; Patteson, M.W., Gibbs, C, Wotton, P.R., and Cripps, P.J.: Echocardiographic measurements of cardiac dimensions and indices of cardiac function in normal adult Thoroughbred horses. *Equine Vet J Suppl* 19; 18-27, 1995a; Patteson, M.W., Gibbs, C., Wotton, P.R., and Cripps, P.J.: Effects of sedation with detomidine hydrochloride on echocardiographic measurements of cardiac dimensions and indices of cardiac function in horses. *Equine Vet J Suppl* 19:33-37, 1995b; Rewel, A.: Vergleichende Messungen von Herzdimensionen unci Bewegungsmustern bei Warmblut-Sportpferden mit Hilfe de M-mode Echokardiographie. *Dissertation* Tierarztliche Hochschule Hannover, 1991; Slater J.D. and Herrtage, M.E.: Echocardiographic measurements of cardiac dimensions in normal ponies and horses. *Equine Vet J Suppl* 19:28-32, 1995.

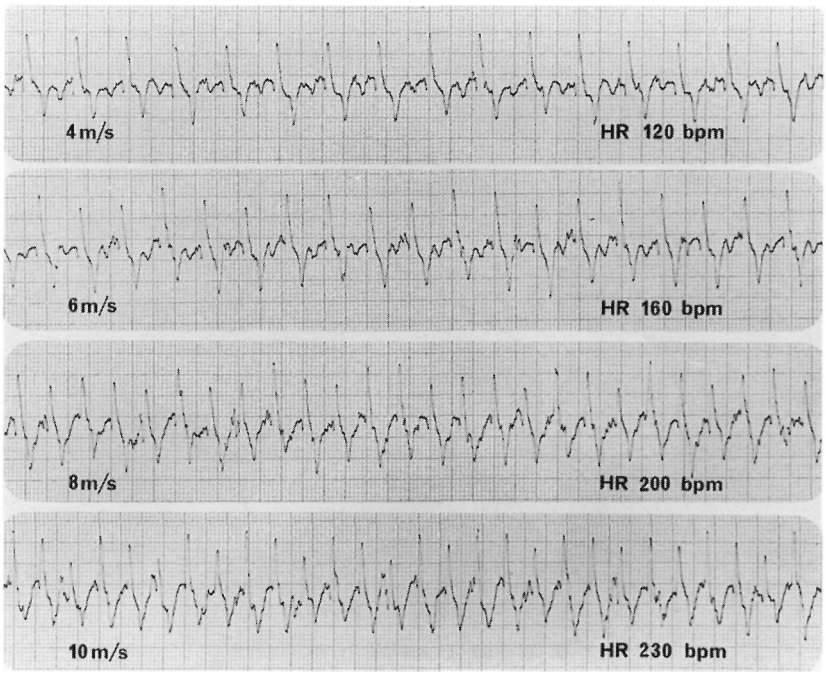


Figure 6-23. Telemetric ECG recording from a 3-year-old Thoroughbred horse during exercise on a treadmill at a slope of 6 degrees (10%) and at speeds varying from 4 to 10 m/s. Recording was made at 25 mm/s and an amplitude of 1 mV/cm. HR, heart rate.

and the heart rate can be measured using a cardiotachometer, of which several types are commercially available (EQB, Philadelphia, PA; Polar, Copenhagen, Denmark). The heart rate responses to exercise of a normal fit Thoroughbred racehorse and a horse with a grade 4 systolic cardiac murmur, localized over the mitral valve region, are shown in Figure 6-24.

Cardiovascular Diseases

CARDIAC ARRHYTHMIAS

Arrhythmias of various types are quite common in the horse, although most may not be of pathologic significance. Investigation of arrhythmias must include an ECG. Services such as Cardiopet are available that allow the ECG to be transmitted by telephone for analysis. Most of the "common" arrhythmias are regularly occurring, with the normal heart rhythm being regularly interrupted by an absence of a beat.

HISTORY AND PRESENTING SIGNS

- Poor racing performance
- Dyspnea
- Slow recovery after exercise
- Prepurchase or routine examination finding

CLINICAL FINDINGS AND DIAGNOSIS

- Arrhythmias affecting cardiac function during exercise will usually be pronounced and cause irregular heart sounds.
- With some arrhythmias (e.g., second-degree AV block), there may be no history of a presenting problem, but an abnormality of rhythm may be noted on clinical examination.

KEY POINT

An ECG recording should be performed, and it may be useful to vary the degree of vagal tone during recording by application of a twitch to decrease the heart rate and by stimulating the horse so that the heart rate increases.

A classification of the major arrhythmias and the clinical and ECG findings is presented in Table 6-2.

DIFFERENTIAL DIAGNOSIS

- Second-degree AV block
- Third-degree AV block

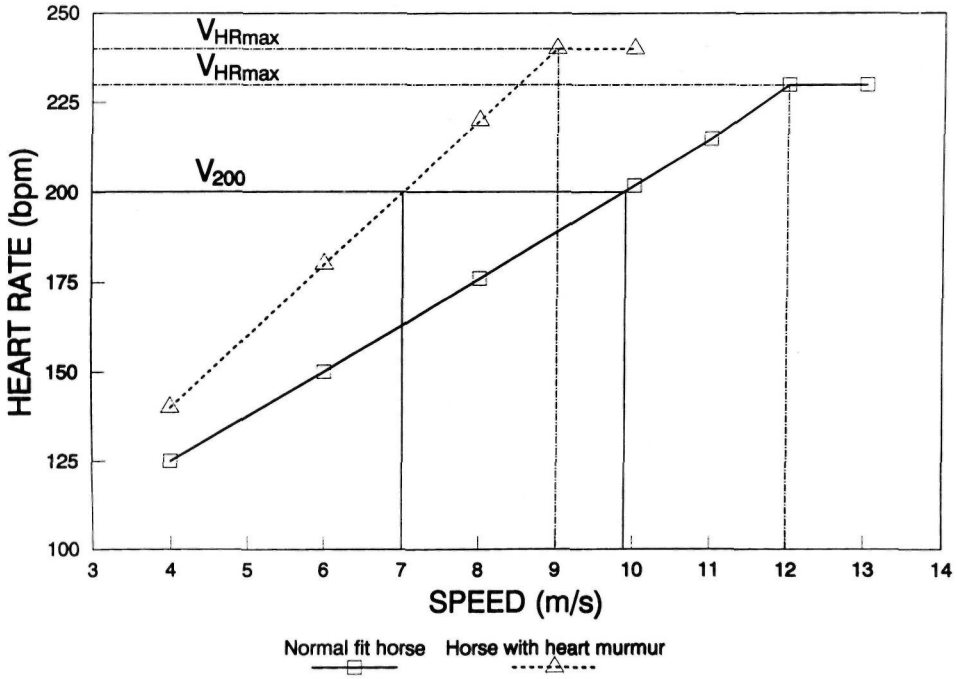


Figure 6-24. Heart rate response to exercise in two horses on a high-speed treadmill during an incremental exercise test at a slope of 6 degrees (+10%). Note the elevated heart response at all stages in the horse with the heart murmur compared with the normal fit Thoroughbred. The steeper slope of the heart rate versus speed line is also quite obvious. Two useful reference points are the speed (velocity) at a heart rate of 200 bpm (V_{200}) and the speed at maximum heart rate (V_{HRmax}) that are shown on the graph. In the normal fit horse, the V_{200} is 9.8 m/s and the V_{HRmax} is 12.0 m/s. In contrast, the horse with the heart murmur has a V_{200} of 7.0 m/s and a V_{HRmax} of 9.0 m/s.

- Sinoatrial block
- Atrial fibrillation
- Paroxysmal atrial fibrillation
- Atrial premature contractions
- Ventricular premature contractions
- Sinus arrhythmia
- Ventricular tachycardia
- AV dissociation

TREATMENT

For most arrhythmias, little can be done in the way of therapy. Some cases, where there is a viral myocarditis, will resolve with rest. Others may require various forms of therapy. Specific treatments are indicated in the following section, which provides details of each common arrhythmia.

Atrioventricular Block

KEY POINT

AV block, of which there are three types, is the most common of the atrial arrhythmias.

First-degree AV block is diagnosed on the basis of progressive lengthening of the PR interval to values greater than 0.50 second. This upper limit of normal is arbitrarily defined, and the PR interval will lengthen with increasing age. No abnormalities are detected on auscultation. First-degree AV block has no pathologic significance.

Second-degree AV block is the most common arrhythmia and is characterized by a P wave that is not followed by a QRS complex or T wave (Fig. 6-25). There are two types of second-degree AV block, the most common being associated with a progressive lengthening of the PR interval until the blocked beat occurs (Wenkebach type). In the other type of AV block, the PR interval is regular in length. Second-degree AV block usually occurs regularly, every 4 to 8 beats, but it may be intermittent. On careful auscultation, the atrial (S_A or S_4) sound can be heard without the following S_1 and S_2 sounds.

KEY POINT

Although some clinicians regard second-degree AV block as abnormal, most would

TABLE 6-2. Classification of Arrhythmias and their Clinical and ECG Findings

Arrhythmia	Clinical Findings	ECG Findings
Supraventricular Rhythm Disturbances		
Sinus bradycardia	Slow regular rhythm	Long RR interval, normal P-QRS-T
Sinus tachycardia	Rapid regular rhythm (60-90 bpm)	Short RR interval, normal P-QRS-T
Sinus arrhythmia	Slow/normal heart rate Variable S_1-S_1 interval	Variable RR interval ± wandering pacemaker (variable P-wave morphology)
Sinoatrial block	Slow/normal heart rate Regular rhythm with pauses	Regular underlying rhythm with absence of P waves for 2 PP intervals
Sinoatrial arrest	Slow/normal heart rate Regular rhythm with long pauses	Regular underlying rhythm with absence of P waves for >2 PP intervals
First-degree AV block	Slow/normal heart rate Regular rhythm	Prolonged (>0.5) PR interval
Second-degree AV block	Slow/normal heart rate Regular irregular rhythm with occasional dropped beats Isolated S_4 sounds Pulse deficit	Occasional P wave not followed by QRS Mobitz type I (Wenckebach) progressive lengthening of PR preceding the dropped beat Mobitz type II Constant PR
Third-degree AV block	Slow heart rate Regular rhythm S_4s have no relation to S_1-S_2	No evidence of conduction (AV dissociation) junctional or ventricular escape rhythms
Atrial premature complexes	Premature beat ± compensatory pause	Premature P wave that may or may not be followed by a normal QRS-T
Atrial tachycardia	Fast heart rate Regular or irregular Paroxysmal or sustained	P waves may or may not differ from normal sinus P-waves, normal QRS complex
Atrial fibrillation	Normal or fast heart rate Irregularly irregular rhythm Paroxysmal or sustained No S_4 heart sounds of variable intensity Pulse of variable quality	Irregular RR interval Absence of P-waves f fibrillation waves
Junctional tachycardia	Fast heart rate Regular or irregular	P waves unrelated to QRS but can be retrograde normal QRS
Ventricular Rhythm Disturbances		
Ventricular premature complexes	Premature S_1 + S_2 (loud) Usually followed by compensatory pause ± pulse deficit	Premature QRS not preceded by P wave QRS morphology differs from normal T wave of VPC oriented in opposite direction of its QRS
Ventricular tachycardia	Rapid inappropriate heart rate ± regular rhythm	4 or more consecutive VPCs Sustained or paroxysmal uniform VT: single VPC morphology Multiform VT: more than one VPC morphology
Accelerated idioventricular rhythm	Slightly raised heart rate (40-60 bpm) Regular rhythm S_4 may not be heard, if heard it has no relationship to S_1 + S_2	Abnormal QRS No relationship between QRS and P waves Ventricular rate slightly faster than sinus rate
Ventricular fibrillation	No pulse No clear heart sounds	No clear complexes Bizarre undulating baseline (if horse is standing—jump clear!)

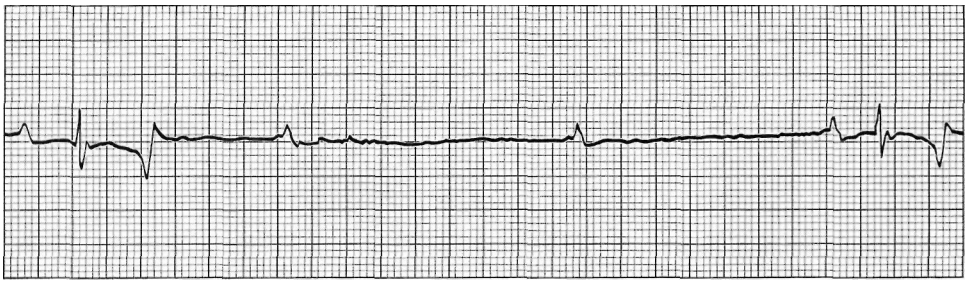


Figure 6-25. ECG tracing (lead II) from a horse with second-degree AV block showing isolated P waves. Note the progressive lengthening of the PR interval in the beats before the block. Recording was made at 25 mm/s and an amplitude of 1 mV/cm.

regard this block as normal and a consequence of the high vagal tone in the resting horse.

The block always disappears when the heart rate increases. However, there have been some studies demonstrating histopathologic changes in the myocardium of horses with second-degree AV block. From the ECG findings in several thousand horses that we have undertaken, we believe that if this block occurs frequently during a trace and is found in a young horse, there should be an index of suspicion about the pathologic significance.

Third-degree AV block is quite rare in the horse and represents a complete block in conduction between the atria and the ventricles. The atria beat at a normal sinus rate, whereas the ventricles have a slower rate. On auscultation, this block is characterized by a very low heart rate (15-20 beats/min) and a prominent jugular pulse. In some cases, third-degree AV block can be the cause of syncopal attacks.

There is no treatment for the various types of AV block, and the prognosis will vary with the type found. Third-degree AV block obviously has a poor prognosis for athletic performance and tends not to resolve with time. First- and second-degree AV blocks are usually of no pathologic significance.

Atrial Fibrillation

Atrial fibrillation is an arrhythmia most commonly found in Standardbred horses, although it also has been found in a variety of other breeds, including Clydesdales. In many cases it can occur during a race and causes a severe decrease in the horse's performance.

KEY POINT

On auscultation, the condition is characterized by an irregular heart rhythm with a series of rapid heart beats followed by no heart sounds. These findings are unusual and a presumptive diagnosis can be made by auscultation alone.

If the pulse rate is taken at the same time as the heart rate, there may be a pulse deficit. The resting heart rate is usually higher than normal and may range from 30 to 100 beats per minute. In some cases there may be signs of an increased jugular pulse, indicating congestive heart failure.

If an arrhythmia indicates the probability of atrial fibrillation, an ECG recording is the method of choice for diagnosis. The ECG is characterized by the presence of f, or flutter, waves between irregularly spaced QRS complexes (Fig. 6-26).

KEY POINT

If there are no signs of congestive heart failure, often it is possible to correct the abnormality using quinidine sulfate (Treatment No. 98) administered by stomach tube.

Most clinicians use a test dose of quinidine sulfate at a dose rate of 10 to 15 mg/kg before the treatment schedule. Quinidine usually is available in 200-mg tablets, which should be crushed and suspended in water. Although there are a number of possible protocols for quinidine sulfate administration, we have found that a simple and effective technique is a dose of 10 g for an adult horse (22 mg/kg) given every 2 hours until there is cardioversion to normal sinus rhythm. The total amount of quinidine sulfate usually required is 30 to 60 g. However, in some horses, a dose of up to 90 g may be required. Once the total dose has exceeded 60 g, the risk of toxicity is increased, and considerable care is required. If the horse fails to convert, treatment can be restarted the next day, and some horses may require several days of therapy before conversion. In a few cases where blood levels have been measured, it appears that

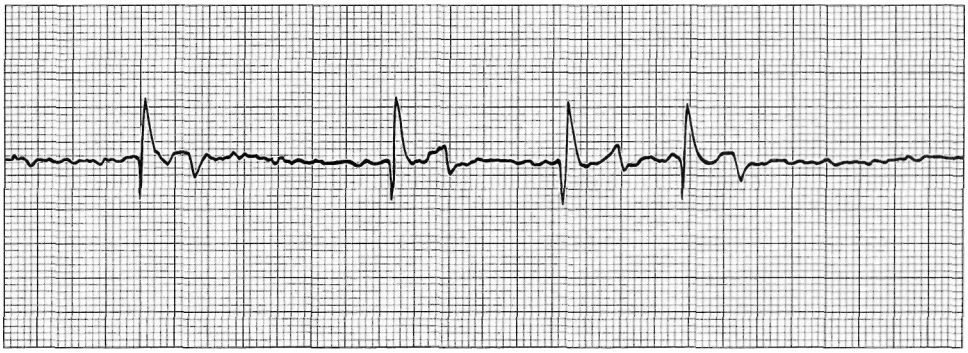


Figure 6-26. ECG tracing (lead Y) from a 3-year-old Standardbred horse presented with poor racing performance. Note the irregular spacing of the QRS complexes and the presence of flutter waves. This is typical of atrial fibrillation. Recording was made at 25 mm/s and an amplitude of 1 mV/cm.

at least some of the variability in response is due to poor absorption of the administered drug. Plasma concentrations in the range 2 to 4 μg/mL are usually necessary for cardioversion, and toxicity is found at concentrations greater than 5 μg/mL.

When quinidine treatment of atrial fibrillation is successful, most horses return to previous athletic capacity once sinus rhythm has been reestablished. Toxicity from quinidine therapy is unusual, but we have had one horse die in ventricular fibrillation after a total dose of 80 g. Most commonly, horses will show signs of dullness or depression and sweating, and there may be gastrointestinal signs, including colic and diarrhea. Nasal congestion also has been reported as a common sign of toxicity. Quinidine causes a substantial increase in heart rate, and after doses of 40 to 50 g, heart rates around 100 beats per minute are quite common.

Quinidine gluconate, given intravenously, has been recommended in cases where the history suggests a recent (less than 1 week) onset of the problem. Quinidine gluconate is given at a dose rate of 1.5 mg/kg every 10 minutes until cardioversion occurs, for a maximum of six treatments.

Paroxysmal Atrial Fibrillation

KEY POINT

Paroxysmal atrial fibrillation is a form of atrial fibrillation that disappears spontaneously, usually within several hours of its occurrence.

It is most common in racehorses and is responsible for a dramatic decrease in performance during a race. Monitoring of the ECG in the immediate postrace period in horses that have suffered poor racing performance has shown that paroxysmal atrial fibrillation is more common than was previously thought. The greatest difficulty in cases of paroxysmal atrial fibrillation is in making the diagnosis. Horses that are examined the day after racing because of poor performance may have normal ECG findings despite suffering atrial fibrillation at the time of the race. Therefore, horses with racing form reversals or poor performance that have arrhythmias on auscultation after competing should have an ECG performed soon after the race or event.

Paroxysmal atrial fibrillation also has been reported in newborn foals. There is no treatment for the condition, which usually resolves spontaneously. Some cases may go on to suffer persistent atrial fibrillation, whereas others may recur intermittently.

Atrial Premature Contractions

KEY POINT

Atrial premature contractions cause an arrhythmia due to generation of impulses in an ectopic pacemaker located in the atria.

This type of arrhythmia is quite rare in the horse. Horses with atrial premature contractions usually are presented for poor racing performance.

Auscultation will reveal a largely regular rhythm with an occasional premature beat. An ECG is necessary for diagnosis, and a typical example of the problem is shown in Figure 6-27. There is little that can be done apart from rest for 3 to 6 months followed by a further ECG to assess progress. The condition may be of little clinical significance if it occurs as an isolated event, but

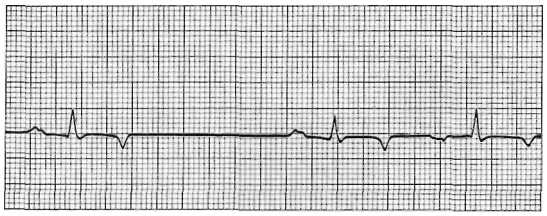


Figure 6-27. ECG tracing from a 4-year-old Thoroughbred horse with an isolated atrial premature contraction (lead II). There were no other ECG abnormalities, and the condition did not appear to be associated with cardiac pathology. Recording was made at 25 mm/s and an amplitude of 1 mV/cm.

if it is more frequent during an ECG recording, it may reflect atrial pathology.

Atrioventricular Dissociation

AV dissociation can occur in cases of parasystole, third-degree AV block, and ventricular tachycardia. If the dissociation is intermittent, an irregular arrhythmia is usually heard on auscultation. A tracing from an 8-year-old endurance horse presented because of poor heart rate recoveries is presented in Figure 6-28, demonstrating some unrelated P waves and QRS complexes.

KEY POINT
Problems that lead to AV dissociation are quite severe, and the prognosis should be guarded.

Rest for 8 to 12 weeks followed by rerecording of the ECG should be undertaken to assess changes in the condition.

Bundle-Branch Block (Intraventricular Block)

This disorder is difficult to diagnose, and there is disagreement about whether the condition truly occurs in the horse. It does not result in any arrhythmia but is characterized by a prolongation of the QRS complex duration to greater than 0.16 second.

KEY POINT
Poor racing performance is the main clinical sign of bundle-branch block, and therefore it needs to be distinguished from other causes of poor performance.

We have seen several cases where sequential ECG recordings over 1 to 2 months, indicating a prolongation of the QRS complex, have enabled a diagnosis to be made. This disorder appears not to resolve and has a very poor prognosis for successful athletic performance when exercise at maximal intensities is required.

Intra-atrial Block

KEY POINT
As with intraventricular block, intra-atrial block does not cause any arrhythmia, and there are no abnormalities on auscultation. Again, it is a controversial diagnosis.

It can be diagnosed only by an ECG. The main clinical sign is a reduction in racing performance. Clinical examination and auscultation do not reveal any abnormalities. The ECG will reveal a P-wave duration in lead II of more than 0.17 second and a P-wave peak interval of more than 0.08 second (Fig. 6-29).

Intraatrial block cannot be treated, and in our experience there have been no cases of spontaneous resolution. The condition appears to have an adverse effect on athletic performance.

Sinoatrial Block

KEY POINT
Sinoatrial block is characterized by a regularly "dropped" beat and on auscultation may appear similar to second-degree AV block.

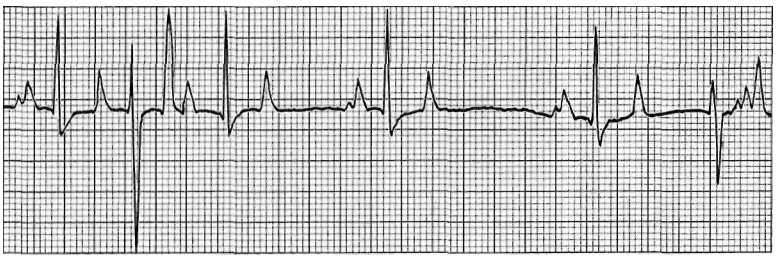


Figure 6-28. ECG tracing (lead II) from an 8-year-old endurance horse presented because of poor heart rate recoveries. Note the AV dissociation. Recording was made at 25 mm/s and an amplitude of 1 mV/cm.

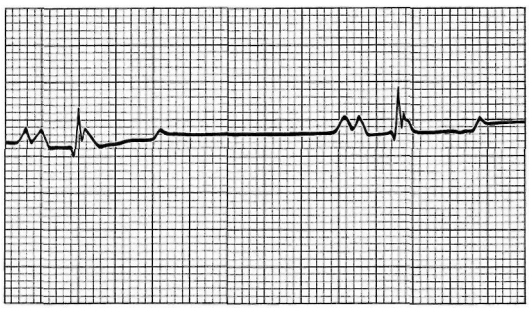


Figure 6-29. ECG tracing (lead II) from a 4-year-old Thoroughbred racehorse presented with poor racing performance. Note the prolonged P-wave duration (0.19 second) and P-wave peak interval (0.09 second), which are typical of intraatrial block.

In contrast to second-degree AV block, no atrial (S_4) sound can be heard when the block occurs. Diagnosis is made by ECG in which there is a doubling of the normal PP interval when the block occurs so that there is a gap on the ECG trace with no P wave, QRS complex, or T wave (Fig. 6-30). This may be difficult to distinguish from sinus arrhythmia (a normal phenomenon), but in such situations, the PP interval is not exactly doubled. Sinoatrial block is abolished when the heart rate is elevated (as during exercise).

Most veterinarians regard sinoatrial block as a normal finding, reflecting increased vagal tone. There are no studies to indicate that the problem induces lowered cardiac output during exercise and the likelihood of poor performance.

It is of value to assess the heart rate response to a graded incremental exercise test, as described previously, and to determine whether ECG abnormalities occur during exercise. There is no effective treatment for this condition.

Sinus Arrhythmia

The horse's heart rate is normally quite regular, with little variation in the interval between successive heart beats. In some cases, there may be variation in the interval between beats, and on the ECG tracing the time between the RR intervals is somewhat irregular.

KEY POINT

In some horses we have noted pronounced sinus arrhythmia during the first 1 to 2 minutes of recovery after treadmill exercise tests (Fig. 6-31).

Our impression is that in horses where this response is pronounced, the heart rate response to exercise is higher than normal. Further work is necessary to determine if there is an association between the finding of marked postexercise sinus arrhythmia and reduced performance capacity.

Ventricular Premature Contractions

The problem of occasional ventricular premature contractions (VPCs) is a relatively common cause of cardiac arrhythmias in the horse. On auscultation, the abnormal beat usually results in a louder S_1.

KEY POINT

A VPC is easily diagnosed on ECG by the characteristic premature nature of the beat in relation to normal sinus rhythm and the unusual QRS waveform, as shown in Figure 6-32.

Our experience is that VPCs are of clinical significance and indicate myocardial irritation. It may be useful in cases where there are only one or two isolated VPCs on a resting ECG to perform a telemetric ECG during exercise to determine whether more abnormal beats occur and whether the heart rate response to exercise is abnormal. There may be an association with some upper respiratory tract infections, particularly equine influenza.

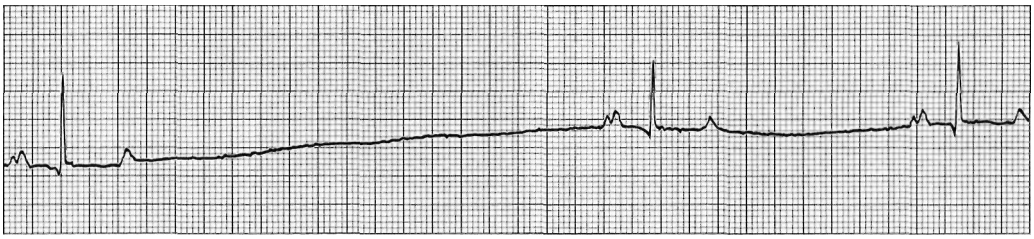


Figure 6-30. ECG tracing (lead II) from a horse with a sinoatrial block showing the doubling of the RR interval, with no P wave, QRS complex, or T wave. Recording was made at 25 mm/s and an amplitude of 1 mV/cm.

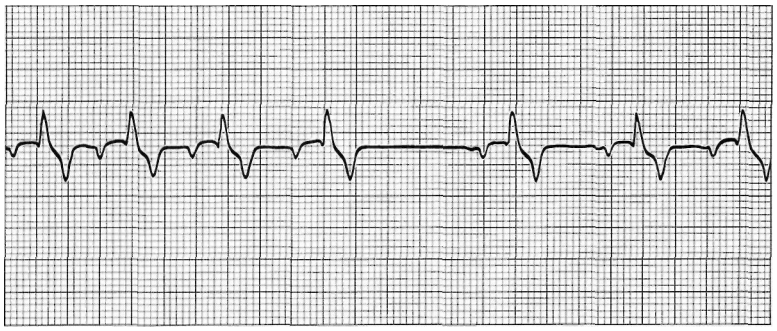


Figure 6-31. Telemetry ECG recording from a horse at the completion of a treadmill exercise test. Note the marked sinus arrhythmia in the period after exercise. Recording was made at 25 mm/s and an amplitude of 1 mV/cm.

It may be useful to rest horses with VPCs 8 to 12 weeks, because in many cases the arrhythmia will resolve.

Ventricular Tachycardia

Ventricular tachycardia occurs most commonly in the horse in a paroxysmal form.

KEY POINT

The problem is characterized by a very much higher heart rate than normal and a characteristic abnormal ECG tracing.

The QRS complexes are abnormal in appearance, and P waves may not be apparent. The T waves are large and follow immediately on from the QRS complex. These features are shown in an ECG tracing from a 3-year-old Standardbred pacer in Figure 6-33, where the waveform changes suddenly to normal sinus rhythm. Ventricular tachycardia is a particularly severe arrhythmia but in many cases is secondary to other metabolic disturbances, such as electrolyte and acid-base abnormalities. We have seen this problem most commonly under general anesthesia and in the immediate postexercise period after high-intensity exercise.

Wolff-Parkinson-White Syndrome

The condition, also known as preexcitation syndrome, is unusual in the horse and usually occurs together with other abnormal ECG findings.

KEY POINT

The condition is characterized by a very short PR interval, together with a prolonged QRS complex duration.

It can often appear in an intermittent form, interspersed by normal sinus rhythm. The pathologic significance is debatable, but if the problem is persistent, it is likely to have significant deleterious effects on athletic performance.

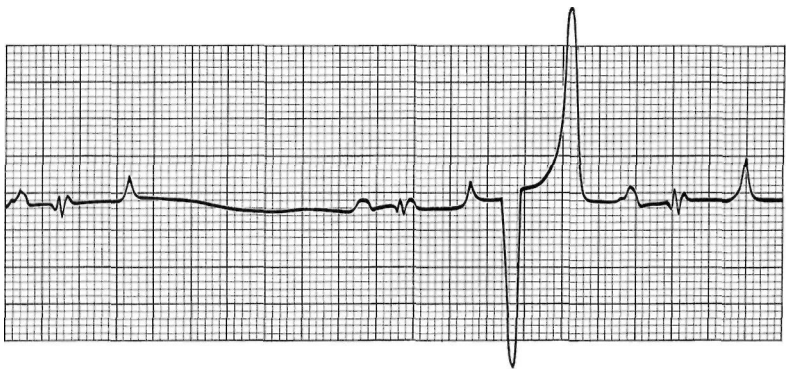


Figure 6-32. ECG tracing (lead II) from the 5-year-old Standardbred gelding presented because of poor racing performance. Note the bizarre QRS complex in an otherwise normal ECG recording. This is typical of ventricular premature contractions. Recording was made at 25 mm/s and an amplitude of 1 mV/cm.

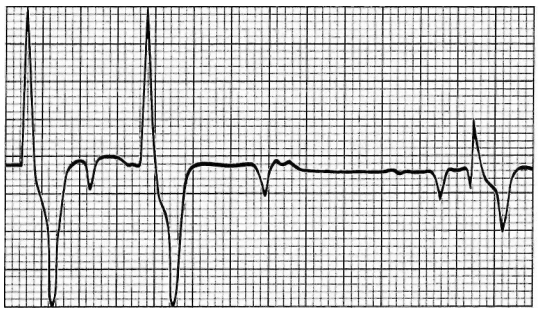


Figure 6-33. ECG tracing (Y lead) from a 3-year-old Standardbred pacer presented for routine evaluation of fitness by treadmill exercise testing. The ventricular tachycardia occurred in a paroxysmal form, interspersed by periods of normal sinus rhythm, immediately after exercise. Recording was made at 25 mm/s and an amplitude of 1 mV/cm.

CONGENITAL HEART DISEASES

Congenital heart disease is relatively common in horses, but many cases do not become evident until the horse is first trained or at the time of an insurance or prepurchase examination.

Ventricular Septal Defects

Ventricular septal defect (VSD) is the most common congenital cardiac abnormality in the horse. It also can occur together with a range of other less common cardiac abnormalities, such as tetralogy of Fallot, patent ductus arteriosus, atrial septal defects, and tricuspid atresia. Simple VSD may result in few abnormal findings, but there is usually a significant systolic heart murmur, heard best over the right hemithorax in the third to fourth intercostal spaces just dorsal to the sternum. VSDs, if small, may not result in adverse clinical signs until the horse is required to perform competitively. Some horses have even been reported to race successfully with small VSDs, especially early in their career. Signs of congenital cardiac disease will vary with the extent of the defect and amount of work required from the horse.

Most VSDs occur relatively high in the heart, immediately below the aortic valve and therefore can sometimes be difficult to visualize on echocardiography, particularly if the lesion is small. It is also easy for a clinician to miss this problem on clinical examination, particularly if a routine auscultation of the right chest is not undertaken.

Color-flow echocardiography is usually capable of mapping out the site of the lesion, and pulsed-flow Doppler echocardiography can help in assessing its severity by measuring the velocity of the stenotic jet. With large defects, clinical signs are quite pronounced and the foals are hypoxemic. These defects are easily seen on an echocardiogram (Fig. 6-34).

Atrial Septal Defects

Many atrial septal defects do not produce clinical signs, and there may not be a heart murmur present because of the low pressure and blood flow in this region. In large septal defects, pulmonary hypertension can occur, and right-sided heart failure may be the result. Some cases may show splitting of S_2 on auscultation. This also may be heard in cases of pulmonic stenosis.

Tetralogy of Fallot

This is an uncommon complex congenital cardiovascular disorder that consists of VSD, dextrorotated aorta, pulmonic stenosis, and right ventricular hypertrophy. There is often right-to-left shunting of blood through the septal defect, and arterial hypoxemia is severe.

Tricuspid Atresia

In combination with tricuspid atresia, there is an atrial septal defect and right ventricular hypopla-

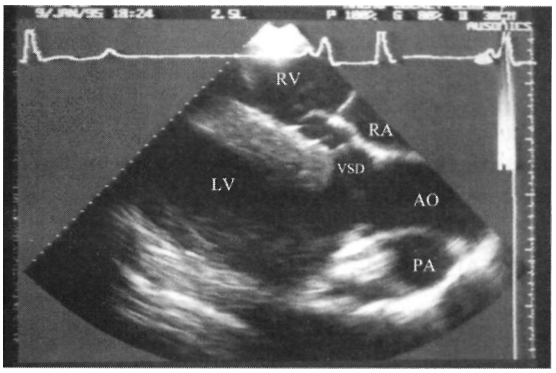


Figure 6-34. Echocardiogram obtained from a 4-year-old Thoroughbred horse with a ventricular septal defect. Structures shown include the right ventricle (RV), right atrium (RA), the ventricular septal defect (VSD), left ventricle (LV), aorta (AO), and pulmonary artery (PA). Note the large ventricular septal defect in the membranous portion of the interventricular septum. This particular horse raced successfully as a 2 year old (won two class 1 races) but had a record of decreasing performance over the next 2 years.

sia. The cases reported also have included a VSD and in some cases a patent ductus arteriosus.

Foals are usually poorly grown, and there is severe hypoxemia. Because of the range of anomalies, there is a loud murmur, usually systolic, that radiates widely.

Patent Ductus Arteriosus

The ductus arteriosus, a fetal connection between the pulmonary artery and the aorta, is usually patent for a short period after birth. However, in almost all foals, the ductus closes within the first 24 hours after birth. A persistent ductus will result in a loud "machinery" type of murmur that radiates extensively on auscultation. A systolic murmur is commonly present for several months after birth in foals, and some clinicians mistake this murmur for a ductal murmur. Patent ductus arteriosus is unusual in the horse and is associated most commonly with a variety of other congenital cardiac abnormalities. Because of this, it carries a poor prognosis.

One finding that can be confusing for the clinician inexperienced in examining foals is that a systolic murmur often is present from shortly after birth until the foal is 3 to 4 months old. This murmur is not associated with a patent ductus arteriosus but appears to be a functional murmur, similar to but often louder than that found in fit racehorses. The murmur is usually localized well forward on the left chest wall and is not of clinical significance.

HISTORY AND PRESENTING SIGNS

- Dyspnea
- Poor growth rate
- Exercise intolerance
- Cyanosis
- Jugular vein distention
- No presenting signs

CLINICAL FINDINGS AND DIAGNOSIS

- With most congenital cardiac abnormalities, a murmur is heard. In some cases, this murmur may be loud and continuous (e.g., persistent patent ductus arteriosus) or may be more localized, as in the case of VSDs.
- Careful auscultation to determine the relation of the murmur to the phase of the cardiac cycle is important if an accurate diagnosis is to be made.
- The resting heart rate will provide a broad guide to the degree of the problem. Horses with congenital cardiac problems that cause little cardiac dysfunction or arterial hypoxemia will have a normal resting heart rate, whereas those in which there is severe cardiac dysfunction will have rapid heart rates.
- Careful note should be made of signs of jugular venous distention, which can indicate cardiac decompensation.
- Echocardiography often is useful to define problems such as septal defects. However, considerable experience is necessary to obtain good-quality images and for interpretation of changes in chamber dimensions.
- Arterial blood gas sampling is often useful to determine the extent of arterial desaturation. In foals, this can be accomplished via the great metatarsal artery with the foal in lateral recumbency (see Chapter 9). In adults, arterial blood samples can be obtained from the carotid artery. Normal arterial blood gas values together with details of sampling are discussed in Chapters 5 and 9.
- Recording of the ECG is not helpful in many cases, but where there is cardiac enlargement, gross changes in electrical axis will be demonstrable, even using a frontal lead system.
- Cardiac catheterization may be performed at specialist clinics and institutions.
- Catheterization of the right side of the heart, with measurement of central venous pressure and pressures in the right atrium, right ventricle, and pulmonary artery, can be of considerable use in reaching a diagnosis.

DIFFERENTIAL DIAGNOSIS

- Ventricular septal defect
- Persistent patent ductus arteriosus
- Atrial septal defect
- Tetralogy of Fallot
- Tricuspid valve atresia

TREATMENT

In the absence of an increased jugular pulse and other signs of congestive heart failure, the prognosis for survival may be quite good. If the arterial blood gases are normal and there is no evidence of cardiac enlargement on echocardiography, horses can go on to lead normal lives. However, with most congenital cardiac abnormalities, the horse will not be a useful athlete.

ACQUIRED HEART DISEASE

Valvular Heart Disease

Although stenoses of the various heart valves have been reported in individual case studies, such lesions are unusual in clinical practice.

KEY POINT

Most valvular disorders involve insufficiency rather than stenosis. The most common abnormalities that lead to clinical signs affect the mitral and aortic valves.

HISTORY AND PRESENTING SIGNS

- Decreased exercise tolerance
- Poor recovery rates after exercise

CLINICAL FINDINGS AND DIAGNOSIS

- Heart murmurs are heard in all cases and are usually grade II or III if there are no overt signs of heart failure.
- Because of the large cardiac reserve of the horse, heart failure is uncommon and is usually found toward the end stage of disease.

KEY POINT

Right-sided heart failure is more common than left-sided failure and is manifest by prominent jugular vein distention and possibly edema of the ventral abdomen.

- Left-sided heart valve lesions are found more commonly than those on the right side of the heart.
- Echocardiography is useful, in gross valvular abnormalities, to determine the extent of the changes (Fig. 6-35). Unfortunately, in most horses in which valvular disorders are suspected, nothing abnormal may be found on echocardiography. However, it is always useful to determine chamber sizes so that it is clear whether compensatory enlargement has occurred. Doppler echocardiography can be of use in demonstrating the flow disturbances across the valves.
- ECG examination is not usually of value unless there is gross cardiac enlargement, in which there will be a shift in the cardiac axis.

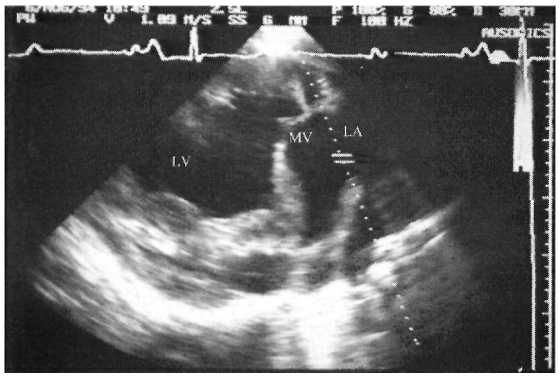


Figure 6-35. Echocardiogram obtained from a 5-year-old Standardbred horse with bacterial endocarditis. The view is that from the left parasternal long axis, left ventricular inlet. Structures shown are the left ventricle (LV), mitral valve (MV), and left atrium (LA). Note the large vegetative lesions on the mitral valve.

Aortic valve insufficiency is the other common valvular disorder in the horse. It causes a diastolic heart murmur that begins just after S_2. The murmur is usually of greatest intensity over the left chest wall, just below the level of the point of the shoulder around the fourth to fifth intercostal space. In some cases there will be a characteristic prominent pulse because of the increase in end-diastolic volume associated with reflux of blood into the left ventricle.

Right AV valve (tricuspid) insufficiency is encountered more rarely than that of the left AV and aortic valves. We see one to two cases a year that are presented with a decrease in performance, and the only clinical finding is a localized right-sided systolic heart murmur. Few of these horses develop congestive heart failure in the short term.

DIFFERENTIAL DIAGNOSIS (IN ORDER OF LIKELIHOOD OF OCCURRENCE)

- Left AV (mitral) valve insufficiency
- Aortic valve insufficiency
- Right AV (tricuspid) valve insufficiency
- Pulmonic valve insufficiency
- Valvular stenosis (very uncommon)

TREATMENT

- No long-term treatment is possible in valvular insufficiency. However, if there are signs of congestive heart failure, some treatment that may relieve the clinical signs is possible.
- By the time edema of the ventral abdomen develops, it is difficult to give any worthwhile therapy. However, the use of a diuretic such as furosemide (Treatment No. 55) is helpful, and digoxin (Treatment No. 33) may be considered.
- Digoxin may be given intravenously at a loading dose of 14 μg/kg, followed by a maintenance dose of 3.5 μg/kg twice daily. Alternatively, the drug may be given orally at a loading dose of 70 μg/kg once daily, followed by 12 hourly dosing at dose rates of 35 to 40 μg/kg. Because of great variability in the absorption of digoxin when given orally, it is helpful if plasma concentrations can be measured. Effective plasma con-

centrations of digoxin are in the range 0.5 to 2.0 ng/mL.

Mitral Regurgitation

KEY POINT

Mitral regurgitation is associated with a holosystolic heart murmur that has its point of maximal intensity over the left thorax around the fifth intercostal space, a little below the level of the point of the shoulder.

In cases of severe mitral regurgitation, the third heart sound is exaggerated and a pronounced flow murmur is heard in diastole due to the increased inflow into the left ventricle.

Cases of chordae tendinae rupture associated with left AV valve insufficiency result in a more severe systolic heart murmur.

Mitral regurgitation is usually the consequence of degenerative changes on one of the two valve leaflets or their support apparatus. Apart from pulmonary regurgitation, the incidence and importance of which are still relatively unclear, mitral regurgitation is the most uncommon of all regurgitant murmurs in the horse. It is, however, the most significant regurgitant murmur for athletic performance and therefore always warrants further investigation. It is usually insidious in onset, and the owners may not associate signs of poor performance with cardiac disease. In cases of rupture of one of the major chordae tendineae, the onset of clinical signs can be very sudden, with some horses being reported to show an abrupt decrease in performance during an event or after a jump. In these cases, pulmonary edema may develop within a few days, and the horse will present with froth pouring out of both nostrils during episodes of coughing.

HISTORY AND PRESENTING SIGNS

- Poor performance
- Coughing
- Weak arterial pulse
- Froth from both nostrils
- Ventral edema
- Signs of depression

CLINICAL FINDINGS AND DIAGNOSIS

In cases of rupture of one of the major chordae tendineae, major clinical signs are evident. A loud heart murmur with the point of maximum intensity over the lower left chest wall is typical. Echocardiography is valuable in establishing a diagnosis.

TREATMENT

See treatment for valvular insufficiency. The prognosis is poor.

Other Cardiovascular Disease

Most other heart disease that does not primarily involve conduction disturbances is associated with valve disorders and sometimes disorders of the great vessels. Most cases of heart disease are not responsive to treatment, making the treatment options limited. Of greatest interest to horse owners and trainers is the prognosis. Although there are a large number of possible disorders affecting the heart and vessels, most of these are unusual in a normal practice setting. The cardiovascular disorders described below are those that are most likely to be found in equine practice.

Aortic Rupture

Aortic rupture is uncommon but often spectacular, particularly if it occurs in association with exercise. Aortic rupture may be the result of a weak point such as an aneurysm in the vessel wall or may occur spontaneously around the level of the aortic valve. In some cases in which histopathology has been done, necrosis has been found involving the media of the aortic wall.

HISTORY AND PRESENTING SIGNS

- Sudden death

CLINICAL FINDINGS AND DIAGNOSIS

- If the problem occurs at rest, the horse will be found dead.

KEY POINT

If the problem occurs during exercise, most horses will show a sudden decrease in speed, become ataxic, and collapse. In most cases there is sufficient warning that there is something wrong for the rider to dismount before the horse collapses.

- Necropsy reveals large amounts of blood in the thorax. In one case, this was localized within the pericardium so that cardiac tamponade resulted.

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DIFFERENTIAL DIAGNOSIS

- Snake bite
- Lightning strike
- Severe trauma

TREATMENT

- Stand out of the way!

Endocarditis

KEY POINT

Endocarditis is an uncommon disease that is usually the result of bacterial localization in the endocardium and may involve the valves.

HISTORY AND PRESENTING SIGNS

- Coughing
- Signs of depression
- Ventral edema

CLINICAL FINDINGS AND DIAGNOSIS

- Horses are usually depressed and may have intermittent fever.
- If localization of the problem is in the wall of the endocardium and there is no valvular involvement, there may be no heart murmur.
- There may be signs of heart failure late in the course of the problem.
- If there is valvular involvement, case studies have shown that the aortic and left AV valves are the most common valves affected.
- Commonly there is a heart murmur. This is usually a diastolic murmur because aortic valve lesions are most common. However, systolic murmurs may be present also.
- Vegetative lesions of the valves often can be detected on echocardiography if the valves are thickened.
- Hematology and plasma biochemistry may show anemia, neutrophilia, and hyperfibrinogenemia.
- Blood culture can be helpful to determine the bacteria involved. This is most useful in the acute stages when there is usually fever and bacteremia.
- *Streptococcus* spp. are most commonly involved.

DIFFERENTIAL DIAGNOSIS

- Valvular insufficiency
- Congenital heart problems
- Thrombophlebitis
- Pleuritis

TREATMENT

- Treatment is often unrewarding.
- Assessment of the degree of cardiac compromise is important for prognosis.
- Antimicrobial therapy may be useful in the acute stages of endocarditis, and the results of blood culture may be helpful in deciding which drug to use. If no bacteriologic results are known, therapy with penicillin (20,000 IU/kg of Na or K penicillin given IV or IM qid; Treatment Nos. 84 and 85) and gentamicin (6.6 mg/kg sid; Treatment No. 56) may be used.

Jugular Thrombosis

KEY POINT

Thrombosis and thrombophlebitis are common problems in horses, particularly racehorses, because of the range of medications administered via the jugular veins.

In horses that require prolonged jugular catheterization, thrombophlebitis is not infrequent. Predisposing factors include endotoxemia, bacteremia, and mechanical factors such as irritation by the catheter or drugs. The perivascular injection of irritant drugs such as thiopentone, phenylbutazone, and aminoglycosides can lead to intense local phlebitis.

HISTORY AND PRESENTING SIGNS

- Prolonged catheterization
- Systemic infection
- Repeated injections of irritant drugs
- Perivascular injections

CLINICAL FINDINGS AND DIAGNOSIS

- The most consistent sign is swelling around the jugular vein, with vein thrombosis being palpable as a hard cord-like structure.
- There may be swelling around the proximal part of the neck and the head because of the venous compromise. However, in most cases the collateral venous circulation is sufficient to resolve the congestion.
- Horses may have an elevated body temperature and show signs of depression and anorexia in the acute phase of the disease.
- Septic thrombophlebitis may be characterized by a draining tract over the vein, but this is uncommon.
- Ultrasound examination is useful to determine whether the vein is patent or occluded.
- A hemogram and fibrinogen estimation are use-

ful to determine evidence of infection. In early cases where there is intermittent fever, blood culture is useful to isolate significant bacteria.

DIFFERENTIAL DIAGNOSIS

- Localized cellulitis

TREATMENT

- Because many cases of thrombosis and thrombophlebitis are secondary to intravenous catheterization, it is important to use good technique (see Chapter 19). In this way, problems of thrombophlebitis may be avoided.
- Local anti-inflammatory therapy can include cold or warm compresses to the site, depending on the stage at which the problem is found. Nonsteroidal anti-inflammatory drugs such as phenylbutazone (Treatment No. 88) at a dose rate of 4.4 mg/kg bid orally for 4 to 7 days and topical dimethylsulfoxide (Treatment No. 34) can be useful.

KEY POINT

Despite an occluded jugular vein, many athletic horses perform satisfactorily and have no adverse effects once the initial active inflammatory process has resolved.

- Antimicrobial therapy is important and may have to be maintained for several weeks. Where possible, this should be based on results of culture and sensitivity tests, although culture of a discharging sinus will not result in useful information. Procaine penicillin (Treatment No. 83) given intramuscularly at a dose rate of 15,000 lU/kg (15 mg/kg) and gentamicin (Treatment No. 56) given intravenously at a dose rate of 6.6 mg/kg sid are the antibiotics of choice.
- Surgical "stripping" of the affected vein is indicated in severe cases that are refractory to medical therapy. This is a major procedure and is best undertaken at an institution or a major referral practice.

Pericarditis

KEY POINT

Pericarditis is not common in horses. When it does occur, it may be the result of extension of pneumonia and pleuritis.

Pericarditis produces a range of severe clinical abnormalities, and the problem can be suspected where there are signs of a severe and progressive cardiac disorder. The cause for the pericarditis is mostly unknown, but cases have been reported associated with trauma, chest infections, and systemic viral or bacterial infections.

HISTORY AND PRESENTING SIGNS

- Ventral edema
- Prominent jugular venous distention
- Dyspnea
- Usually found in mature horses

CLINICAL FINDINGS AND DIAGNOSIS

- Most horses will have an increased heart rate, and the pulse is weak and thready.
- The classic sign of pericarditis is muffled heart sounds.
- Because of the direct pressure on the heart, venous obstruction is a common finding. The result is jugular vein distention and ventral edema. These findings are typical of many cases of congestive heart failure, and therefore, pericarditis must be distinguished from other causes.
- Most horses show signs of dyspnea, and there may be alterations in the appearance of the mucous membranes, with some horses showing cyanosis.
- Prolongation of capillary refill time is common, indicating poor peripheral perfusion. Diagnosis is made by the typical signs together with ultrasound examination of the heart (Fig. 6-36). Fluid can be seen on the echocardiogram.

KEY POINT

To establish a definitive diagnosis, pericardiocentesis should be performed. This can be done using ultrasound guidance but is also possible "blind."

- Pericardiocentesis is best performed a little caudal to the heart, over the sixth intercostal space. At this location there is less risk of trauma to the heart compared with a more anterior site.

DIFFERENTIAL DIAGNOSIS

- Pleuritis
- Endocarditis
- Congestive heart failure
- Thrombophlebitis

TREATMENT

- Drainage of the pericardial sac should be performed using a 12- to 14-gauge catheter that is

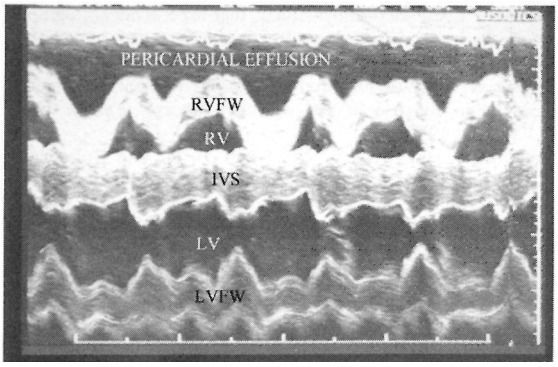


Figure 6-36. Echocardiogram obtained from a 7-year-old Standardbred horse with pericarditis. M-mode image taken from a right parasternal short-axis view, chordal level. Structures shown include right ventricular free wall (RVFW), right ventricle (RV), interventricular septum (IVS), left ventricle (LV), and the left ventricular free wall (LVFW). Note the massive pericardial effusion and the small size of the left and right ventricles. Also note the increased thickness of the right ventricular free wall, interventricular septum, and left ventricular free wall. Of particular interest is the complete loss of coordinated contraction between the left and right ventricle. This horse presented with signs of severe congestive heart failure and was euthanized on humane grounds.

at least 12.5 cm long. In most cases there is a substantial amount of fibrin deposition, and therefore, the catheter may become blocked.

Great care should be taken when advancing the catheter because if the stylet damages the surface of the myocardium, the horse may die. The most direct access is over the fourth or fifth intercostal space, but great care must be taken in this location.

If there is profound fluid accumulation in the pericardium, it may be difficult to distinguish the fluid from that found in pleuritis.

Some horses will recover after drainage of fluid from the pericardium, but the prognosis is poor for most horses.

Viral or Bacterial Myocarditis

Myocarditis, a localized inflammatory response in the myocardium, is diagnosed infrequently and is not common. In the past in some parts of the world, this disorder was diagnosed on the basis of abnormalities of the T wave. However, it is now apparent that such a diagnosis on this basis is incorrect. More commonly, myocarditis results in atrial premature contractions (APCs) and/or VPCs due to the inflammatory focus in the myocardium.

KEY POINT

Although there have been few confirmed cases, the equine veterinary literature often mentions myocarditis as a sequel to equine influenza, African horse sickness, and strangles.

This may be because a percentage of horses that recover from these conditions show a decrease in exercise capacity after infection. It is likely that the reason for the reduced performance in many such cases is respiratory rather than cardiovascular in origin.

HISTORY AND PREVENTING SIGNS

- Previous respiratory infection
- Usually in young horses
- Exercise intolerance

CLINICAL FINDINGS AND DIAGNOSIS

- Exercise intolerance is the most common history, usually with evidence of distress after hard exercise.
- On auscultation, there may be an arrhythmia if the myocarditis involves one of the conducting pathways. An ECG will be necessary to determine the precise nature of the electrical disturbance.
- Auscultation of the heart may not indicate rhythm disturbances, but there may be alterations in electrical activity, with abnormalities of conduction, such as bundle-branch block or intraatrial block, or abnormalities during repolarization, APCs, or VPCs.
- If the resting ECG is normal, it may be valuable to record the ECG during exercise, because arrhythmias may be more obvious during or immediately after strenuous exercise.
- In the acute stages of myocarditis, we have found increases in serum concentrations of lactate dehydrogenase (LDH). However, because few laboratories measure LDH isoenzymes, it is difficult to be sure that elevations in serum or plasma LDH indicate myocardial damage.

KEY POINT

Even when the cardiac isoenzyme of LDH is measured, it is important to note that it is not necessarily myocardial specific and can be increased due to skeletal muscle damage.

- Echocardiography may not be helpful for diagnosis unless there is significant impairment to myocardial function.

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DIFFERENTIAL DIAGNOSIS

- Lower respiratory tract disease
- Endocarditis
- Minor ECG conduction abnormalities

TREATMENT

- No treatment is likely to be useful if there is myocarditis, apart from rest and time. In some cases, we have seen the ECG return to normal after 3 to 6 months of rest.
- If there is evidence of systemic bacterial infection, it may be useful to give a course of an antibiotic such as procaine penicillin (Treatment No. 83) at a dose rate of 15,000 lU/kg (15 mg/kg) bid and possibly nonsteroidal anti-inflammatory drugs.
- If there are indications of severe cardiac disease, including heart failure, the prognosis is very poor.

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CHAPTER 7

Alimentary System

Mark T. Ethell, Andrew J. Dart, David R. Hodgson, and Reuben J. Rose

EXAMINATION OF THE ALIMENTARY TRACT

A thorough and careful examination of the digestive system is particularly important when evaluating horses with suspected gastrointestinal tract disorders. In general, as with the examination of any other body system, a logical sequence of events is followed during the examination procedure. After the collection of appropriate information relating to signalment, history, and duration of signs (see Chapter 2), examination usually begins at the head and proceeds caudally (see Chapter 1).

History

Important questions relating to gastrointestinal dysfunction include the following:

- Is the horse eating normally?
- Does the horse lose food from its mouth when eating?
- Does the horse salivate excessively when eating?
- Is there evidence of weight loss?
- Is there evidence of abdominal pain?
- What is the duration of the problem?
- Is this the only animal on the premises showing these abnormalities?
- What is the vaccination, deworming, and dental prophylaxis history of this and other animals on the farm?
- Is the horse stabled or does it live outdoors?
- Is the horse hand fed or does it eat pasture?
- Does the horse live in an environment where there is much sand?

After collection of the history, the clinician should then undertake a physical examination.

Initial Inspection

Examination of a horse with suspected alimentary tract disease has a number of components, including general physical examination, auscultation, rectal examination, passage of a nasogastric tube, clinical pathology, and other diagnostic aids. Initial inspection involves determination of the cardinal signs (heart rate, respiratory rate, capillary refill time, pulse quality, mucous membrane color, and body temperature), followed by specific examination of the alimentary tract. Examination begins at the mouth, looking for evidence of malocclusion and the presence of sharp edges on teeth, damage to the lips and gums, and other dental abnormalities. Nasal discharge, excessive salivation, and other abnormalities (e.g., swellings) are then noted. Next, the neck and external abdominal contours are determined, with note being made if distension of the flanks is present. This is usually the result of gas accumulation within the large bowel. The presence of edema or other abnormalities such as hernias also should be noted.

DIAGNOSTIC AIDS

Auscultation

After initial inspection, auscultation of the abdomen is performed. The left and right sides of the abdomen should be auscultated; approximately 4 to 5 minutes is required to perform this procedure thoroughly. Normal sounds often are referred to as "rumbling," "bubbling," and "splashing" sounds. The clinician should note the tone, intensity, and duration of sounds. Large-intestinal

sounds are often of lower intensity and deeper than those originating from the small intestine. Examination should begin in the region of the paralumbar fossa, subsequently moving from the middle to ventral abdominal regions (see Figs. 1-20 and 1-21). In the right paralumbar fossa, ileocecocolic sounds can be heard, which are a high-pitched rumble that sounds like water flushing down a drain pipe. Ileocecocolic sounds occur approximately one to three times per minute in the normal horse. When the lower part of the abdomen is auscultated, small intestinal sounds often can be detected. These are referred to as borborygmi and are low-pitched fluid sounds. Where horses have been fasted or without feed, these sounds may be less prominent.

KEY POINTS

Repeated auscultation is vital when monitoring horses with acute abdominal pain. Although auscultation alone will not be diagnostic, a progressive decline in the frequency or intensity of intestinal sounds may be associated with an unfavorable prognosis.

Although sounds may be temporarily reduced in some cases of colic, a persisting absence of gut sounds is often ominous. In contrast, increased intensity of gastrointestinal sounds and evidence of the presence of large amounts of gas may be indicative of spasmodic colic.

In horses that have gaseous distension of the abdomen, simultaneous auscultation and percussion may reveal a resonant ping.

Rectal Examination

Rectal examination is useful in horses with suspected gastrointestinal tract disease, particularly those suffering from abdominal pain. Any horse with a history of persistent or recurrent colic, chronic weight loss, fever of unknown origin, or chronic diarrhea should be considered a candidate for rectal examination. The clinician should be thorough and systematic when performing this examination, which can provide valuable diagnostic information. However, care is required because of

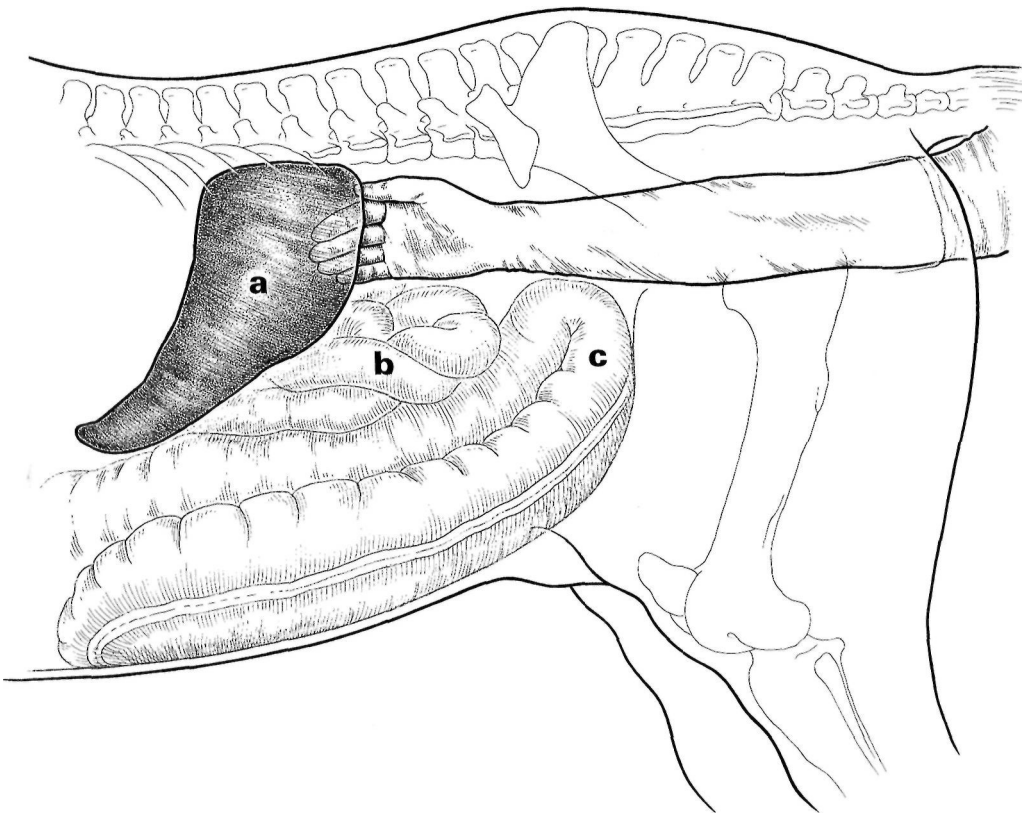


Figure 7-1. Rectal examination. Position of examiner's arm in the rectum when palpating the spleen (a). The spleen normally lies in the dorsal part of the abdomen on the left-hand side adjacent to the body wall. The tapering posterior edge of the spleen is most easily palpable. The small intestine (b) and pelvic flexure (c) are shown.

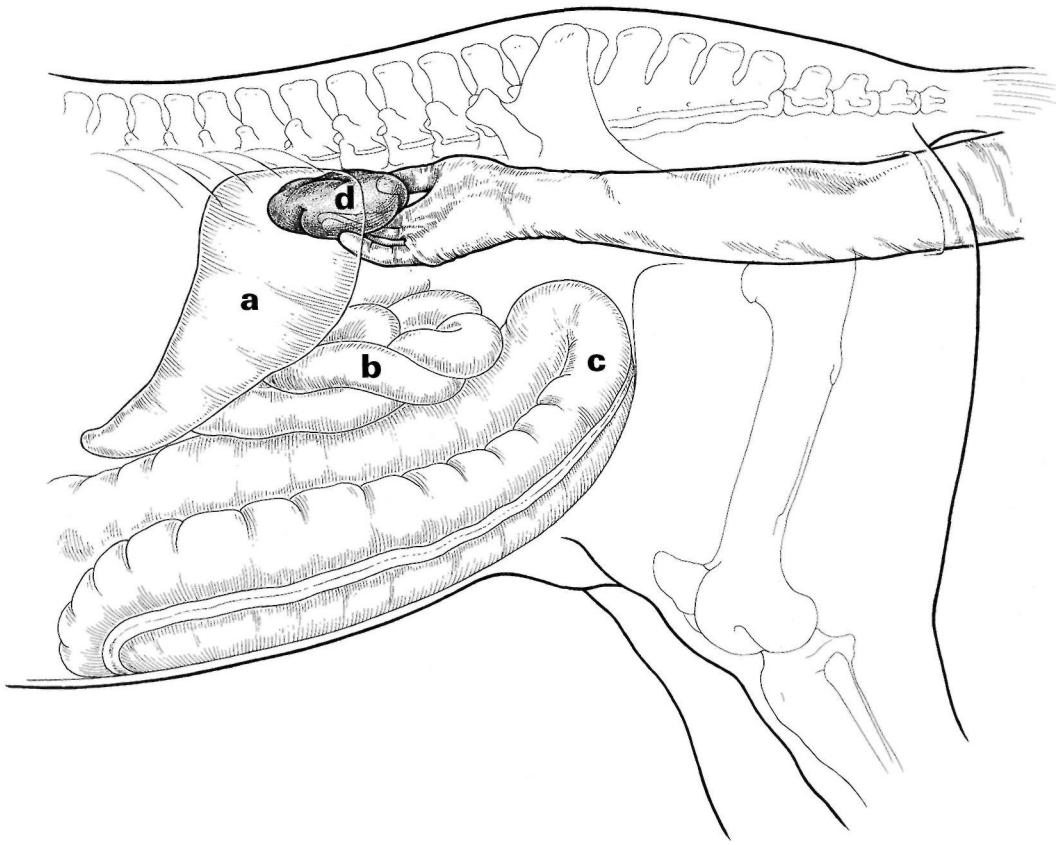


Figure 7-2. Rectal examination. Position of examiner's arm in the rectum when palpating the left kidney (d). The kidney is dorsal and medial to the spleen and is attached to the body wall. The aorta lies medial to the kidney. The spleen (a), small intestines (b), and pelvic flexure (c) are shown.

the risk of irritation or rupture of the rectum. Appropriate restraint of the horse is necessary, which may be done using stocks. Alternatively, in horses with good temperament, the examination may be done over a stall door, using hay bales behind the horse, or hobbles may be used. The clinician should wear a thin plastic rectal examination glove that should be well lubricated, with a methylcellulose lubricant, before insertion in the rectum. Because some rectal examination gloves have sharp edges, it can be useful to invert the glove before putting it on. The examiner should be patient, introducing the arm into the rectum slowly, and if a peristaltic wave is encountered as the arm is introduced, no attempt should be made to move the arm forward, otherwise there is a great risk of rectal rupture. After evacuation of the rectum of feces, if the horse still continues to strain, 30 to 60 mL of lidocaine (Treatment No. 67) alone or mixed with an equal volume of obstetrical lubricant can be infused into the rectum. This will provide local analgesia and help reduce straining. We examine the pelvic structures first and then survey the left side of the abdomen, sweep across the midline, and complete the examination on the right side. In the normal horse, the following structures may be identified: bladder, female reproductive tract, inguinal rings, cecum, small and large colon, including pelvic flexure, spleen, left kidney, nephrosplenic ligament, mesenteric root, aorta, ventral band and dorsal attachment of the cecum, and the peritoneal surface. The structures that can be examined and some of the abnormalities detected are shown in Figures 7-1 to 7-9.

KEY POINT

The following points should be considered when examining organs per rectum: position, size, mobility, thickness, evidence of edema, tight mesenteric bands, or distension due to gas and presence of fluid or ingesta.

The following points may be noteworthy:

Feces—May be dry, hard, or contain blood, mucus, or sand.

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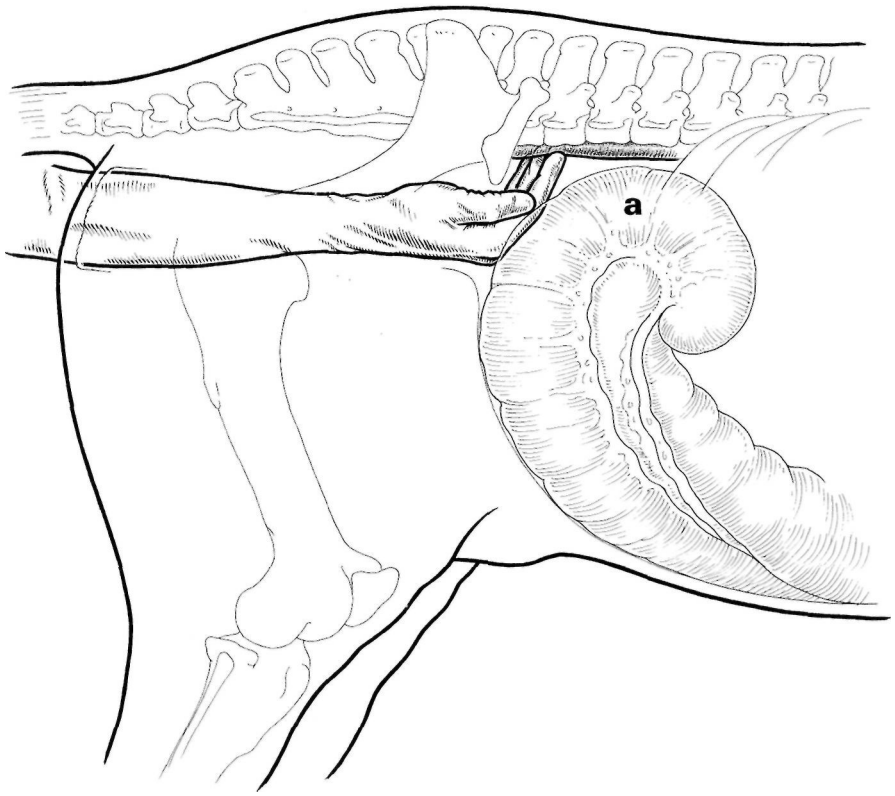


Figure 7-3. Rectal examination. Position of examiner's arm in the rectum when palpating the aorta. It lies in the midline attached to the dorsal body wall. A pulse should be felt. Moving caudad, the bifurcation of the aorta is palpable. The base of the cecum (a) is shown.

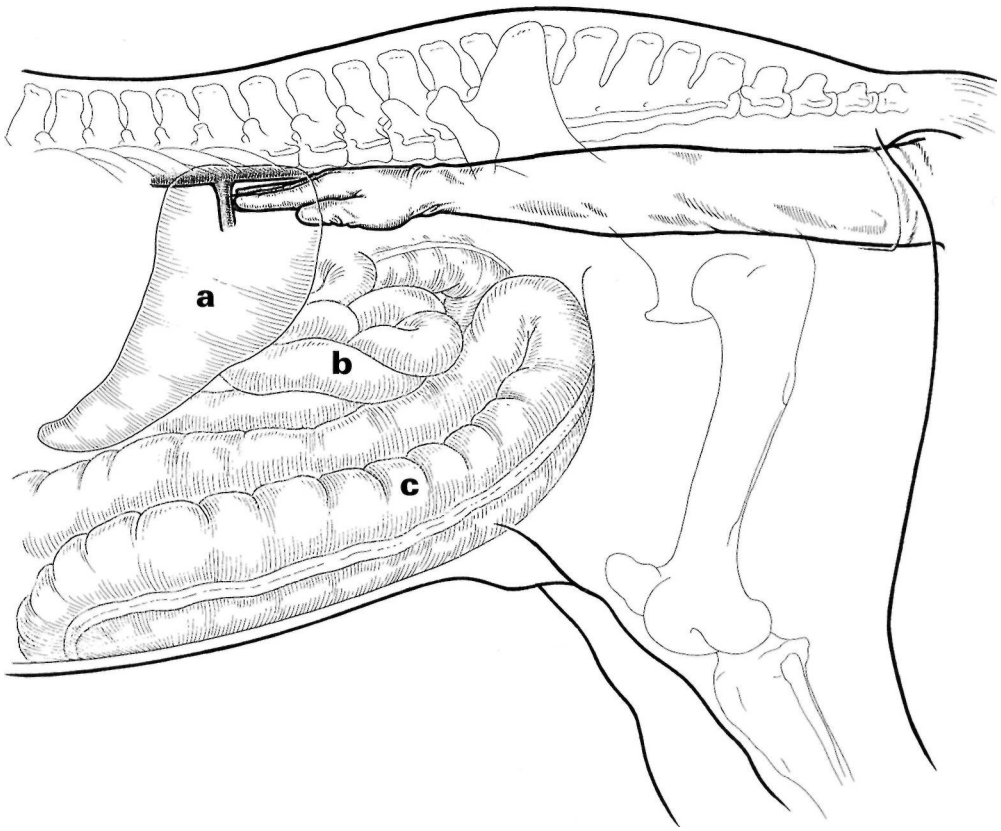


Figure 7-4. Rectal examination. Position of examiner's arm in the rectum when palpating the mesenteric root. The arm should be inserted as far into the rectum as possible. The mesenteric root is a taut structure running dorsoventrally in the middle of the abdomen. The spleen (a), small intestine (b), and left ventral colon (c) are shown.

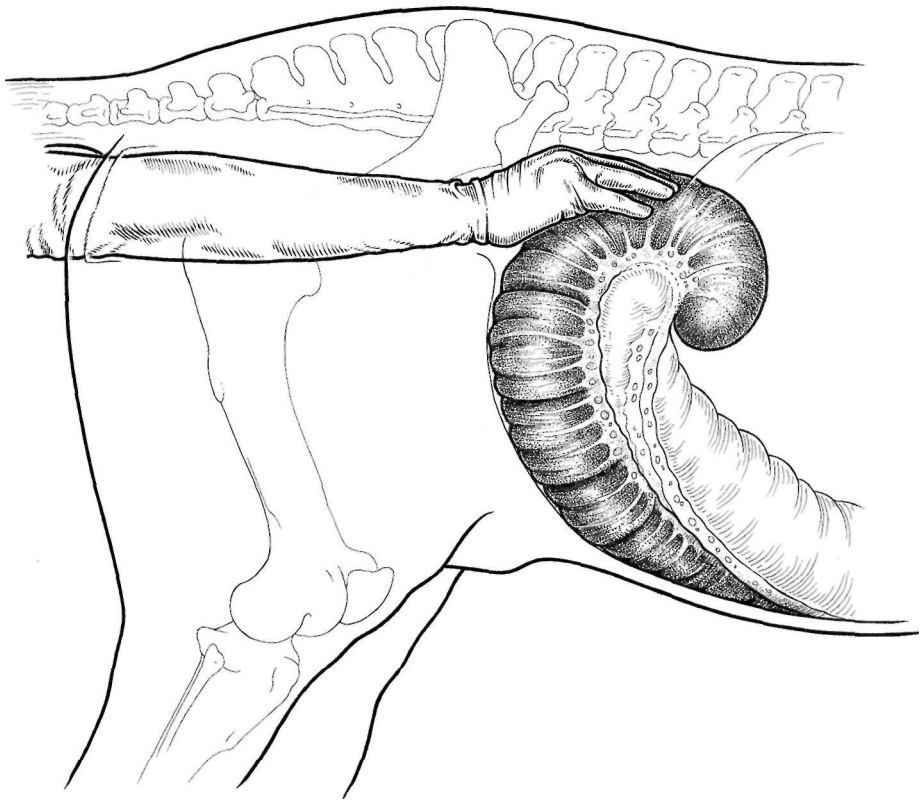


Figure 7-5. Rectal examination. Position of examiner's arm in the rectum when palpating the cecum. The taenia on the body of the cecum can be felt on the dorsum of the cecum coursing ventromedially.

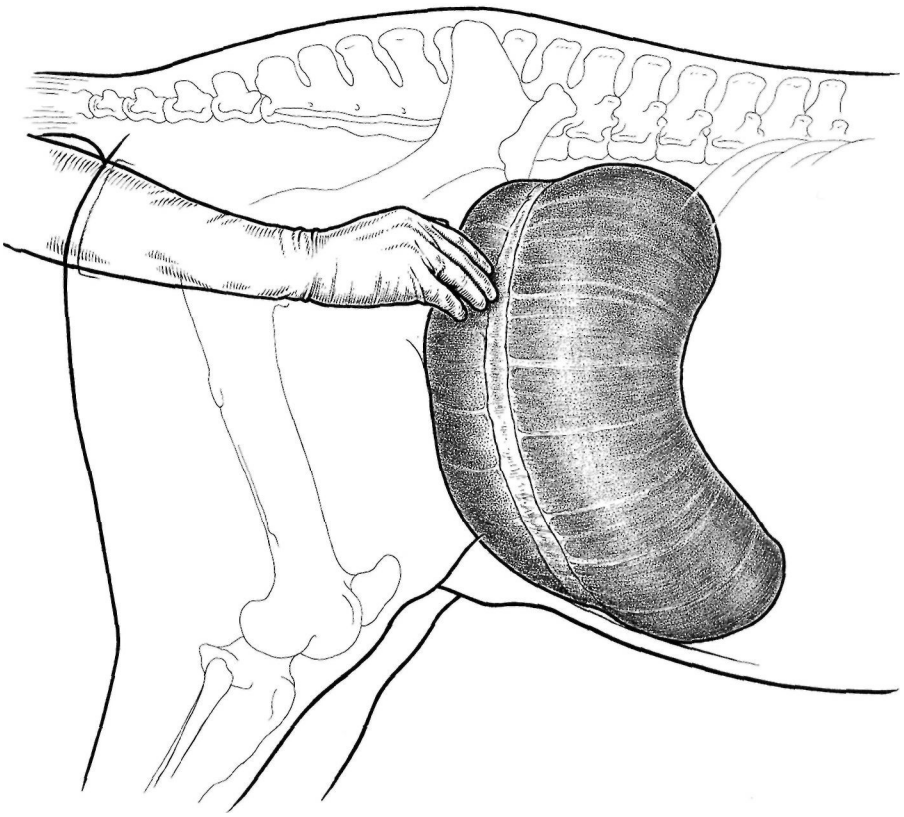


Figure 7-6. Rectal examination. Position of examiner's arm in the rectum when palpating cecal tympany. The cecum is distended and moves caudally to the pelvic inlet. The ventral band is taut and curves dorsoventrally from left to right.

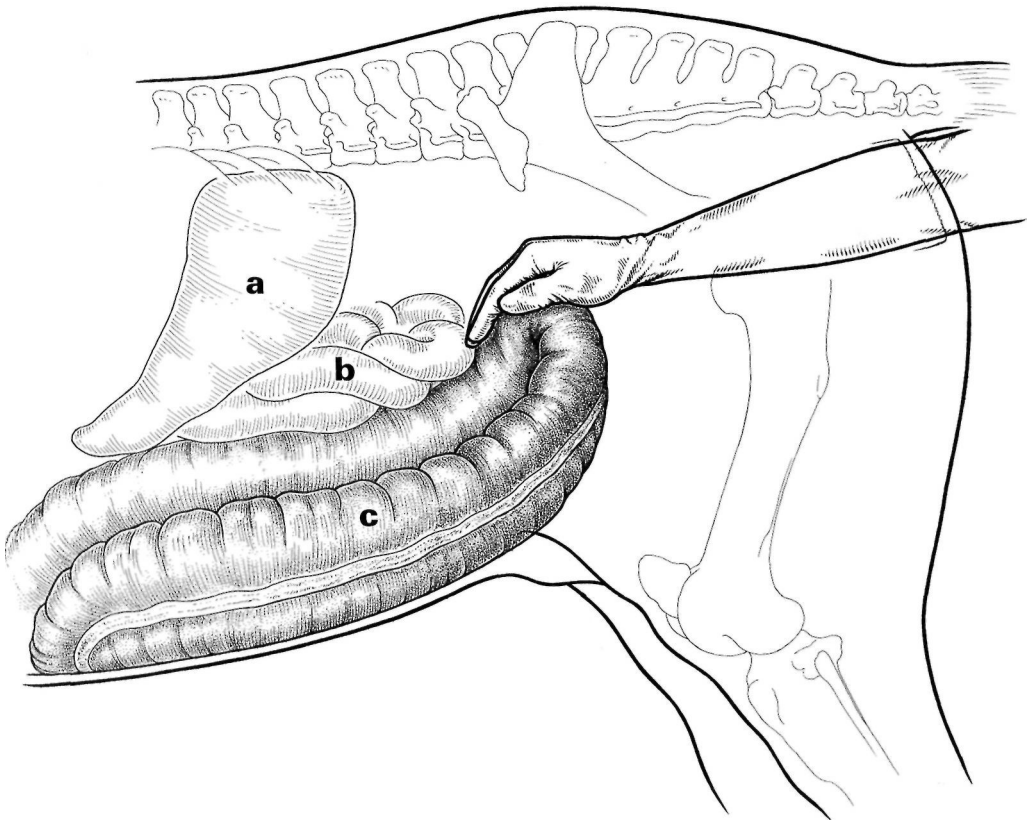


Figure 7-7. Rectal examination. Position of examiner's arm in the rectum when palpating the pelvic flexure of the large colon. It lies just over the pelvic brim on the left-hand side of the midline on the ventral abdominal wall. The spleen (a), small intestine (b), and left ventral colon (c) are shown.

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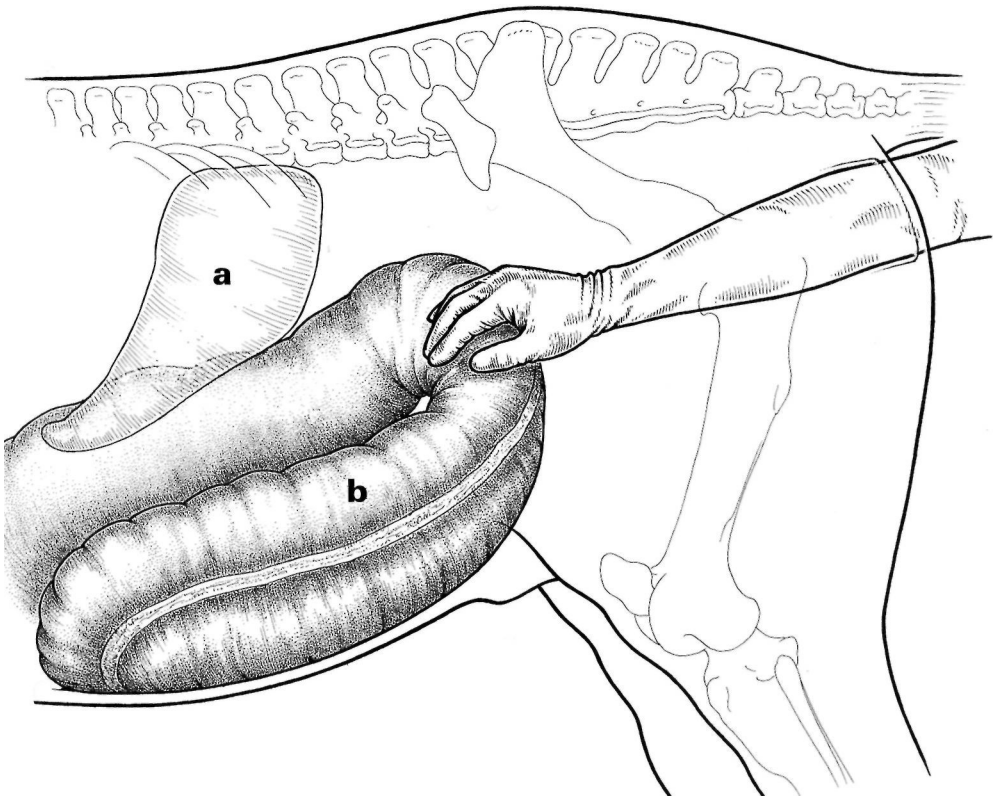


Figure 7-8. Rectal examination. Position of examiner's arm in the rectum when palpating an impaction of the large colon. The usual location is in the left or right ventral quadrants, although position is variable. The spleen (a) and left ventral colon (b) are shown.

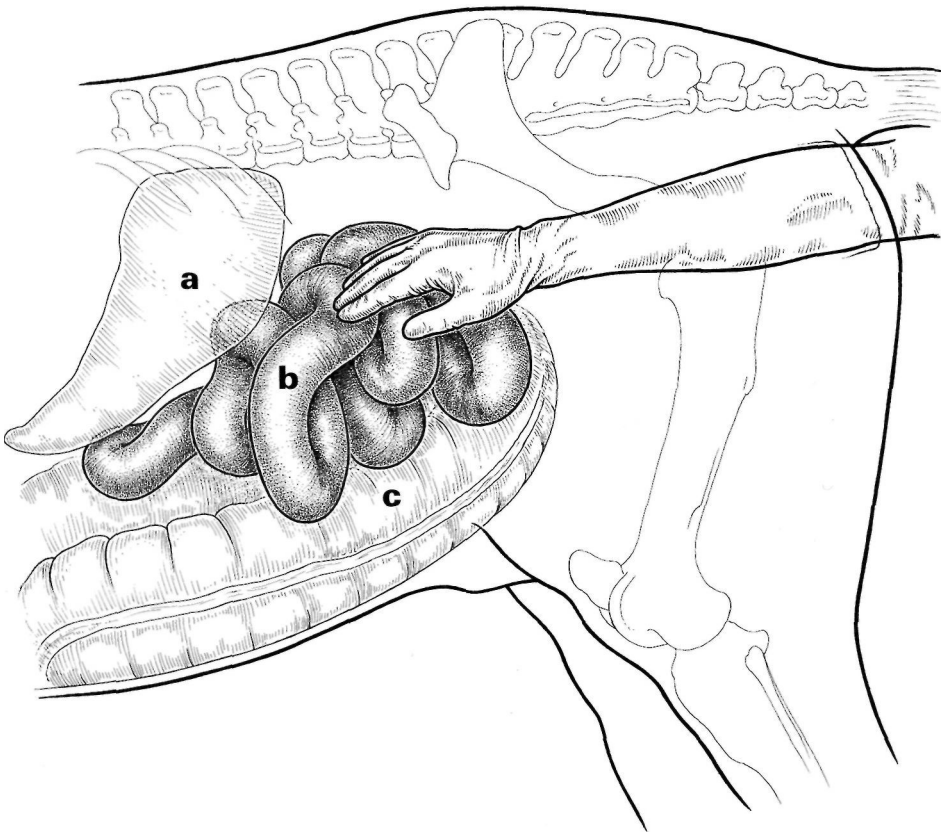


Figure 7-9. Rectal examination. Position of examiner's arm in the rectum when palpating a small intestinal strangulation. Taut loops of gas- and fluid-filled small intestine are often palpable (b). In some cases only one loop is palpable, whereas in others many loops are involved. The spleen (a) and left ventral colon (c) are shown.

Rectal Mucosa—Should be smooth and folded. Significant abnormal changes include roughening or thickening of the rectal wall.

Inguinal Rings—Normally located about 10 cm (4 inches) lateral to the midline on the rim of the pelvis. One of the most common abnormalities of the inguinal rings in colts and stallions is herniation and incarceration of small intestine through the inguinal canals. This can be detected by palpating the loops of bowel passing through the internal inguinal ring.

Small Colon—Detected by palpation of fecal balls within its lumen. The small colon has marked sacculations, is relatively mobile, and has a prominent antimesenteric band. Abnormalities in the small colon include distension and impaction.

Small Intestine—Usually cannot be examined in the normal animal. If examination of the middle portion of the abdomen indicates some form of space-occupying structure that is not easily identifiable, it is usually the small intestine. Distended loops of small intestine are commonly found in association with strangulation of the small bowel or anterior enteritis. At times, intussusception of the small intestine can be palpated, with the bowel becoming thick, turgid, and feeling somewhat like a sausage. Taut mesenteric bands in the small intestine also can be suggestive of abnormalities.

Pelvic Flexure—May be identified in the caudoventral portion of the abdomen near the midline, usually on the left-hand side. One of the most common abnormalities associated with the pelvic flexure is impaction, and firm masses may be indented on digital palpation. Impactions due to sand often give evidence of a gritty sensation. In these cases, sand frequently can be found in the feces.

Large Colon—Distension associated with taut bands and pain may be suggestive of large colon torsion or displacement.

Spleen—Can be identified on the left side of the abdomen next to the abdominal wall near the level of the last rib. It is usually firm and has a distinct shape with a sharp caudal edge. Spleno-



Figure 7-10. Passage of a nasogastric tube showing introduction of the tube into the ventral nasal meatus.

megaly may be reflective of neoplasia, abscessation, or disorders of the reticuloendothelial system.

KEY POINT

One should always palpate from the spleen dorsally toward the midline, across the nephrosplenic ligament, to the caudal pole of the left kidney because at times the colon may displace dorsally and lodge in the area of the nephrosplenic ligament.

Mesenteric Root—This is difficult to palpate because it is located well forward. Enlargement is usually the result of increased size of lymph nodes or lymph node abscessation. This is one of the most common sites for internal abscessation in horses.

Right Dorsal Colon—May be palpable in some horses but is not normally detected unless the colon is distended due to gas or fluid accumulation. Causes of distension of the right dorsal colon include enteroliths, fecaliths causing luminal blockage of the small colon, or impaction where the large diameter right dorsal colon narrows at the transverse colon.

Ventral Band of the Cecum—Descends from dorsal to ventral on a right to left diagonal path. In addition, the dorsal attachment of the cecum can be identified in the region of the right dorsal abdominal wall. The base and body of the cecum are usually only detected when the cecum is distended with gas or ingesta. Cecal impactions may be noted as firm doughy masses. The ileum is usually not palpated as it passes into the cecum unless there is an impaction or intussusception.

Peritoneum—Should be the final structure examined during the rectal examination. Evidence of nodules, roughening, and response to pain or adhesions can be valuable indicators of dysfunction when evaluating a horse with evidence of gastrointestinal tract disease.

Passage of a Nasogastric Tube

Nasogastric intubation is important in the approach to a horse with suspected alimentary tract disease. Passage of a tube is used to achieve several major functions. These include decompression of the proximal gastrointestinal tract to prevent rupture of the stomach, assistance in the location of obstructions (e.g., "choke"), and administration of fluids and medications. The tube is introduced, via the ventral nasal meatus, into the pharynx and then into the esophagus (Figs. 7-10 and 7-11).

KEY POINT

Passage of a nasogastric tube is indicated in all horses exhibiting signs of moderate to

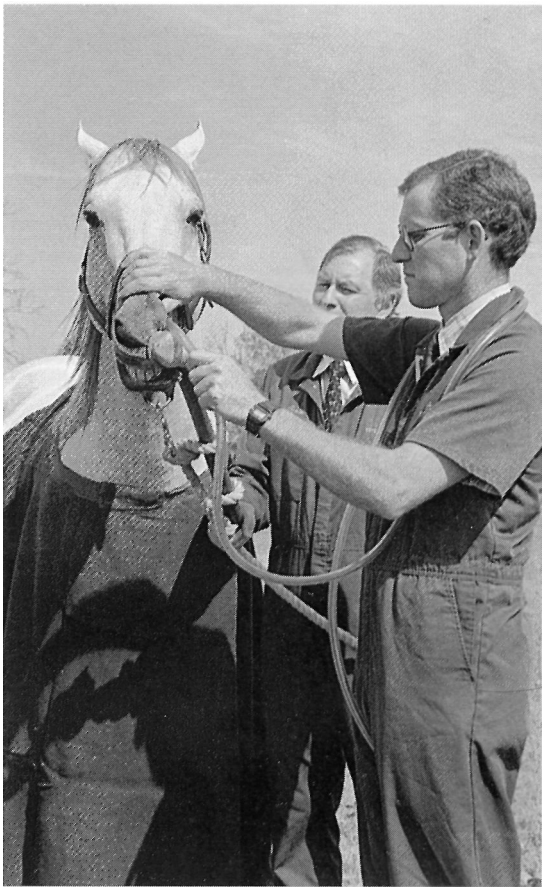


Figure 7-11. Passage of a nasogastric tube showing the position of the handler and veterinarian.

severe abdominal pain that persists for more than 15 minutes.

It is important to ensure that the tube is passed into the stomach if decompression is to occur. However, even when in the stomach, spontaneous decompression will not always occur, and it is necessary to prime the tube with water. This can be done by flushing about 500 mL of water into the tube using gravity feed with a funnel, fluid pump (Fig. 7-12), or garden hose. After the water is infused, the gastric fluid then is allowed to siphon off (Fig. 7-13), and this can be encouraged by rapidly withdrawing the nasogastric tube by 10 to 15 cm. We have found that up to a dozen flushes may be required to ensure that the stomach is fully decompressed. The presence of particulate matter in the stomach may block the tube, making it essential to pass a large tube that has a number of fenestrations in the distal end. Examination of the fluid obtained after gastric reflux may provide some indication of the cause of the problem. The quantity, smell, content, and pH of the reflux should be assessed.



Figure 7-12. Infusion of water into the tube, the distal tip of which has been placed in the stomach. This is to assist in reflux of any excess fluid present in the upper gastrointestinal tract.

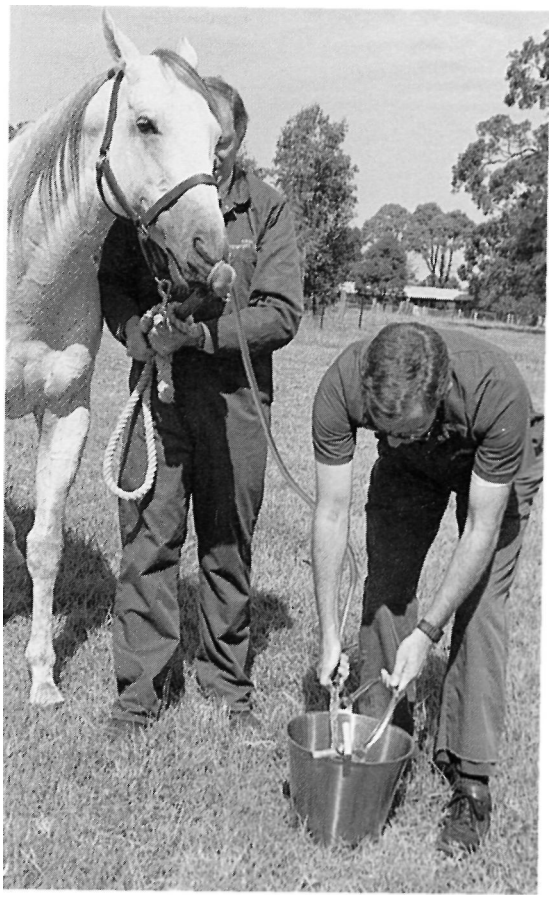


Figure 7-13. Reflux of water from the stomach tube after infusion of several hundred milliliters of water.

Abdominocentesis

The composition of abdominal fluid is determined by the condition of the organs that are bathed by this fluid. As a result, evaluation of abdominal fluid can provide important information in the assessment of horses with alimentary tract disease.

KEY POINT

Abdominocentesis is indicated in horses with a history of acute or recurrent colic, chronic weight loss, or chronic diarrhea.

Abdominocentesis is a safe procedure with a very low complication rate. Puncture of the bowel can occur but generally results in few problems to

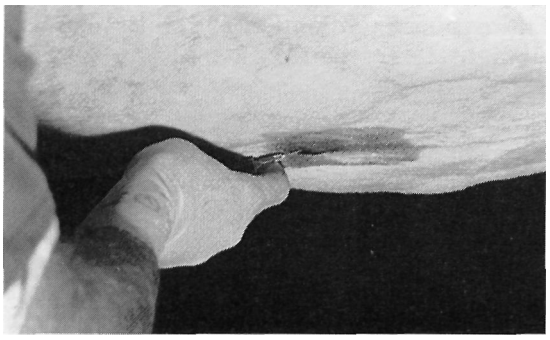


Figure 7-14. Abdominocentesis. Injection of local anesthetic into the most dependent point of the ventral abdomen.

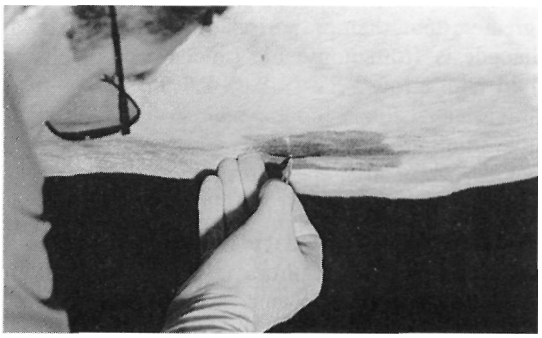


Figure 7-16. Abdominocentesis. Stab incision through the skin and subcutaneous tissues over the midline of the ventral abdomen before insertion of a teat cannula.

the horse. Fluid is collected most frequently from the most dependent site of the ventral abdomen, usually about 10 cm (4 inches) behind the xiphisternum. Ideally, hair along the midline is clipped and shaved, and the skin is prepared aseptically. A small bleb of local anesthetic can be infused under the skin and into the linea alba using a 25-gauge, 15-mm (5/8-inch) needle (Fig. 7-14). However, these procedures, with the exception of skin disinfection, are not necessary if an 18-gauge, 3.75-cm (1.5-inch) needle is to be used for abdominocentesis (Fig. 7-15). In most horses, an 18-gauge, 3.75-cm needle is adequate for collection of abdominal fluid, although is more likely to result in contamination of the sample with blood or intestinal contents. With the clinician standing alongside the horse at the elbow, facing the rear, a position where he or she is least likely to be kicked by a hindleg, the needle is inserted rapidly into the ventral midline, through the skin, and into the abdomen. If no fluid is obtained, the needle should be repositioned further caudally. On occasion, rotation of the needle, injection of air with a sterile syringe, or insertion of several other needles assists in drainage of fluid.

In larger breeds and fat horses, a teat cannula, stainless steel bitch urinary catheter, or spinal needle may be required to penetrate the layer of fat that lies on the internal side of the linea alba. When using this technique, local anesthetic is infiltrated at the site of entry, and a stab incision using a no. 11 or 15 scalpel blade is required (Fig. 7-16) to allow insertion of the cannula. After the stab incision, contamination of the sample with blood is a potential problem. This can be overcome by inserting the cannula through a gauze sponge before insertion of the needle through the abdominal wall (Fig. 7-17). When peripheral blood contamination does occur, the fluid draining from the cannula is clear but is streaked with swirls of blood. In contrast, splenic puncture yields fluid with a high packed cell volume (PCV)

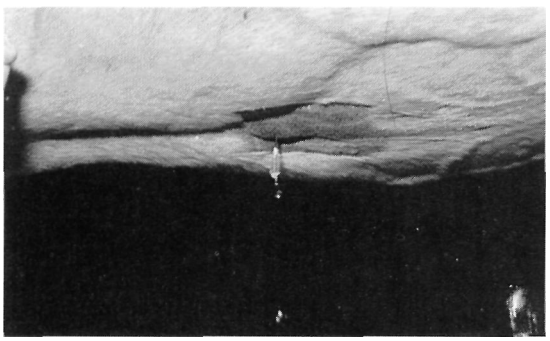


Figure 7-15. Abdominocentesis. Position of an 18-gauge, 3.75-cm needle inserted through the ventral abdominal midline. Abdominal fluid can be seen dripping from the needle.

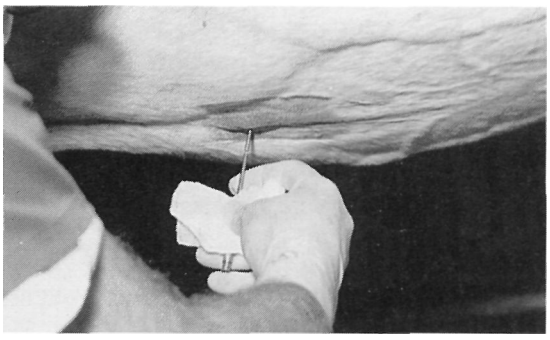


Figure 7-17. Abdominocentesis. Insertion of a teat cannula into the abdomen via the ventral midline. Note that a sterile gauze swab has been used to prevent blood contamination of the sample from blood associated with the skin incision.

(greater than that of peripheral blood), whereas diapedesis from nonviable bowel wall results in fluid with a low PCV (<0.05 L/L or 5%).

LABORATORY ANALYSIS OF ABDOMINAL FLUID

Abdominal fluid is collected into a tube containing Na EDTA as an anticoagulant and a sterile tube if bacteriology is required. Usual determinations include total red cell count, total and differential nucleated cell count, and total protein concentration. Changes in abdominal fluid for a variety of diseases are shown in Table 7—1.

Endoscopic Examination of the Upper Alimentary Tract

Endoscopic examination of the alimentary tract has become more popular in recent years with the advent of flexible fiberoptic endoscopes. However, most endoscopes are made for human use and are not long enough for examination of the lower esophagus and stomach in the horse. The readily available endoscopes are up to 180 cm (70 inches) in length and 13.5 mm (0.6 inches) in diameter. With these, examination of the nasal passages, larynx, pharynx, and proximal esophagus in adult horses is possible. The use of longer endoscopes

TABLE 7-1. Changes in Abdominal Fluid with Different Abdominal Disorders

Condition	Gross Appearance	Total Nucleated Cell Count (cells/μL)	Total Protein Concentration (mg/dL)	Cytologic Appearance
Normal	Clear to yellow	<7,500 (<peripheral WBCC)	<2.0	Neutrophils 40-80% Mononuclear cells 20-50%
Abdominal abscess	Yellow to light brown, ± turbid, blood tinged	15,000-250,000	4.0-6.5	Predominantly neutrophils with mild degenerative changes, ± intracellular Gram + cocci
Simple large bowel impaction or small intestinal obstruction (no devitalized tissue)	Yellow, clear	3,000-15,000	<3.0	Predominantly neutrophils good morphological condition
Small intestine strangulation (prerupture)	Red to brown, turbid to opaque. ± blood tinged	>5,000-50,000	2.5-6.0	Neutrophils predominant, degenerative changes moderate/severe
Anterior enteritis	Yellow, turbid, possibly serosanguinous	< 10,000	3.0-4.5	Predominantly neutrophils, fair morphological condition
Bowel necrosis (rupture)	Orange to brown to green tinged, turbid ± particulate matter	> 150,000	5.0-6.5	Highly cellular >95% neutrophils, degenerative. Many bacteria: Gram — and + intra- and extracellular
Intestinal contents (sample from bowel lumen)	Green, turbid, much particulate matter	< 1,000	Variable	Relatively few cells, many free bacteria: Gram — and +

WBCC, white blood cell count.

(360 cm [140 inches]), allowing examination of the distal esophagus and stomach, is described, but this equipment is usually custom-made and is therefore restricted to use in large referral hospitals. Indications for endoscopy of the upper alimentary tract include discharge of food or other material from the nares, dysphagia, excessive salivation, abnormal discharge from the mouth, dyspnea, and suspected "choke." The endoscope is normally passed down the ventral meatus in a manner similar to that described for passage of a stomach tube. After examination of the nasal passages, pharynx (including the guttural pouch openings), and larynx, the endoscope is passed into the cervical esophagus. The esophagus is dilated with air, and the lumen and mucosa of the esophagus are examined.

KEY POINT

Evidence of esophageal obstruction ("choke"), ulceration, diverticula, and at times perforation or strictures can be gained from endoscopic examination of the esophagus.

However, when considering esophageal strictures, it has been our experience that at times they may not be visible because the folded contours of the esophageal mucosa may disguise their presence. On occasion, material that is lodged in the esophagus can be removed by the use of small flexible forceps that are passed down through the biopsy port of the endoscope. With the use of longer custom-made endoscopes, there are many reports of gastric ulceration in adult horses. Similarly, use of smaller diameter endoscopes has increased awareness of the high incidence of ulceration of the gastric mucosa in foals.

Radiography

With care and patience, high-quality radiographs of the head and cervical esophagus may be obtained using readily available portable veterinary radiographic equipment. A number of disorders that affect the upper alimentary tract can be diagnosed using this technique. These include tooth root abscessation, alveolitis, impacted or fractured teeth, dentigerous cysts, fractures of the mandible/maxilla or hyoid apparatus, fibromas, bony or soft tissue neoplasia, and in some cases disorders of the pharynx (e.g., retropharyngeal abscessation).

KEY POINT

Signs of retropharyngeal abscessation include dyspnea, dysphagia, and radiographic evidence of compression of surrounding tissues.

Radiography of the cranial esophageal region is indicated in cases where there is regurgitation of food from the nares, dysphagia, or suspected "choke." In cases where obstruction of the esophagus has occurred, it may be possible to visualize the site of the obstruction radiographically. Certainly, if there is rupture of the esophagus and localized cellulitis, this will be notable by the accumulation of gas in the periesophageal region. Contrast radiography using barium sulfate also may assist in providing diagnostically useful radiographs of the upper alimentary tract. When attempting to highlight the esophagus, we favor administration of the barium using a small-bore stomach tube that is inserted approximately 10 cm (4 inches) into the esophagus. The dose of barium (about 500 mL for an adult horse) is then injected into the esophagus with a 1-L stomach pump. We find contrast radiography to be useful when attempting to define esophageal strictures and diverticula in adult horses and megaesophagus in foals. Although contrast techniques have been described for the diagnosis of gastric ulcers in foals, we have found this procedure unrewarding. Techniques for radiographic demonstration of enteroliths and sand impactions in adult horses have been reported but require equipment usually limited to large referral practices. Retrograde contrast radiography using barium sulfate infused rectally through a Foley catheter has been used to identify small colon disorders in foals.

Ultrasound Examination

With the increased availability and reduced cost of ultrasound machines, this form of examination has become more popular in recent years. However, the technique is still restricted, particularly in large horses, because the beam will only penetrate to a maximum of about 30 cm. In general, sector scanners provide the best images, with 3.0- and 5.0-MHz heads being the most frequently used. The 3.0-MHz scan head provides greater tissue penetration than the 5.0-MHz head, but this occurs at the expense of image quality. We have found that transabdominal ultrasound examination readily helps in identification of the liver, diaphragm, kidneys, spleen, peritoneal surfaces, and gut wall. For best images, the hair should be clipped and liberal amounts of coupling gel applied. However, preliminary images may be obtained by wetting the hair with alcohol or mineral oil. We routinely use ultrasound guidance when performing biopsies of the liver, kidneys, and

spleen. At times, organs in the caudal abdomen also may be imaged after passing the probe into the rectum.

KEY POINT

Ultrasound examination provides evidence of increased fluid accumulation within the abdominal cavity, fibrinous accumulations on the peritoneal wall, and, at times, the presence of masses in the liver, spleen, or kidneys.

The liver can be imaged on the right side along the ventral border of the diaphragm between the 7th and 15th intercostal spaces. If there is a reduction in the hepatic mass, detection of the presence of the liver on this side may be more difficult. On the left side, the liver is located between the seventh and ninth intercostal spaces. Some indication of hepatic dimensions and architecture can be obtained with ultrasound examination. Other abnormalities that may be detected include evidence of dilated bile ducts and dilated veins, the presence of cholеliths, and in some cases the presence of hepatic tumors or abscesses. The spleen is located on the left side between the 8th and 17th intercostal spaces caudal to the liver. It is located in the cranioventral portion of the abdomen near the diaphragm and rises to a caudodorsal position lateral to the left kidney. We often find it difficult to obtain useful ultrasound information relating to the gastrointestinal tract because the gas within the lumen interferes with ultrasound waves. However, recently it was shown that ultrasound sometimes can be useful in determining the degree of distension of the stomach when the success of gastric decompression is equivocal in horses with colic. Abdominal ultrasound can also be useful for detecting small intestinal strangulating obstructions via assessment of the small intestinal wall thickness, diameter, and motility. An ultrasound finding of edematous small intestine without motility provides an indication of primary small intestinal disease (obstruction or strangulation) and justifies surgical intervention. Additionally, ultrasound examination of the abdominal cavity can be useful in confirming a diagnosis of nephrosplenic displacement of the colon. Further details of the use of ultrasound for examination of the abdominal cavity are provided in Chapter 3.

Clinical Pathology

Clinical pathologic examinations often provide an invaluable adjunct to diagnosis when attempting to define the cause or severity of alimentary tract disease. These examinations should be used to support the clinician's impressions based on the results of history and physical examination findings rather than being a sole means of diagnosis. However, judicious use of well-chosen clinical laboratory tests may have a significant influence on the clinician's diagnostic suspicions.

HEMATOLOGY

Measurement of the hematocrit (PCV) is one of the most frequently performed laboratory tests in equine practice. An increase in the PCV often occurs in association with abdominal disease in the horse. Circulating red blood cell numbers increase after splenic contraction, a response to catecholamine release, or as a result of a decrease in the plasma volume due to fluid loss.

KEY POINT

Hemoconcentration is common in association with abdominal diseases, including gastrointestinal obstructions, peritonitis, diarrhea, and endotoxic shock.

Anemia may occur as a result of blood loss or as a consequence of bone marrow suppression. Blood loss from the alimentary system can occur as a result of parasitism, gastroduodenal ulcers, or gastric carcinoma. In contrast, bone marrow suppression is common in horses as a response to chronic inflammatory conditions (e.g., neoplasia and persistent infections) and is referred to as anemia of chronic inflammatory disease.

Alterations in the leukocyte count may be reflective of stress, inflammation, and infection. Stress (e.g., transport, exercise, or abdominal pain) is the most common cause of a mature neutrophilia that is associated with reductions in the numbers of eosinophils and lymphocytes. This manifestation is referred to as a stress leukogram. In contrast, inflammation and infection will produce an increase in the neutrophil count with more limited changes in the numbers of other leukocytes. However, when extreme demand is placed on the horse's capacity to produce neutrophils, as may occur with severe enteric diseases, a neutropenia with toxic neutrophils may result.

SERUM OR PLASMA PROTEIN CONCENTRATION

Plasma protein is composed of three main fractions that are easily measured in the laboratory. The predominant fractions are albumin and globulins with a lesser but still important contribution by fibrinogen. Total plasma protein concentration

will increase in response to dehydration and contraction of the plasma volume. Reduced water intake and disorders producing abdominal pain and shock may result in dehydration. In these conditions there is normally an increase in both albumin and globulin fractions. Hyperproteinemia also may occur as a result of increases in the globulin concentration in response to chronic antigenic stimulation. Hypoproteinemia occurs in response to protein-losing enteropathy and possibly liver disease. Loss of protein from the bowel may accompany enteritis, parasitism, neoplasia, and nonsteroidal anti-inflammatory drug (NSAID) toxicity.

KEY POINT

Fibrinogen, the third major fraction of total plasma protein routinely measured, reflects the severity and duration of an inflammatory disease.

The normal serum fibrinogen concentration is 2 to 4 g/L (200-400 mg/dL), which may increase to more than 8 g/L (800 mg/dL) in response to severe inflammation.

SERUM BIOCHEMISTRY

Determination of the plasma or serum concentration of electrolytes, minerals, bilirubin, bile acids, activities of a number of cellular enzymes, and evaluation of acid-base status often are performed in horses with suspected alimentary tract disease. With the exception of acid-base measurements, samples can be collected into serum (plain) or lithium heparin tubes. If there is to be a delay of more than a few hours before analysis, the samples should be centrifuged and the serum or plasma decanted and refrigerated until analysis is performed. Horses with acute diarrhea often will show hyponatremia, hypochloremia, hypokalemia, and metabolic acidosis. The extent of these changes will depend on the severity of the diarrhea and whether the horse continues to drink water, thereby further diluting the concentrations of these electrolytes within the plasma. Hypocalcemia and hypomagnesemia also may occur with enteritis, particularly anterior enteritis, where there is loss of these ions with the fluid that is sequestered into the bowel lumen. Elevations in the serum activities of hepatic enzymes are often reflective of hepatic dysfunction. γ-Glutamyl transferase (GGT) is the best screening enzyme for liver disease, but there will also be elevations in the activities of alkaline phosphatase (AP), aspartate aminotransferase (AST), lactate dehydrogenase (LDH), and L-iditol dehydrogenase (L-iDH, or sorbitol dehydrogenase). It should be noted that the extent of the increases in activity does not always correlate with the degree of hepatic dysfunction. Increases in the serum concentration of bile acids will often accompany increases in the activities of hepatocellular enzymes. The concentration of bilirubin is relatively labile in the horse, undergoing mild elevations in response to inappetence, with more marked elevations accompanying liver or hemolytic diseases. Acid-base abnormalities are relatively common in association with acute gastrointestinal tract disorders.

KEY POINT

In addition to losses of electrolytes, a common change is acidosis due to intestinal bicarbonate loss in association with severe enteritis and diarrhea.

In contrast, metabolic alkalosis may occur in diseases where there is substantial loss of hydrogen ions from the gut (e.g., anterior enteritis).

FECAL EXAMINATION

Detection of parasite ova using flotation in salt solutions is commonly used for determination of parasitic infestation. In addition, feces may be examined for parasitic organisms, blood, and bacteria. Specific bacterial species can, at times, be isolated from feces (see Chapter 16). These include *Salmonella* spp., *Campylobacter* spp., *Clostridium* spp., and *Escherichia coli*. In addition, electron microscopy and use of ELISAs for determination of viral infection, particularly rotavirus in foals, also may be undertaken. In the future more sensitive tests (e.g., polymerase chain reaction) for detection of *Salmonella* or *Clostridium* spp. will be used more frequently.

LIVER FUNCTION TESTS

Techniques to measure serum bilirubin concentrations and serum bile acid concentrations are useful in determining liver function. Alterations in specific liver function tests are discussed in the section on liver diseases.

LIVER BIOPSY

Liver biopsy is particularly useful for defining the cause, severity, and prognosis in horses with suspected hepatopathies. The site for insertion of the biopsy needle normally is the 12th to 14th intercostal spaces of the right thorax on a line drawn between the tuber coxa and the point of the

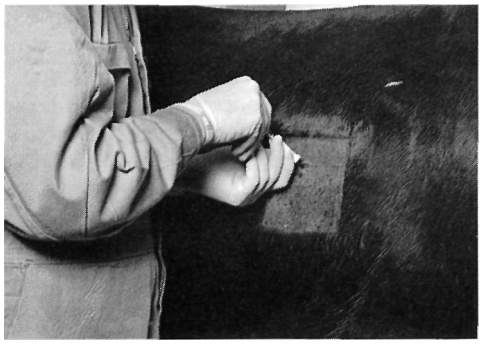


Figure 7-18. Liver biopsy. Position of biopsy needle. The needle has been inserted through the right fourteenth intercostal space, with the tip of the biopsy needle pointing toward the opposite elbow. The needle has been placed along a line drawn between the tuber coxae and the point of the shoulder.

shoulder. An area of skin is clipped, shaved, and aseptically prepared. Local anesthetic (5 mL) is injected subcutaneously and into the intercostal muscles with a 23-gauge, 2.5-cm needle. A stab incision is made with a no. 11 or 15 scalpel blade. A biopsy needle (Tru-Cut, Baxter Travenol, St. Louis, MO; Franklin-modified Vim Silverman, Mueller and Company, Chicago, IL) is inserted and directed craniad and ventrad. The needle is passed through the diaphragm and 10 to 13 cm into the liver parenchyma (Figs. 7-18 and 7-19). The chances of a successful biopsy are improved greatly if ultrasound is used to locate the liver and guide the biopsy needle. If ultrasound is available, biopsies also may be attempted at other sites, including the left side of the horse. After collection, samples should be placed in formalin for subsequent histopathologic examinations or submitted for bacterial culture and sensitivity (if indicated). The skin wound can be left to heal by second intention or is sutured. Contraindications for liver biopsy include evidence of coagulopathies and suspicion of liver abscessation.

SMALL INTESTINAL ABSORPTION TESTS

KEY POINT

Absorptive capacity of the small intestine commonly is evaluated using either the D-glucose or D-xylose absorption tests.

The D-glucose absorption test is easy and inexpensive to perform but is considered less specific than the D-xylose test because blood glucose concentration is affected by the horse's metabolic rate

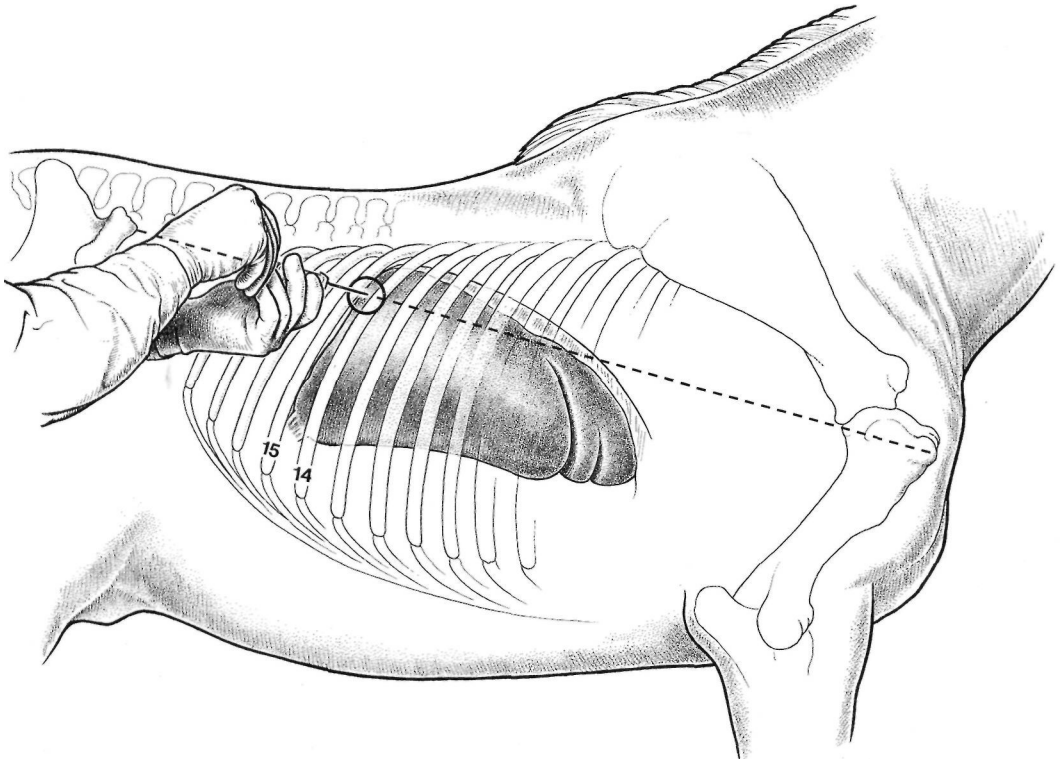


Figure 7-19. Liver biopsy. Line drawing showing position of needle in the liver.

and a number of endocrine factors. To perform the test, the horse should have food withheld for approximately 12 hours before and for the duration of the test. This ensures that food does not delay the passage of glucose into the small intestine or otherwise influence the blood glucose concentration. Glucose is administered at a rate of 1 g/kg as a 20% solution via a nasogastric tube. A normal response is reflected by an increase in blood glucose concentrations of more than 100% over baseline values within 1 to 2 hours (Fig. 7-20).

Xylose is a sugar that is not normally found in equine plasma. Because plasma D-xylose concentration is not influenced by metabolic status, this test is proposed to be a more specific indicator of small intestinal absorptive capacity when compared with the D-glucose test. Disadvantages of the procedure include increased cost and restricted availability of the assay in many laboratories. The horse should have food and water withheld for 12 hours, and 0.5 g/kg of D-xylose (10% solution) is given via a nasogastric tube. A D-xylose concentration of at least 1.0 mmol/L (15 mg/dL) should have occurred within 1 to 2 hours in normal horses (Fig. 7-21).

Examination and Approach to Treatment of the Horse with Abdominal Pain ("Colic")

Over 70 causes of colic in the horse have been identified. This discussion does not address all the known causes but rather focuses on abdominal pain in a problem-oriented fashion. With careful consideration of the history, signs, and appropriate laboratory data, it is hoped that this approach will help the clinician logically define what is the most likely cause of the colic.

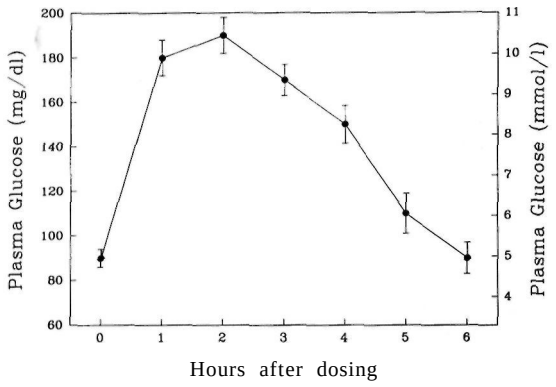


Figure 7-20. Oral D-glucose absorption test. Typical curve of plasma glucose values in adult horses after administration of 1.0 g/kg of D-glucose by stomach tube.

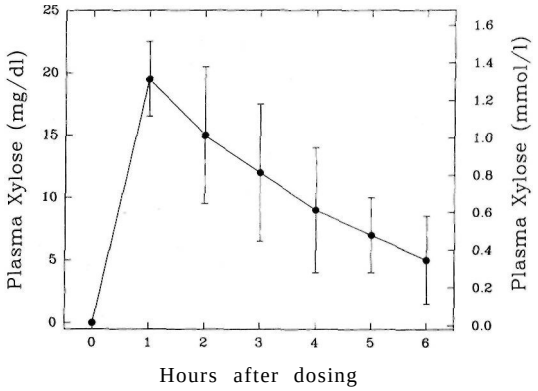


Figure 7-21. Oral D-xylose absorption test. Typical curve of plasma D-xylose values in adult horses after administration of 0.5 g/kg of D-xylose by stomach tube.

Causes of Colic

- Impaction colic (usually large intestine)
- Spasmodic colic (hypermotility, usually small intestine)
- Intussusception (telescoping of bowel, most commonly in the ileocecal region)
- Volvulus (rotation and twisting, usually of small intestine)
- Torsion/volvulus, twisting of bowel (most commonly large colon)
- Strangulation, interruption to blood supply (may occur with volvulus and torsion)
- Tympany, excessive gas production, particularly involving the cecum and large colon
- Colitis/enteritis
- Thromboembolic (verminous)
- Less common causes of colic not necessarily associated with the gastrointestinal tract:
  - Ascarid impaction of small intestine (young horses)
  - Uterine torsion
  - Cervical tears
  - Uterine artery rupture into broad ligament
  - Hemoperitoneum (e.g., after castration)
  - Cystitis or bladder calculi

Anatomic Factors Predisposing the Horse to Colic

A range of anatomic and functional factors of the gastrointestinal tract predispose the horse to colic:

- Inability of the horse to vomit
- The unfixed position of left colon

- The long mesentery of small intestine
- Upward movement of ingesta and narrowing of lumen at the pelvic flexure
- The cecum is a blind sac
- Termination of the right dorsal colon into much narrower transverse colon

History

The following general points are important to consider when gastrointestinal colic occurs:

- Husbandry (diet and diet changes)
- Habitat (box stall, pasture, sandy soil, and weather changes)
- Routine (changes in training, exercise, transport)
- Vices (cribbing, windsucking, indiscriminant appetite)
- Medical history (of case and of other horses on the farm)
- Parasite control

Specific Historical Aspects Pertaining to Colic

- Attitude—signs of depression, alert
- Signs of pain—onset, duration
- Intensity and nature of pain
- Possible causes
- Therapy—type and response
- Defecation—frequency, composition
- Pregnancy, breeding history

PHYSICAL EXAMINATION

Initial Inspection

- Determination of cardinal signs (temperature, heart rate, pulse quality, respiration rate)

Presence of abdominal distension (usually reflective of cecal or large colon distension)
Signs (pawing, rolling, recumbency, abrasions)

KEYPOINT
It is important to be thorough and systematic when undertaking the examination and to assess the whole horse.

A checklist to assist the clinician undertaking an examination of a horse with abdominal pain is provided in Table 7-2.

Assessment of Pain

- Regardless of the lesion, the manifestations of pain consist of pawing, rolling, lying down, kicking at the abdomen, looking at the flanks, sweating, and repeated attempts to urinate. Intense continuous pain often is associated with the most severe lesions, particularly when there is vascular compromise. Ischemic intestine produces intense pain as the tissue becomes anoxic. Once the intestine becomes severely compromised and nonviable, pain usually subsides and horses become depressed and exhibit signs of endotoxic shock.
- Obstruction of the bowel leads to accumulation of gas and fluids, resulting in pain and possibly shock. Where distention is rapid, pain is usually more severe. If the small intestine is involved, pain is often more intense as there is a limited capacity for distension when compared with the large intestine.

Date: _____
Time: _____
CHECKLIST
1. Previous medication _____
2. Attitude _____
3. Severity of pain • Severe • Moderate • Mild
4. Heart rate _____ beats/min
   Rectal temperature _____ degrees
   Respiratory rate _____ breaths/min
   Capillary refill time _____ seconds
   Mucous membrane color _____
5. Peristalsis Left side _____ Right side _____
6. Rectal palpation • Yes • No Result _____
7. Passage of nasogastric tube • Yes • No Result _____
8. Abdominal paracentesis • Yes • No Result _____
9. Packed cell volume _____ % Total plasma protein _____ g/dL

KEY POINT

In general,

- *Severe pain = proximal lesion with strangulation and fluid accumulation or colon torsion.*
- *Mild/moderate pain = nonobstructive lesions without strangulation, often associated with the large intestine.*
- *Spontaneous remission of severe pain may accompany resolution of the problem. However, it always should be remembered that gastrointestinal decompression (and a dramatic reduction in the signs of pain) may accompany rupture of the stomach or intestine.*

Pulse and Heart Rate

- Pulse rate and strength are related to pain, vascular volume, and response to endotoxemia. The pulse can be felt in the facial, digital, brachial, and great metatarsal arteries.
- Heart rate is important to assist in the evaluation of cardiovascular status. Pulse rate and strength usually indicate the severity of the cardiovascular dysfunction occurring in response to the disease.
- With severe pain, usually there is an increase in heart rate to more than 80 beats per minute. In contrast, with simple large intestinal obstruction, there is an increase in heart rate to 40 to 65 beats per minute. With strangulating obstructions, the heart rate usually varies from 50 to 100 beats per minute and in cases of anterior enteritis, the heart rate may increase up to 90 beats per minute. The site of the lesion, volume of fluid loss, degree of toxemia, and duration of the problem will influence the response.

KEY POINT

When examining a horse with colic, it is a good idea to

- *Take the heart rate with a stethoscope while concurrently measuring the pulse rate.*
- *Assess the pulse at several sites.*
- *Assess the blood pressure, most easily done by using a blood pressure cuff applied to the tail and Doppler assessment of blood flow in the coccygeal artery. Normal systolic blood pressure in the adult horse is 100 to 125 mm Hg. Results of a study equating systolic blood pressure with survival in horses with colic revealed that horses with a systolic pressure of less than 80 mm Hg generally have a poor prognosis. Note: Prolonged hypotension (systolic pressure <70 mm Hg) for several hours will usually lead to irreversible tissue damage.*

Peripheral Perfusion

- Peripheral perfusion is best assessed by color and refill time of the gingival mucosa.
- The normal color is pale pink and the normal capillary refill time is 1 to 2 seconds.
- Dehydration and endotoxemia will be associated with altered perfusion and cardiovascular dysfunction, with resultant prolongation of capillary refill time and alteration in the color of the gingival mucosa.

Gut Sounds

- Normal sounds are gurgling associated with fluid mixing with gas.
- Auscultate at least four sites, including the dorsal and ventral abdomen, on both sides of the horse (see Figs. 1-20 and 1-21).
- Altered sounds may indicate the severity of involvement.
- In the right paralumbar fossa, ileocecocolic sounds, or "water down a drain pipe," are heard, usually one to three per minute.
- Obstructions cause decreased peristalsis.
- Spasmodic contractions result in hyperperistalsis.

KEY POINT

Gastrointestinal sounds should be reassessed frequently in a horse with continuing abdominal pain.

Rectal Examination—Important Points Relating to Signs of Colic

When performing a rectal examination in a horse with signs of colic, the following points should be noted:

- Distended bowel, altered texture, tight bands
- Peritoneal surfaces—rough texture and fibrin tags indicate pathology. Also, gritty surfaces and empty feel to abdomen are suggestive of a ruptured viscus
- Location of the base of the cecum, feeling for the presence of tympany or impactions
- Medial displacement of the caudal edge of the spleen and large intestine in the nephrosplenic space that is suggestive that the large colon is displaced laterally and dorsally over the nephrosplenic ligament.

KEY POINT

Always attempt to locate and examine the pelvic flexure because it is a frequent site of impaction.

This structure is mobile and may reside in a number of positions but is not necessarily displaced. The inguinal rings should be checked for herniation, especially in stallions.

DIAGNOSTIC AIDS

Passage of a Stomach Tube

- Because the horse cannot vomit, passage of a nasogastric tube allows decompression of the stomach and anterior gastrointestinal tract.
- Introduction of a tube into the stomach will provide a passage for reflux of gas and fluid. This reduces pain associated with distension and aids in prevention of gastric rupture. In general, proximal lesions in the gastrointestinal tract result in the most rapid accumulation of fluid. Regular manipulation of the tube and priming with water are necessary to ensure gastric decompression (see Figs. 7-10 to 7-13).
- In horses with severe pain, nasogastric intubation should be performed at the initiation of the examination so that if gastric distension is present, gastric rupture can be prevented.
- In cases where reflux persists, the tube can be left indwelling by taping it to the halter or suturing it to the nostril.

KEY POINT

It is important not to introduce medications into the stomach via the nasogastric tube before attempting decompression.

Abdominocentesis

- Abdominocentesis is an easy and reliable way of assessing the condition of the abdominal contents (see section on diagnostic aids).

KEY POINT

Abdominocentesis is indicated in all cases of severe, persistent, or recurrent colic.

- A number of abnormalities may be found in fluid (see Table 7-1), but one of the most important and simple evaluations that can be done rapidly with a refractometer is measurement of the total protein concentration.

ASSESSMENT AFTER INITIAL EXAMINATION

KEY POINT

Most horses with colic recover spontaneously or respond to simple medical management and do not require surgery.

The typical colic case shows signs of mild intermittent pain with moderate elevations in heart rate and normal gut sounds. Blood pressure remains normal or is mildly elevated, and the capillary refill time and mucous membrane color are normal. There are no abnormalities detected on rectal examination or reflux when gastric intubation is performed. Several studies have shown that less than 2% of horses afflicted with colic subsequently require surgical intervention. Therefore, although advances in fluid therapy, anesthesia, and surgery have increased survival rates in horses with severe colic in recent years, animals requiring surgery still represent a minority of colic cases.

After the initial examination, it is more the exception than the rule to have arrived at a definitive diagnosis. Usually, the clinician will have sorted out a list of problems and their possible causes and have arrived at a plan for subsequent therapeutic and/or diagnostic measures.

ADDITIONAL DIAGNOSTIC TESTS

Depending on the severity of the colic case, other diagnostic tests may be indicated.

Hematocrit and Total Protein

- These procedures provide a relatively easy and accurate method for assessing the hydration status. Some general principles when assessing PCV and total plasma protein (TPP) are presented in Table 7-3.

Blood Gas Analysis

- Many gastrointestinal abnormalities will produce a metabolic acidosis with respiratory compensation. Typical values are pH 7.3 units, Pco_2 35 mm Hg, and $HC0_3^-$ 15 mmol/L (mEq/L). Although more precise interpretation of acid-base status is possible using arterial samples, analysis of jugular venous blood gas samples provides accurate information about the horse's metabolic condition. Rapid deterioration in acid-base status often is associated with a poor prognosis.

Serum/Plasma Electrolyte and Biochemistry Profiles

- Because colic usually has a relatively rapid course and fluid losses are generally isotonic, substantial alterations in plasma electrolyte values are uncommon. Some exceptions exist. Gas-

TABLE 7-3. Interpretation of Hematocrit and Plasma Total Protein Changes Associated with Colic in Horses

PCV (%)	Total Plasma Protein (g/L)	Indications for Fluid Therapy
<40	<75 (<7.5 g/dL)	None required; observe for deterioration
40-45	75-85 (7.5-8.5 g/dL)	Intravenous isotonic fluids indicated (20-40 mL/kg); note any continued deterioration
45-55	85-95 (8.5-9.5 g/dL)	Intravenous fluid definitely required (40-60 mL/kg)
>55	>95 (>9.5 g/dL)	Rapid and large-volume intravenous fluid therapy required (60-100 mL/kg)

Note: Few horses with PCVs > 60% and TPPs > 100 g/L (10 g/dL) survive, regardless of therapy.

tric dilatation produces sequestration of fluids and HC1, leading to dehydration, hypochloremia, and alkalosis. Anterior enteritis leads to substantial gastric reflux with large fluid, electrolyte, and protein losses. Hypocalcemia may accompany these changes. Large colon obstructions produce progressive dehydration and a decrease in plasma Cl due to sequestration in the intestinal lumen.

Prerenal azotemia, due to increases in serum urea nitrogen and creatinine concentrations, is common in horses with complicated abdominal disease. This usually is due to the combined effects of hypovolemia and endotoxemia.

Changes in activities of serum enzymes may be reflective of certain disorders in horses with colic. Increased serum AP activity results from its liberation from damaged intestinal walls and leukocytes. This is a common feature of complicated abdominal disease in the horse.

Increases in serum GGT activity provide a good screening test for hepatic dysfunction.

Increased bilirubin concentration may reflect inappetence, hemolysis, or a hepatopathy. Mild increases are a feature of reductions in feed intake, whereas larger elevations often accompany hepatopathies and hemolysis.

Elevations in the serum bile acid concentration accompany cholestasis.

Muscle damage associated with pain and self-inflicted trauma may result in increased serum creatine phosphokinase, AST, and LDH activities.

Is Surgery Necessary?

Some guidelines relating to the decision to refer for intensive therapy are given in Table 7-4. Early decisions should be taken about surgical management, because the more long standing the problem, the less likely of a successful long-term surgical outcome.

MEDICAL MANAGEMENT OF COLIC

In addition to abdominal pain, gastrointestinal diseases result in alterations in gut motility, obstructions, vascular compromise of affected abdominal organs, endotoxemia, and shock. If untreated, these may lead to debility or death of the patient. Considering these factors, a clinician must approach the medical management of colic in a logical manner and use appropriate medications.

Control of Pain

Analgesia is one of the most important aspects in the management of colic. Although analgesic drugs relieve many of the physical signs of pain, their administration must be tempered, given that they may mask signs of progression of the disorder.

NONSTEROIDAL ANTI-INFLAMMATORY DRUGS

NSAIDs inhibit cyclooxygenase, an enzyme involved in the arachidonic acid cascade, which is integrally involved in the production of prostaglandins, many of which are potent mediators of abdominal pain in horses. NSAIDs constitute one of the most frequently used groups of drugs in the treatment of abdominal pain. They exert their effects by reducing the concentration of inflammatory mediators at the site of the lesion, decreasing the perception of pain by the central nervous system, and reducing fever. Phenylbutazone (Treatment No. 89), flunixin meglumine (Treatment No. 52), dipyrone (Treatment No. 36), and ketoprofen (Treatment No. 66) are the most com-

TABLE 7-4. Guidelines for Referral for Possible Intensive Care and Surgical Intervention

Referral for Surgery Indicated	Sign/Clinical Manifestation	Continued Medical Management Indicated
Depressed	Attitude	Alert, responsive
Moderate-severe, progressive	Severity of pain	Mild-moderate, transient
HR > 75/min, pulse—weak, CRT > 2.5 sec, injected mucous membranes, cold extremities, "shock"; normal/slightly increased temperature	Cardinal signs	HR <55/min, pulse—strong, CRT <1.5 sec, pink mucous membranes, warm extremities; pyrexia—persistent
Ileus, no feces passed	Peristalsis	Peristalsis present, passage of feces
>5 L ± rapid reaccumulation of fluid	Nasogastric reflux	<2 L
Bowel distension/displacement	Rectal palpation	No abnormalities detected
See Table 7-1	Abdominal paracentesis	See Table 7-1
Moderate-severe elevation (see Table 7-3)	PCV and TPP	Mild elevation (see Table 7-3)
Poor: continuing pain, metabolic deterioration, progressive CNS depression	Response to therapy	Good: decreased pain, improved cardinal signs and hydration status

HR, heart rate; CRT, capillary refill time; C NS, central nervous system.

monly used NSAIDs. Dosage schedules are provided in Tables 7-5 and 18—4. Phenylbutazone and dipyrone have mild effects when used to relieve abdominal pain, whereas flunixin meglumine and ketoprofen exert more profound analgesic effects. The latter two drugs appear to be the most potent NSAIDs for control of visceral pain, with effects lasting up to 8 hours. A disadvantage of ketoprofen and flunixin meglumine is their duration of actions, which may mask the progression of clinical signs associated with potentially devastating gastrointestinal lesions. Drugs that mask signs and therefore delay the decision for referral for surgery should be used with caution. Frequent reassessment of the horse's condition should be made when using these drugs. If signs of pain persist after the use of these drugs or signs of pain reappear 6 to 8 hours after administration, a more complete diagnostic workup or referral to a hospital facility may be warranted.

SEDATIVE/ANALGESICS

Xylazine (Treatment No. 109) and detomidine (Treatment No. 28) are α_2-adrenoceptor agonists that induce analgesia (by suppression of central nervous system neurotransmission) and muscle relaxation. At high doses (1.1 mg/kg IV or IM), xylazine is a potent drug for the control of abdominal pain, with effects lasting up to 45 minutes. In many cases where pain is less intense, lower doses (0.3-0.7 mg/kg IV or IM) provide effective analgesia. Many clinicians routinely use lower doses, because the short duration of action allows repeated assessment of the animal's condition without long-term masking of signs. The most potentially serious side effects of α_2-adrenoceptor agonist drugs are hypotension, decreased cardiac output, ileus, and decreased intestinal blood flow, all of which are dose dependent. Detomidine and romifidine are more potent than xylazine, with analgesia and sedation lasting up to 90 minutes. Although these drugs produce similar side effects to xylazine, doses producing equipotent analgesic effects induce fewer detrimental effects on cardiovascular function and gastrointestinal motility. Dosage information for xylazine, detomidine, and romifidine are provided in Tables 7-5 and 18—4.

NARCOTICS

Although nonnarcotic analgesics (e.g., flunixin meglumine and xylazine) are now used more commonly for the treatment of abdominal pain in horses, narcotics retain a place in the treatment of abdominal pain because of their potent analgesic and sedative effects. Morphine, oxymorphone, and meperidine all have been used. Another agent, butorphanol (Treatment No. 15), has the most predictable analgesic effects and, apart from potentiating the effects of the α_2-agonists on gastrointestinal motility, have no significant adverse side effects.

TABLE 7-5. Drugs Utilized for the Medical Management of Colic

Drug	Action/Drug Group	Dosage	Side Effects
Phenylbutazone	NSAID	2.2-1.4 mg/kg IV q12-24h	Gut mucosa and renal toxicity
Flunixin meglumine	NSAID	0.25 mg/kg IV, IM q6-8h 0.5-1.1 mg/kg IV, IM q12-24h	At higher doses may mask signs of endotoxemia; gut mucosa and renal toxicity
Dipyronc	NSAID	5-22 mg/kg IV, IM q12-24h	Limited pain relief
Xylazine	Sedative/analgesic	0.3-1.1 mg/kg IV, IM as required	Hypotension, decreased cardiac output, ileus, GIT blood flow
Detomidine	Sedative/analgesic	10-20 μg/kg IV as required	Hypotension, cardiac output, ileus, GIT blood flow
Butorphanol	Opioid analgesic	0.05-0.1 mg/kg IV, IM as required 0.01-0.06 mg/kg IV, IM in combination with xylazine (0.2-0.4 mg/kg IV, IM)	Excitement at higher doses, possibly GIT motility
Mineral oil	Laxative	5-10 mL/kg PO q12-24h	Contraindicated when reflux or complete obstruction present
Psyllium hydrophilic mucilloid	Laxative	1 g/kg PO q6-24h	Prolonged use may result in efficacy
Dioctyl sodium sulfosuccinate	Detergent/laxative	10-20 mg/kg POq48h as a 5% solution	Concentrated solutions and increased dosing frequency may result in diarrhea
Neostigmine	GIT motility stimulant	0.02 mg/kg SC q30min for up to 5 doses	Pain, sweating, inappropriate changes in motility
Bethanechol	GIT motility stimulant	0.025 mg/kg SC q6-8h for 3-4 doses Then 0.3-0.4 mg/kg PO q6-8h	Sweating, pain, inappropriate GIT motility
Dimethyl sulfoxide	Anti-inflammatory agent, free-radical scavenger	0.1-1.0 g/kg IV q12-24h as a 10-20% solution for 2-3 days	Hemolysis

GIT, Gastrointestinal tract.

KEY POINT

Butorphanol combined with an a_2 agonist (xylazine) provides good analgesia that lasts for 10 to 90 minutes, depending on dose and cause of the pain.

Many clinicians combine xylazine (up to 0.2-0.4 mg/kg IV) with butorphanol (0.01-0.1 mg/kg IV) to provide improved analgesia in horses with severe abdominal pain (see Table 7-5). Morphine, oxymorphone, and meperidine are potent analgesics but have the unfortunate side effects of paradoxical excitation, increased activity/agitation, and a reduction in gastrointestinal transit time.

LAXATIVES/LUBRICANTS

KEY POINT

Softening (hydration) and lubrication of impactions in the large colon are the usual indications for administration of lubricants.

A combination of fluid therapy (intravenous or oral) and administration of inert lubricating agents usually achieve hydration and passage of intraluminal obstructions/impactions.

KEY POINT

Care should be taken not to administer oral fluids to horses with gastric reflux.

Large volumes (100-120 ml/kg) of intravenous fluids are effective in softening many impacted masses. Methods for administration and objectives of fluid therapy are discussed elsewhere in this chapter and in Chapter 18.

Mineral oil (Treatment No. 77) is the lubricant of choice of many equine clinicians. It is a surface lubricant that facilitates the passage of ingesta through the gastrointestinal tract by direct lubricant effects and reduction of intestinal water absorption. The latter effect increases the hydration of intraluminal contents. The therapeutic efficacy of mineral oil is limited to the treatment of mild obstructions. Dose rates of 5 to 10 mL/kg orally every 12-24 hours usually are prescribed (see Table 7-5).

Psyllium hydrophilic mucilloid (Treatment No. 98) is administered orally and absorbs water, acting as a laxative by increasing the water content and bulk of the fecal mass. Psyllium can be administered safely up to four times per day. The most common indication for this agent is in the management of sand ingestion. After diagnosis of sand impaction, psyllium (1 g/kg PO q24h) should be prescribed for several weeks to encourage the expulsion of sand from the large colon. Longer term therapy is thought to result in decreased effectiveness because of a more rapid microbial degradation of the mucilloid in the colon.

Dioctyl sodium sulfosuccinate (DSS; Treatment No. 35) is a detergent designed to decrease the surface tension of intraluminal masses and allow increased penetration of water and ions. The dose rate is 10 to 20 mg/kg orally every 48 hours as a 5% solution (see Table 7-5). Higher dose rates, more frequent administration, or the use of more concentrated solutions can result in diarrhea.

Magnesium sulfate has been a popular laxative in years past. It is now used less frequently because of its potential toxic effects on the gastrointestinal mucosa.

AGENTS AFFECTING GASTROINTESTINAL MOTILITY

Ileus is defined as a functional obstruction of the gastrointestinal tract and is characterized by a loss of coordinated propulsive motility of the stomach and intestine. Shock, electrolyte imbalances, hypoalbuminemia, peritonitis, endotoxemia, together with distension, ischemia, and inflammation of the intestinal tract have all been incriminated as causes. As a result, attempts have been made to find agents that assist in promoting gastrointestinal motility (see Table 7-5).

Adrenergic receptor antagonists block the depressive effects of sympathetic tone on the gastrointestinal tract. Agents used in the horse include propranolol, yohimbine, acepromazine, and phenoxybenzamine. There is little evidence that these drugs are effective clinically.

Cholinergic agonists promote parasympathetic tone and consequently are proposed to increase gastrointestinal motility. The drugs that have been used in the horse include bethanecol and neostigmine. Bethanecol alone has no apparent therapeutic benefit. Neostigmine (Prostigmin, Hoffman-LaRoche, Nutley, NJ) administered subcutaneously (0.02 mg/kg, q 30 min) clinically appears to promote large intestinal motility and induce defecation. However, neostigmine has been shown to delay gastric emptying and has minimal effect on small intestinal motility and is therefore contraindicated in most motility disorders in the horse.

The effects of benzamides are not understood. The most commonly used benzamides in the horse include metoclopramide and cisapride. Metoclopramide (0.25 mg/kg diluted in 1 L saline delivered by infusion over 1 hour, q6h) has been shown to promote gastrointestinal transit experimentally. Side effects reported include centrally mediated excitement. Recently, a continuous infusion (0.04 mg/kg/h) has been found to decrease the rate, volume, and duration of gastric reflux and increased survival to discharge in a series of horses that underwent resection and anastomosis of the small intestine. Using a continuous infusion, no adverse side effects were noted.

Repeated doses of cisapride (0.1 mg/kg, IM, PO, or per rectum, q8h) have been shown experimentally to promote gastrointestinal motility in experimental ileus. In uncontrolled clinical trials, similar doses have been shown to reduce the prevalence of postoperative motility disorders where an underlying persistent endotoxemia is not a problem.

Macrolide antimicrobials, such as erythromycin, act as a motilin receptor agonist. In horses, erythromycin lactobionate (0.5-2.2 mg/kg, IM or IV, q6h) has been used for the treatment of motility disorders. Some clinicians dilute the drug in saline and deliver the dose intravenously over 1 hour. Higher doses and continuous infusions have

been postulated to be contraindicated because they may cause downregulation of motilin receptors.

Local anesthetics, such as lidocaine hydrochloride (1.3 mg/kg), delivered as a bolus by slow intravenous injection followed by a continuous infusion (0.05 mg/kg/min IV) for 24 hours has been suggested to be effective in treating motility disorders.

KEY POINT

There are a variety of drugs available for treating motility disorders in the horse. Unfortunately the efficacy of most of these drugs in treating ileus is still not clear.

NSAIDs ameliorate some of the toxic side effects of endotoxemia invoking ileus. Flunixin meglumine (Treatment No. 52) is the most commonly used NSAID in the treatment of gastrointestinal tract disorders. Administration of lower doses (0.25 mg/kg q6-8h IV) of this drug results in a reduction of many untoward cardiovascular effects of endotoxemia without posing the risk of masking of signs of deterioration or inducing toxicity.

Dimethyl sulfoxide (DMSO; Treatment No. 34) is a solvent that is thought to exert good tissue penetration and anti-inflammatory effects. This latter effect is purported to be the result of the scavenging of free radicals produced by inflammatory cells. Empirical studies indicate that DMSO given to horses with endotoxemia and subsequent to bowel ischemia (0.1-1.0 g/kg q24h slowly IV as a 10%—20% solution) may improve the chances of survival when compared with untreated horses.

FLUID THERAPY

Large-volume fluid therapy must be strongly considered in horses with severe abdominal pain or gastrointestinal disease showing evidence of compromise of cardiovascular function. Fluid therapy is discussed elsewhere in this chapter and in Chapter 18.

KEY POINT

Fluid therapy is also valuable when treating intraluminal obstructions, particularly impactions of the large colon.

In these cases, therapy is designed to augment cardiovascular function and to assist in increasing the volume of fluid in the gastrointestinal tract. This increase in fluid serves to assist in hydration and maceration of the impacted mass. In cases where there is only partial obstruction of the lumen, as evident by the passage of small amounts of feces, and no gastric reflux, fluids administered by nasogastric tube may provide a convenient and inexpensive means of providing fluid support. An adult horse can tolerate volumes of 6 to 8 L of isotonic fluid administered via nasogastric tube every hour.

KEY POINT

If complete obstruction or gastric reflux is present, fluids should not be administered by nasogastric tube and nasogastric fluid administration should be stopped if there are signs of abdominal pain shown during or immediately after administration.

Diseases of the Oral Cavity

Cleft Palate

Cleft palate is a disorder that is most commonly recognized in young foals, resulting in ill thrift and discharge of food from the nostrils. The etiology of this disorder is unknown but it is a congenital condition.

HISTORY AND PRESENTING SIGNS

- Foals usually presented because of milk or water discharging from the nares.
- Failure to gain weight at an appropriate rate.

CLINICAL FINDINGS AND DIAGNOSIS

- On physical examination, foals are frequently in poor body condition, may have secondary pneumonia due to inhalation of material into the lungs, and, as a result, lung sounds can be harsh and also there can be nasal discharge.
- When foals attempt to drink, milk often discharges from the nares.
- The disorder usually is diagnosed on the basis of clinical signs and by visual inspection.
- A cleft palate can vary in degree from fissures in the soft and hard palates to only a small cleft in the posterior soft palate.
- Lesions can be defined either by oral examination or via endoscopic examination of the mouth and palate.

DIFFERENTIAL DIAGNOSIS

- Pneumonia from other causes
- Dysphagia

- Megaesophagus
- Choke

TREATMENT

- The fundamental principle of treatment is surgical repair of the deficit in the palate. Usually this is successful only in cases where there is a small cleft in the palate.
- Because access to the palate requires a mandibular symphysiotomy, it is important to assess the foal's general state of health before general anesthesia and surgery. This assessment will involve determination of the degree of aspiration pneumonia that may have occurred, a process usually requiring careful auscultation, hematology, and thoracic radiographs.

KEY POINT

The surgical procedure is relatively complex and has a reasonably high risk of complications. As a result, most foals with a cleft palate that are likely candidates for surgical repair are sent to referral centers for treatment.

Cystic Sinuses

This is a condition in which there appears to be multiple cyst formation, usually within the nasal turbinate region.

HISTORY AND PRESENTING SIGNS

- Usually occurs in young animals.

CLINICAL FINDINGS AND DIAGNOSIS

- Maxillary, mandibular, or facial distortion.
- In severe cases, the nares become partially occluded, resulting in dyspnea. In such cases, "kissing" lesions in the nasal mucosa may produce secondary ulceration.
- Diagnosis is based on radiographic changes, and in early cases, differentiation between dentigerous cysts and cystic sinus disease may be difficult.

DIFFERENTIAL DIAGNOSIS

- Dentigerous cysts
- Secondary hyperparathyroidism
- Dental tumors
- Trauma
- Retained tooth caps
- Paranasal sinus cysts

TREATMENT

- In severe cases, attempted treatment is usually unrewarding.
- Some animals survive for protracted periods if the cysts are small and relatively noninvasive.

KEY POINT

Surgery via maxillary and/or frontal sinus flaps to enable curettage of the cysts and associated structures may give excellent results in many milder cases. Note that hemorrhage in such cases can be severe.

Dentigerous Cysts

Dentigerous cysts are tumor-like structures of epithelial origin that frequently contain structures with tooth-like appearance. When cysts occur on the head, they are considered important because of their potential to disfigure the affected animal.

HISTORY AND PRESENTING SIGNS

- Usually in younger animals.
- Presence of a lump or facial/mandibular distortion is commonly reported.

CLINICAL FINDINGS AND DIAGNOSIS

- The most common site for dentigerous cysts is in the area near the ear, where they are referred to as temporal cysts.
- Discharge from these cysts draining either into or near the ear may be seen.
- Other sites for dentigerous cysts include the maxillary sinus and elsewhere on the face.

KEY POINT

Diagnosis is based on the presence of characteristic lesions in younger horses. The lesions are usually located in temporal, aural, or maxillary sinus regions.

- In some cases, a draining tract may be present.
- Radiographs usually reveal a cystic cavity with a high likelihood of dental-like structures within the cavity.

DIFFERENTIAL DIAGNOSIS

- Retained tooth caps with secondary alveolar periostitis
- Dental or paranasal sinus tumors
- Other causes of maxillary or facial deformity

(e.g., nutritional secondary hyperparathyroidism)
- Local abscesses
- Trauma
- Paranasal sinus cysts

TREATMENT

- Resection of the dentigerous cyst is the usual course of action, with particular care being taken to remove all the lining of the cystic cavity.
- After removal, the cavity is packed and the dead space is allowed to heal by second intention.

Gingivitis/Stomatitis

A number of conditions, including bacterial, viral, and mycotic diseases, can cause primary stomatitis, whereas other diseases (e.g., periodontal disease) may result in secondary manifestations of stomatitis.

HISTORY AND PRESENTING SIGNS

- Inappetence or reluctance to eat
- Depression
- Salivation
- History of NSAIDs administration

CLINICAL FINDINGS AND DIAGNOSIS

- Generalized swelling and redness with ulceration noticed around the stomal and gingival tissues.
- In severe cases, there can be large ulcerative lesions that result in increased salivation and substantial discomfort to the horse, producing a reluctance to eat and drink.
- Severely affected animals may become progressively dehydrated because of their reluctance or inability to consume fluids.
- The most common causes of stomatitis in horses are vesicular stomatitis, horse pox, candidiasis, and possibly infections caused by *Pseudomonas* and *Rhodococcus* spp.
- Secondary stomatitis may accompany phenylbutazone toxicity, photosensitization, uremia, and possibly mercury toxicity. Other causes of stomal lesions may include lampas, where there is a swelling of the palate just caudal to the incisors. This process occurs in young horses during eruption of the permanent incisors. Lampas has clinical manifestations similar to those occurring in horses with stomatitis. Affected animals may be reluctant to eat and may salivate excessively when eating.

DIFFERENTIAL DIAGNOSIS

- Primary or secondary stomatitis
- Lampas
- Neoplasia
- Dental problems (e.g., retained tooth caps, malocclusion, enamel points, periodontal disease)

TREATMENT

- If treatment of primary stomatitis is undertaken, it involves attempts to reduce the local inflammatory response, including lavage of the mouth with mild antiseptic solutions (e.g., 0.05% chlorhexidine, povidone-iodine).
- In cases where phenylbutazone toxicosis is the cause of the stomatitis, the drug should be discontinued.
- In animals that are severely affected, systemic nonsteroidal anti-inflammatory therapy and antibiotic coverage may be required. We commonly use phenylbutazone at a dose rate of 2.2 mg/kg ql2h and procaine penicillin at 15,000 to 20,000 IU/kg (15-20 mg/kg) ql2h.
- Those animals reluctant to eat or drink may require fluid administration either via a nasogastric tube or intravenously (see Chapter 18).
- Horses with secondary stomatitis require diagnosis and treatment of the primary disease to allow management of the secondary problems.
- Lampas is temporarily debilitating and resolves spontaneously.

Oral Ulceration

A number of conditions result in oral ulceration in the horse. Ulcers can be asymptomatic or cause dysphagia and painful mastication, thereby producing inappetence. Lesions are described by a variety of terms, including vesicles, ulcers, crusts, and growths. Sites where lesions occur include the lips, tongue, gingiva, palate, and pharynx.

HISTORY AND PRESENTING SIGNS

- The main infectious cause of oral ulceration is the vesicular stomatitis virus.
- Other causes include phenylbutazone toxicity, yellow bristle grass ingestion, ulcers due to plant thorns, and oral foreign bodies.
- Other causes include the ingestion of toxic chemicals (e.g., when horses lick mercury blisters off their legs, after ingestion of blister beetles), as a consequence of periodontal disease, and also possibly as a result of uremia.

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CLINICAL FINDINGS AND DIAGNOSIS

- Horses with vesicular stomatitis first develop small vesicles in the mouth, and then these vesicles turn into large ulcers. The tongue often is severely involved.
- There may be excessive salivation and fever associated with the early stages of the condition.
- Diagnosis usually is based on the presence of lesions within the mouth, on the lips or gums, or in the pharynx. In the case of toxicity, there is a history of ingestion of toxic materials such as high doses of phenylbutazone.
- The oral cavity should always be checked for plant thorns and foreign bodies and for the presence of periodontal disease.
- Horses that have ingested blister beetles often will have other systemic signs, such as enteritis, polyuria, and hypocalcemia.
- Clinical pathology readily will determine the presence of uremia.

DIFFERENTIAL DIAGNOSIS

- Vesicular stomatitis
- Phenylbutazone toxicity
- Foxtail or plant thorn stomatitis
- Oral foreign body
- Ulcers secondary to yellow bristle grass
- Chemical stomatitis
- Periodontal disease
- Blister beetle toxicosis
- Uremia

TREATMENT

KEY POINT

Vesicular stomatitis has an appearance similar to foot and mouth disease, although horses are not affected by foot and mouth disease. However, it is important that vesicular stomatitis is appropriately diagnosed and government authorities notified.

- If vesicular stomatitis is not suspected and another cause of oral ulceration is most likely, treatment strategies should involve local therapy to relieve the oral irritation, such as mouthwashes, removal of plant thorns or foreign bodies if present, and removal of any chemical irritants that may be causing oral ulceration (e.g., phenylbutazone).
- If the oral ulceration is secondary to periodontal disease, appropriate dental prophylactic measures (e.g., removal of affected teeth) should be undertaken.

Salivary Gland Diseases

Diseases of the salivary glands or their ducts are relatively rare, with the most common disorder being trauma to the parotid duct or sialoliths.

HISTORY AND PRESENTING SIGNS

- History of trauma (e.g., kicks, wire cuts) or surgery to the parotid or guttural pouch region.
- Owners may report salivary discharge, particularly in association with feeding.
- Horses with sialoliths usually present with localized firm swelling of the cheek. The parotid salivary gland may also be swollen and painful if the condition is acute and the duct is completely blocked.

CLINICAL FINDINGS AND DIAGNOSIS

- Affected horses will usually have evidence of trauma to the area of the mandible. Discharge of saliva from the wound is common.
- Trauma to the parotid salivary gland also may occur if the animal receives an injury caudal to the angle of the mandible near the location of the guttural pouch.
- The discharge of saliva from the wound is most profuse when the animal eats, when saliva production is at its greatest.
- If the duct ruptures subcutaneously after trauma, saliva may accumulate in that area, and the horse may be presented with a swelling in the mandibular region.
- Diagnosis usually is based on a history or evidence of previous trauma and the discharge of saliva from the wound. If the fluid is accumulating subcutaneously, a salivary mucocele can be differentiated from a seroma by aspiration of the fluid. Saliva has much higher concentrations of calcium and potassium than is found in most other fluid accumulations.
- A diagnosis of a sialolith can be made on a physical and oral examination, but radiographs of the mass can confirm this diagnosis.

DIFFERENTIAL DIAGNOSIS

- Trauma (e.g., seroma, hematoma, or fracture)
- Sialolithiasis
- Bone sequestrum and local osteitis
- Mandibular or guttural pouch abscessation
- Neoplasia

TREATMENT

- Wounds to the parotid gland usually require appropriate cleansing, debridement of devitalized tissue, and suturing.

- To promote healing and decrease gland secretion, the horse is usually fed via a nasogastric tube and given an electrolyte mix and gruels for several days to decrease parotid gland secretion.
- Sialoliths are removed surgically. An incision is made over the mass from the buccal side and the sialolith removed. The wound is left to heal by second intention.

KEY POINT

Salivary gland fistulas occurring as a result of trauma to the parotid duct often will heal spontaneously.

- However, those that do not heal after several weeks probably require surgical management with the aim to either attempt to reestablish the duct or destroy the salivary gland.
- An incision is made down to the duct and the duct is isolated. An appropriate-diameter medical-grade silicone or polypropylene tube is passed into the distal portion of the duct and tunneled into the oral cavity. The tube is secured to the buccal mucosa. The tube is allowed to remain in place for approximately 2 weeks and is then removed. Alternatively, chemical involution of the gland can be performed. Chlorhexidine (2%), silver nitrate, and 10% formalin have been used to chemically involute the salivary gland. Formalin (35 mL of 10% solution) produces effective involution with minimal necrosis and suppuration. The duct is catheterized and the formalin infused and held within the gland for 90 seconds. The solution is then allowed to drain. Swelling and pain are a feature for several days and can be managed with NSAIDs. Complete involution can be expected within 3 weeks.

Trauma to the Lips and Tongue

The most common injuries to the lips and tongue involve direct chemical irritation from exposure to caustic substances or injury to these structures by foreign-body penetration or direct trauma. The tongue and lips often are injured by wire, nails, pieces of wood, and plant thorns. Injury also can be iatrogenic secondary to dental procedures or overzealous manipulation of the tongue. Most injuries are minor, but at times damage can be severe.

HISTORY AND PRESENTING SIGNS

- Reluctance to eat
- Blood or saliva discharging from the mouth
- History of trauma or overzealous grasping of the tongue during oral/dental procedures

CLINICAL FINDINGS AND DIAGNOSIS

- Increased salivation. Sometimes the mouth will remain open, and evidence of trauma will be obvious. The tongue may hang out.
- Lacerations and foreign bodies within the tongue usually produce substantial pain in the horse. There may be reluctance to eat, evidence of salivation, or evidence of blood coming from the mouth.
- In some cases, the damage to the tongue will be readily obvious.
- Given the extensive blood supply to the tongue, the potential for hemorrhage after lacerations is relatively great.
- Diagnosis of lingual lacerations or foreign bodies is usually made on the basis of careful visual and physical examination.
- Lacerations are usually self-evident.
- Foreign bodies (e.g., wire or wood) in the tongue or lips may be difficult to locate. The only clinical sign may be local swelling and a draining sinus. Careful palpation of the tongue is often required to define the presence of a foreign body.
- Palpation of the region and possibly radiographs using contrast techniques after infusion of contrast media into the draining tract may be required to confirm the diagnosis.
- Tears, swelling, and pain in the region of the frenulum of the tongue may be visualized. Sublingual cellulitis also may be present. Sublingual cellulitis commonly occurs after damage to the frenulum resulting from overzealous forceful exteriorization of the tongue during dental or oral procedures. Diagnosis is based on visualization of damage to the frenulum and local swelling.
- Ingestion of yellow bristle grass. Thorns from the grass lodge in the oral cavity and produce granulating wounds.
- Diagnosis of injuries to the tongue usually is based on direct physical examination.

DIFFERENTIAL DIAGNOSIS

- Trauma to the tongue
- Lingual foreign bodies
- Sublingual cellulitis
- Primary or secondary stomatitis
- Oral ulceration
- Lampas
- Periodontal disease
- Dysphagia
- Botulism
- Nigropallidal encephalomalacia

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TREATMENT

- Most lacerations of the lips and tongue, if small, will heal spontaneously with little direct treatment. However, severe contusions of these structures require wound debridement and suturing.
- Primary repair of the tongue is possible in fresh wounds and ensures good apposition of the various layers of tissue. However, in many cases lacerations to the tongue are not discovered until there is severe contamination, making primary repair impossible. Healing by second intention is generally good. Horses can manage with severe tongue deficits.
- In some severe cases of tongue laceration, where the tip of the tongue is almost severed, partial glossectomy may be indicated.
- When performing the partial glossectomy, care should be taken to ensure that large vessels are appropriately ligated to prevent the potential for significant hemorrhage. If the lesion is small, only a wedge-shaped segment of the tongue may need to be removed.
- Healing of the tongue appears to occur rapidly and mostly is uncomplicated.
- Foreign bodies require surgical extirpation and suturing of the wounds.
- Additional treatment involves nonsteroidal anti-inflammatory therapy such as phenylbutazone (Treatment No. 89) at a dose rate of 2.2 mg/kg every 12 hours for 4 to 7 days. In severe cases, antibiotic coverage is indicated. Procaine penicillin (15,000-20,000 IU/kg [15-20 mg/kg] ql2h; Treatment No. 84) or trimethoprim-sulfonamide combinations (15-30 mg/kg of the combined agent PO ql2h; Treatment No. 108) are good choices.
- Nursing care is important, with provision of adequate fluids and palatable nutritious foods indicated.

Diseases of the Teeth

Dental disorders are relatively common in horses and frequently require veterinary attention. As a result, the attending veterinarian must have a good working knowledge of the dental formulas, structure of the teeth, and normal times for tooth eruption. Horses have an initial set of deciduous teeth that are subsequently replaced by permanent teeth. The times at which the deciduous teeth erupt in the foal are presented in Table 7-6, whereas the times for eruption of permanent teeth can be found in Table 7-7. Integral to the successful diagnosis of dental problems is the clinician's ability to perform a safe, thorough, and expedient examination of the dental arcades and oral cavity.

TABLE 7-6. Times for Eruption of Deciduous Teeth in the Foal

Tooth	Time of Eruption
Incisors	
1st deciduous	By 1 wk
2nd deciduous	1-2 mo
3rd deciduous	6-9 mo
Premolars	
1st deciduous	All within 2 wk
2nd deciduous	
3rd deciduous	

Because of their potential to interfere with the animal's ability to eat, dental problems often manifest as either a reluctance to eat or a spillage of food from the mouth during mastication. Sometimes the sharp edges on teeth may cause pain when the horse is being ridden with a bit in its mouth. Affected horses may show behavioral changes such as head shaking, failure to respond to commands, and other refractory behavior.

DENTAL EXAMINATION

An appropriate history should be obtained outlining the dietary habits, any evidence of quidding (spillage of food from the mouth during mastication), vices or foul breath, and taking note of the

TABLE 7-7. Times for Eruption of Permanent Teeth in the Horse

Tooth	Time of Eruption
Incisors	
1st permanent (I1)	2.5 yr (in wear 3 yr)
2nd permanent (I2)	3.5 yr (in wear 4 yr)
3rd permanent (I3)	4.5 yr (in wear 5 yr)
Canine	
Permanent (C)	4-5 yr
Premolars	
1st permanent (wolf tooth)	~6 mo
2nd permanent (P2)	2.5 yr
3rd permanent (P3)	3 yr
4th permanent (P4)	4 yr
Molars	
1st permanent (Ml)	~12 mo
2nd permanent (M2)	2 yr
3rd permanent (M3)	~4 yr

animal's age. A thorough dental examination is then performed. It is important that the horse is relatively relaxed, and in anxious individuals, mild tranquilization may be necessary. The first part of the examination involves visual inspection of the incisors, with note being made of abnormalities in bite, such as "parrot mouth" or "sow mouth," and whether there are deciduous teeth still present. If so, it should be determined whether those teeth are about to be shed. The veterinarian also should check for the presence of supernumerary teeth and sharp edges on the incisors. Some indication of the presence of sharp edges on the buccal edges of the cheek teeth, particularly those in the upper arcade, can be obtained by external palpation through the cheeks. After this initial examination, a more thorough visual and manual examination of the buccal cavity is performed.

KEY POINT

Visualization of the teeth can be enhanced by use of a light source and an angled mirror.

Several methods can be used. One method involves the use of a mouth gag or speculum (see Chapter 1), whereas the other involves a manual technique without a mouth gag. Techniques not using mouth gags are divided into a two-handed and a one-handed procedure.

Two-Handed Teeth Examination

With the two-handed procedure, the tongue is grasped on one side of the mouth through the interdental space and held in position on that side. The operator's other hand is then inserted into the mouth to feel the teeth and dental arcades on the opposite side to that to which the tongue is being held. When the tongue is held to the side, the horse usually will have its mouth open, and a light source can be used to enhance visual examination of teeth on the opposite side.

KEY POINT

Care must be exercised when pulling out the tongue not to be too aggressive, because damage to the frenulum on the ventral side of the tongue may occur, resulting in sublingual cellulitis.

The operator then inserts a hand between the dental arcade and the cheeks with the knuckles toward the cheek and the palm toward the teeth. The cheek teeth are then palpated using the fingers. The idea of this procedure is to have the tongue held in a position between the teeth on the opposite side so that if the horse attempts to bite the operator's hands, it will bite onto its own tongue first. Under *most* circumstances, this procedure will prevent the horse from biting the operator's fingers.

One-Handed Teeth Examination

An alternative method involves the one-handed technique, whereby the operator inserts his or her hand through the interdental space with the back of the hand forcing the tongue between the opposite dental arcade. The palm is then facing toward the teeth that the operator wishes to examine (see Figs. 1-3 and 1-4). Therefore, the hand will be lying between the lingual surface of the cheek teeth to be examined and the tongue. After insertion of the hand, the operator is able to use a thumb and forefinger to rapidly palpate the buccal, lingual, and table surfaces of the teeth. The operator also can examine areas of mucosa, gingiva, and parts of the tongue using this procedure. After examination of one side, the other hand is inserted through the opposite interdental space to facilitate examination of the opposite dental arcade. By inserting a hand, particularly the first finger and thumb, into the interdental space, the wolf teeth (first premolar teeth) and first upper and lower cheek teeth can be palpated. Particular note should be made of the presence of wolf teeth and whether there is any local irritation or the presence of sharp edges on the first cheek tooth.

Examination of Teeth with the Aid of a Mouth Gag

If use of a mouth gag is favored, we have found the Swales gag to be the simplest and most useful for examination of the teeth (see Figs. 1-6 and 1-7). This gag is safe for the veterinarian and horse and functions by forcibly holding the arcades open, allowing the operator to examine the dental arcade on the opposite side. Many clinicians also like to use the Hausmann gag, which is more cumbersome and in a difficult horse can act as a lethal weapon.

Examination of Teeth and Mouth under General Anesthesia

On occasion, the only means by which a satisfactory examination of the mouth and dental arcades can be achieved is after the induction of short-term general anesthesia (see Chapter 18). This allows more thorough evaluation because patient compliance is ensured.

Radiography

Radiographs of the head and teeth also may form an integral part of the dental examination. With care, adequate radiographs of the head and teeth can be obtained using portable equipment in the standing tranquilized horse. The quality of radiographs of the head and teeth has improved in recent years because of the advent of appropriate grids, films, and rare-earth screens. If greater detail is required, oblique or dorsoventral views are indicated; these procedures should be performed with the horse sedated or under general anesthesia (see Chapter 18).

TREATMENT PROCEDURES

Teeth Floating

Veterinarians frequently are required to undertake routine dental procedures such as teeth floating. Adequate attention to dental prophylactic procedures will provide a horse with much more satisfactory function when it is masticating and also will potentially prevent the onset of untoward sequelae such as gingivitis. Floating usually is performed without a mouth gag, and the mouth is opened by holding a thumb on the hard palate on the side opposite to that in which the clinician wishes to insert the float. Once the float is inserted and movement of the float commenced, the hand can be withdrawn from the head/hard palate because the float will tend to maintain the mouth in an open position. An alternative method involves insertion of a gag (Fig. 7-22).

KEY POINT

It must be remembered that when performing teeth floating in horses, the operator may have to stand directly in front of the horse and is thus vulnerable to being struck.



Figure 7-22. Rasping of upper premolar teeth using an angled rasp. Note that the rasp is positioned on the labial surface of the upper premolar teeth.

If the veterinarian feels particularly vulnerable, a horse rug can be tied around the neck of the horse, providing a shield if the horse attempts to strike (see Fig. 1-27).

In general, it is not necessary to grasp the horse's tongue when attempting to perform tooth floating. The objective of tooth floating is to remove enamel points or sharp projections on teeth that are causing problems with mastication or irritation to the horse's gums or lips. When floating the upper arcade, the buccal (labial) aspect of the arcade should be rasped at approximately a 60-degree angle, whereas on the lower arcade, the lingual aspect of the arcade should be floated. An angled float is used for the first few upper premolars, and a straight float is used on the caudal premolars, molars, and lower arcade. Particular care should be taken when rasping the teeth not to injure the mucosa. It is important to remove any sharp points or hooks that may be present on the rostral aspect of the first upper cheek tooth. This is usually done with a 45-degree float or a small hand-held file. In general, better control of the procedure is achieved if the float or file is held very close to the horse's head. Also, a hook on the first upper cheek tooth is often accompanied by a hook on the last lower cheek tooth.

Removal of Deciduous Teeth/Caps and Wolf (First Premolar) Teeth

Wolf teeth can be present on both the upper and lower arcades but are most commonly found on the upper arcade.

KEY POINT

There is often a strong and most likely unfounded belief within the equine industry that wolf teeth will create problems for young horses when working with bits in their mouths.

As a result, wolf-teeth extraction is a commonly practiced procedure, but one for which there may be limited basis. Logic would seem to dictate that wolf teeth probably only need to be removed if they are impacted, causing gingival irritation or damage to the lips, or if they are displaced in some way. If wolf teeth require removal, several techniques have been described. In most circumstances, a dental elevator is used to loosen the tooth, and the tooth is then removed using a pair of forceps. However, special wolf

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tooth extractors are available that are designed like a circular gouge to loosen the tooth within the gingiva and associated attachments.

Retention of deciduous caps frequently occurs in young horses. One indication for removal of caps is the presence of sharp edges (which can be detected by palpation), causing irritation to the gingival and buccal mucosa. Another indication for removal of caps is the significant periosteal reaction and pain that occurs in association with the emergence of the permanent premolars in the maxillary and mandibular arcades. This reaction often is associated with local deformation, pain, and difficulty for the young horse when eating. Caps usually are pried off using a bone elevator, a pair of forceps, or a pair of bone-cutting forceps. An alternative lies in the use of a high-quality screwdriver. In horses that resent manipulation of the oral region, injection of local anesthesia around the gum line of the affected tooth or application of topical anesthetic sprays can be used.

Large hooks and sharp points may require cutting with tooth cutters or a chisel or grinding with a dental grinder.

KEY POINT

When attempting to remove sharp points and hooks with tooth cutters or chisels, it must be emphasized not to try to cut too much of the tooth at one time because there is a high risk of cracking the remaining healthy tooth.

Tooth Removal

In cases where there is severe periodontal disease, affected cheek teeth may be loose enough to allow removal using a pair of large animal tooth forceps. The use of molar spreaders may loosen the tooth to facilitate extraction. The procedure usually requires general anesthesia, and if the attempt to remove the tooth with the forceps is unsuccessful, repulsion of the teeth using trephine techniques and retrograde removal of the tooth are usually the next course of action. This procedure is rather more complex than attempting to remove the tooth with tooth forceps, and procedures for tooth removal are described in a variety of equine surgical texts.

Dental Caries (Apical Infections, Pulpitis)

Dental caries is a disease in which there is destruction of the cementum in teeth by food and microorganisms, resulting in destruction of the integrity of the tooth. This is similar to the situation occurring in humans. Dental caries is a common disorder in older horses.

HISTORY AND PRESENTING SIGNS

- Commonly in horses over 5 years of age
- Often only noticed when there is maxillary or mandibular swelling
- Affected horses may have a history of quidding, pain on mastication, and/or foul smelling breath.

CLINICAL FINDINGS AND DIAGNOSIS

- Caries/pulpitis may be obvious on oral examination or may require use of a dental probe to make diagnosis.
- Most common in second and third cheek teeth.
- May see broken teeth.
- Pain associated with the affected tooth may be a feature.
- Nasal discharge, halitosis, and pain and swelling of paranasal sinuses occur in some cases.
- Diagnosis of secondary complications is frequently confirmed with radiographs.

DIFFERENTIAL DIAGNOSIS

- Dental/bone tumors
- Dentigerous cysts
- Periodontal disease
- Sinus cysts
- Paranasal sinusitis
- Trauma/osteomyelitis

TREATMENT

- Pulpitis is best prevented by regular dental attention to ensure appropriate mastication.
- Once disease has clinical manifestations (e.g., pulpitis, periostitis, or osteomyelitis), removal of the affected tooth and diseased tissue is the treatment of choice.

Enamel Points, Hooks, and Sharp Edges

The normal masticatory action of the horse produces wear on the teeth, often resulting in sharp enamel points on the premolars and molars.

KEY POINT

Teeth in the upper arcade are slightly wider apart than those in the lower arcade, thereby producing a greater number of sharp points on the outside of teeth (labial surface) in the upper arcade and on the inside (lingual surface) of lower arcade.

These points result in injury to the insides of the cheeks and/or lateral aspects of the tongue

during mastication. Pain during mastication also may be obvious. Enamel points are a common cause of unusual (e.g., head shaking) or refractory behavior when a horse is being ridden because of pain induced by the bit.

HISTORY AND PRESENTING SIGNS

- "Quidding" or spillage of food from the mouth during eating
- Refractory behavior when a bit is in the horse's mouth

CLINICAL FINDINGS AND DIAGNOSIS

- Evidence of pain when eating
- Abrasions on the insides of the cheeks and lateral aspects of the tongue
- Sharp points detected by visual and manual examination of the teeth

DIFFERENTIAL DIAGNOSIS

- Malocclusion
- Supernumerary teeth
- Impacted cheek teeth
- Gingivitis
- Periodontal disease
- Stomatitis

TREATMENT

- Rasping the outside of the upper arcade and inside of the lower arcade as required.
- Removal of the hook (if present) on the rostral edge of the first upper cheek tooth and last lower cheek tooth.
- Topical treatment of oral ulcers/abrasions if required.
- Treatment includes mouthwashes and provision of soft, palatable foods.

Excessive/Disproportionate Dental Wear

Abnormal mastication due to malformations of the mandible or maxilla or the absence of teeth can result in a variety of dental disorders. Common problems include "wave mouth," "step mouth," "shear mouth," and "smooth mouth."

HISTORY AND PRESENTING SIGNS

- Usually horses more than 6 years of age
- Often most severe in older horses
- "Quidding" and evidence of pain when eating
- May show weight loss

CLINICAL FINDINGS AND DIAGNOSIS

- "Wave mouth," from uneven wear of the surfaces of dental arcades, resulting in more pronounced ridges and valleys of the dental surfaces.
- Abnormal wear of incisor teeth is often secondary to stable vices (e.g., "cribbing") or chronic ingestion of sand or soil.
- "Step mouth" is due to long premolars/molars and may involve individual or multiple teeth. Usually this is the result of absence or damage to teeth on the opposing arcade.
- Sharp edges are normally found on the lateral aspect of upper arcade and medial aspect of lower arcade (see above).
- "Shear mouth" is due to the upper arcade being markedly wider than the lower arcade.
- Abrasions on the inside of the cheeks and lateral aspects of the tongue are found with sharp edges.
- Horses may have a worsening of normal wear patterns or unusual wear patterns if the degree of malocclusion is complicated by "parrot mouth."
- In old age, the teeth may be worn down to near the gum level. This is referred to as "smooth mouth." This problem can be exacerbated in horses eating off sandy soils.

DIFFERENTIAL DIAGNOSIS

- Fractured/damaged teeth
- Periodontal disease
- Dental caries/calculus
- Supernumerary teeth
- Impacted teeth
- Stomatitis

TREATMENT

- Vigorous rasping of the teeth should be done to remove sharp points and edges in an attempt to correct malocclusion.
- Correction of the underlying problem may be required, such as prevention of "cribbing" and strategies to reduce ingestion of sand by feeding out of a manger or on large rubber mats.
- Excessively long permanent teeth may require surgical cutting/grinding.
- Frequent dental attention (every few months) usually is required in horses with serious signs of abnormal wear patterns.

Periodontal Disease

Periodontal disease results from abnormal occlusion of teeth leading to gingivitis, which allows

the accumulation of feed in the gingival sulcus. This in turn leads to erosion of the sulcus, alveolar sepsis, and finally tooth loss.

HISTORY AND PRESENTING SIGNS

- Occurs in horses of all ages; however, those over 5 years of age are most commonly affected.
- Halitosis
- "Quidding"
- Weight loss
- Nasal discharge, sinusitis, and colic are less common features.

CLINICAL FINDINGS AND DIAGNOSIS

- Oral examination is the key to diagnosis. This is best done using a mouth gag.
- Gingivitis, alveolar sepsis, and erosion can be evident. In severe cases, tooth loss is possible.
- Radiographs may help confirm secondary changes.

DIFFERENTIAL DIAGNOSIS

- Diseases causing abnormal occlusion or shearing forces
- Fractured teeth
- Dental caries/pulpitis
- Stomatitis
- Gingivitis
- Primary or secondary paranasal sinusitis

TREATMENT

- Routine dental care should be undertaken to ensure appropriate occlusion and to decrease the potential for periodontal disease.
- Mouth washing can be used to assist in flushing necrotic material from pockets of infection.
- If the teeth are severely affected, those involved should be removed.
- For horses with inappetence and weight loss, high-energy palatable feeds should be provided (see Chapter 17).
- Anti-inflammatory drug therapy may be useful to reduce oral sensitivity. Phenylbutazone (Treatment No. 89) given at dose rates of 2.2 mg/kg orally every 12 hours is useful.
- Horses with severe problems and oral inflammation may show a favorable response to antibiotic therapy. Procaine penicillin G (15,000 IU/kg or 15 mg/kg IM q12h; Treatment No. 84) or trimethoprim-sulfonamide combinations (15-30 mg/kg of combined agent PO q12h; Treatment No. 108) usually are active against many of the bacteria involved in periodontal disease.

Supernumerary Teeth

The reason for the occurrence of this disorder is not known. However, it is thought to be the result of division of the permanent tooth germ.

HISTORY AND PRESENTING SIGNS

- Usually occurs in horses more than 3 years old because of the requirement for permanent teeth to be present.
- There is often no relevant history, although evidence of pain when eating, "quidding," and changes in behavior are reported.

CLINICAL FINDINGS AND DIAGNOSIS

- Supernumerary teeth usually are found on routine dental examination.
- Incisors are most commonly affected. The first premolar (wolf) and other cheek teeth are affected in some animals.
- There may be evidence of local injury in association with the offending tooth.

DIFFERENTIAL DIAGNOSIS

- Malocclusion
- Impacted cheek teeth
- Gingivitis
- Periodontal disease
- Dental caries
- Stomatitis

TREATMENT

- No treatment is necessary if only an incisor is involved and there is no interference with mastication.
- In cases where supernumerary teeth are elongated or wear abnormally, cutting, grinding, or removal of the offending teeth may be required.
- Supernumerary cheek teeth should be removed because of their potential to induce impaction, malocclusion, and further dental problems.

Diseases of the Esophagus

Esophageal Obstruction ("Choke")

There are a variety of esophageal diseases resulting from obstructive, traumatic, inflammatory, neoplastic, and congenital disorders, all of which may result in partial or complete obstruction. Obstruction can be due to intraluminal causes ("choke"), strictures of the esophageal wall,

esophageal compression due to extraluminal causes, and diverticulae of the esophageal wall. Dysphagia is the most frequently observed clinical manifestation of esophageal diseases.

HISTORY AND PRESENTING SIGNS

- Inappetence/anorexia
- Pain and retching when attempting to swallow
- Extension of the neck, particularly when attempting to swallow
- Increased salivation (ptyalism)
- Halitosis
- Nasal discharge
- Cervical swelling

CLINICAL FINDINGS AND DIAGNOSIS

KEY POINT
Dysphagia is the most common physical finding in horses with esophageal obstructions.

- When horses attempt to eat, food may discharge from the nares.
- Swelling in the neck may be apparent. Warm painful swelling often is indicative of a cervical abscess or cellulitis. Such lesions may cause a primary extraluminal esophageal obstruction. Perforation of the esophagus may result in a local cellulitis, crepitus, and possibly fistula formation.
- Acute onset of dysphagia and excessive salivation is often the result of acute feed or foreign-body impactions, whereas an insidious onset of dysphagia may be the result of progressive esophageal stricture, neoplasia, or an extraluminal mass.
- Secondary aspiration pneumonia may occur with associated signs present (e.g., dyspnea, increased lung sounds, foul breath).

KEY POINT
Diagnosis of "choke" is confirmed by passage of a nasogastric tube. However, care must be exercised to avoid rupture of the esophagus, because tissue devitalization may have occurred.

- Plain radiographs of the throat and cervical region can assist in identifying the problem. Lateral radiographs are most useful for diagnosis. Accumulated air and feed material may be imaged. Esophageal rupture results in accumulation of periesophageal emphysema.

KEY POINT
Contrast radiography may be necessary to confirm the diagnosis.

- Barium sulfate (50-200 mL) administered orally or via a nasogastric tube can be used to outline intra- and extraluminal obstructions, dilatations, strictures, inflammatory disorders, or diverticulae of the esophagus.
- Esophagoscopy using an endoscope suitable for rhinolaryngoscopy is useful to visualize lesions/obstructions and provides a mechanism by which biopsy samples of suspicious lesions can be obtained. In some cases, foreign bodies can be retrieved with the aid of an endoscope.
- Auscultation of the thorax, transtracheal aspiration or bronchoalveolar lavage, thoracic ultrasound and radiographs, and hematology may assist in the diagnosis of secondary aspiration pneumonia. Mixed bacterial infections are common in cases where aspiration has occurred, with anaerobic organisms frequently implicated.

DIFFERENTIAL DIAGNOSIS

KEY POINT
Acquired intraluminal esophageal obstruction due to impaction of feed material is the most common cause of "choke. "

- External esophageal compression due to retropharyngeal or cervical lymphadenopathy (e.g., "strangles") is the second most common cause of "choke."
- Esophageal stricture, inflammatory disease, or ulceration
- Congenital esophageal disorders (e.g., megaesophagus)
- Dysphagia due to other causes (e.g., cranial nerve deficits, nigropallidal encephalomalacia, rabies)
- Oral foreign bodies
- Stomatitis
- Dental problems
- Gastroduodenal ulceration

TREATMENT

- Acquired intraluminal esophageal obstructions often resolve spontaneously. Those requiring treatment may respond to administration of sedatives and muscle relaxants, such as xylazine (0.5-1.0 mg/kg IV; Treatment No. 109), detomidine (10-20 μg/kg IV; Treatment No. 28), romifidine (0.08-0.12 mg/kg IV), or acepromazine (0.04 mg/kg IV; Treatment No. 1).

- Deposition of a small amount of local anesthetic (20-50 mL lidocaine; Treatment No. 67) onto the impacted mass via a stomach tube can promote esophageal relaxation and decrease discomfort for the horse.
- Lavage of the impaction using warm water administered with a dosing syringe via nasogastric tube can be helpful.
- Administration of oxytocin (0.11-0.22 IU/kg IV, once) may assist in relaxing the proximal esophagus.

KEY POINT

During lavage, ensure that the horse's head is lowered to allow passive drainage of fluid from the nose and prevent aspiration.

- Prior sedation assists in maintaining the horse's head in this lowered position. This lavage process may need to be repeated with intermittent periods of rest to achieve success. External massage of the esophageal mass may be of value. The affected horse should be prevented from having access to feed and bedding during this time. Cuffed nasogastric or small-gauge endotracheal tubes also may be used for delivery of the lavage solution. Inflation of the cuff helps ensure that effluent fluid exits via the tubal lumen.
- In refractory cases, conservative therapy should be continued with the horse under general anesthesia. An endotracheal tube should be inserted, the cuff inflated, and the head placed in a lowered position to reduce the potential for aspiration. In some cases, foreign material can be removed with forceps guided into postion with an endoscope.
- Esophagotomy should be considered a last resort for treatment of esophageal impaction. A number of techniques are described in surgery texts. Healing is usually by second intention, slow, and often complicated. If an esophagotomy is required, referral to a specialized surgical facility should be considered.
- Some cases of esophageal obstruction require fluid therapy support. Isotonic polyionic fluids (see Chapter 18) with added potassium (20-30 mmol/L [mEq/L]) given intravenously are the fluids of choice. When the potassium concentration of intravenous fluids is increased, it is important to ensure that the fluids are administered at rates not in excess of 2 to 3 L/h.
- Judicious use of NSAIDs such as phenylbutazone (Treatment No. 89) or flunixin meglumine (Treatment No. 52) may be of value to decrease esophageal inflammation and local pain.
- After mild obstructions, few food restrictions need apply. Horses with moderate to severe obstructions with subsequent mucosal damage benefit from feed being withheld for up to 3 days to allow mucosal healing to begin. Feed with high moisture content (e.g., fresh-cut green grass) or slurries (e.g., alfalfa gruels or bran mashes) are then slowly introduced to the diet. The progress of mucosal healing often can be assessed using repeat esophagoscopy. Access to grass or feeding off the ground promotes passive drainage of material in the trachea. Clinical signs of aspiration pneumonia are uncommon with uncomplicated cases of "choke."
- Management of aspiration pneumonia should be based on the bacterial organisms involved. A transtracheal aspiration to obtain samples for bacteriology is indicated to define the cause, and antimicrobial therapy is based on these results. Procaine penicillin (15,000-22,000 IU/kg [15-22 mg/kg] IM ql2h; Treatment No. 84), gentamicin sulfate (2-3 mg/kg IV or IM q8-12h or 6.6 mg/kg q24h; Treatment No. 56) and metronidazole (15 mg/kg PO q6h; Treatment No. 75) are good choices for broad-spectrum coverage before sensitivity results are available. Metronidazole is added to the regimen because anaerobic bacteria that are resistant to penicillin frequently are involved in aspiration pneumonia.
- Horses with esophageal perforation or requiring an esophagotomy are treated by allowing the wound to heal by second intention. Broad-spectrum antimicrobial coverage as detailed above and drainage of the local area (in cases of perforation) are indicated. Fluid and nutritional support is provided initially by an esophagostomy tube and then as healing occurs via a nasogastric tube.
- Medium- to long-term complications of esophageal obstruction include mucosal ulceration, diverticulae, and strictures.
- Extraluminal esophageal obstructions due to lymph node abscessation (i.e., secondary to infection with *Streptococcus equi* var. *equi*, "strangles") require long-term antimicrobial therapy. Procaine penicillin G (15,000-22,000 IU/kg [15-22 mg/kg] IM ql2h [Treatment No. 84] for 14 to 28 days), followed by trimethoprim-sulfonamide combination (15-30 mg/kg of the combination PO ql2h; Treatment No. 108) for another 14 to 28 days is indicated. Administration of NSAIDs is also indicated to help reduce pain and local swelling. Horses with respiratory embarrassment due to reduction in tracheal diameter may require a tracheostomy to reduce dyspnea. Drainage or aspiration of the

offending mass is possible under some circumstances but often is difficult.

Diseases of the Stomach

Gastric Impaction, Dilatation, and Rupture

Impaction of the stomach is uncommon and most commonly results from poor dentition or consumption of low-quality foodstuffs (e.g., straw). Dilatation occurs as a primary event as a result of ingestion of excessive amounts of food (e.g., grain), water, or air. The most common cause of secondary dilatation is bowel obstruction (physical or functional), particularly diseases involving the small intestine, although gastric dilatation can occur with obstructions of the large bowel. Because horses are unable to vomit, rupture of the stomach is a frequent sequel to dilatation. Gastric rupture is avoided by rapid recognition of dilatation so that repeated decompression can be used.

HISTORY AND PRESENTING SIGNS

- Evidence of dental abnormalities (e.g., "quidding")
- Rapid eating, a common characteristic of horses low in the social order
- Consumption of poor-quality feed or large volumes of feed or water, especially grain
- Vices (e.g., crib biting or "windsucking")
- Colic

CLINICAL FINDINGS AND DIAGNOSIS

- Abdominal pain ranging from mild with impactions to severe with dilatation. Some horses "dog sit" to decrease pressure on the stomach.
- Increased cardinal signs, dehydration (injected mucous membranes), and shock are common in severe cases.
- Electrolyte deficits (e.g., hypokalemia, hypochloremia) and metabolic alkalosis may occur due to pooling of gastric fluids.
- Significant gastric reflux (5-15 L) is common.

KEY POINT

A nasogastric tube must always be passed.

- It is important to use a nasogastric tube with a number of fenestrations on the side and to repeatedly backflush the tube with water with a large syringe, pump, or hose (500-1000 mL) to promote optimal reflux; otherwise, the tube is likely to block with feed material (see Figs. 7-10 to 7-13). Repeated decompression is necessary. The tube can be removed and replaced or left in situ, being taped to the halter or sutured to the dorsolateral part of the nostril.
- Rectal examination may reveal distended loops of small or large bowel in cases of secondary gastric dilatation.
- Gastric rupture often is accompanied by an immediate reduction in signs of pain, followed by a progressive deterioration of clinical signs (shock) as fulminant peritonitis progresses. In these cases, abdominocentesis will reveal evidence of ingesta and biochemical and cytologic changes consistent with peritonitis.
- Euthanasia is the only option if there is gastric rupture.

DIFFERENTIAL DIAGNOSIS

- Consumption of poor-quality feed
- Ingestion of large volumes of grain, other feeds, water, or air
- Anterior enteritis
- Bowel obstructions
- Other causes of colic (e.g., peritonitis, enteritis/colitis)

TREATMENT

KEY POINT

Gastric decompression is imperative (see above). Lavage of the stomach with warm water also may assist in removal of grain and ingesta.

- Correction of the inciting cause is the key to therapy, and this may require referral for surgery if indicated.
- Supportive therapy. Intravenous fluids are required in dehydrated horses. Isotonic polyionic fluids are usually indicated (see Chapter 18).
- Pain control may be necessary. Choices of analgesics include xylazine (0.3-0.7 mg/kg IV; Treatment No. 109), detomidine (10-20 μg/kg IV; Treatment No. 28), or romifidine (0.08-0.12 mg/kg IV) and NSAIDs such as flunixin meglumine (0.25 mg/kg IV q6h; Treatment No. 52).

Pyloric Stenosis

Pyloric stenosis is a rare acquired disorder in horses occurring secondary to gastroduodenal ulcer disease or as a primary entity because of fibrous masses in the pylorus.

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HISTORY AND PRESENTING SIGNS

- Most common in horses less than 1 year old
- Possible previous history of ulcer disease
- Signs similar to those described for gastric dilatation
- Weight loss, colic, and possibly diarrhea

CLINICAL FINDINGS AND DIAGNOSIS

- Signs of colic usually occur after eating.
- Bruxism (teeth grinding) and increased salivation are common.
- Poor body condition.
- Positive gastric reflux may be found. Decompression often relieves colic signs.
- Nonspecific laboratory findings such as mild inflammatory changes in abdominal fluid may be identified.
- Diagnosis is made from the combination of recurrent clinical signs together with endoscopy, radiography or, ultimately, necropsy.

TREATMENT

- Medical supportive therapy (e.g., fluids and electrolytes, pain control, and possibly antiulcer therapy; see Gastroduodenal Ulcer Disease) is indicated in the acute phase of the disorder.
- Surgical management of the stenosis is reported and may be possible in a referral surgical facility. Surgical correction may be done by performing a gastrojejunostomy.

Gastric Neoplasia

Gastric neoplasia is rare, with squamous cell carcinoma being the most common tumor in the stomach.

HISTORY AND PRESENTING SIGNS

- Occurs in older horses
- Weight loss, anorexia, chronic diarrhea in some cases
- Recurrent low-grade colic related to eating
- Increased salivation or dysphagia in some cases

CLINICAL FINDINGS AND DIAGNOSIS

- Signs are frequently nonspecific with weight loss, colic, and dysphagia being found most commonly. In some cases, volumes of fluid (4-8 L) given by nasogastric tube, which would normally be well tolerated, result in signs of abdominal pain.
- In chronic cases, there may be mild to severe anemia, and fecal examination may show presence of occult blood.
- Abdominocentesis may reveal increased numbers of nucleated cells and protein concentration.
- Diagnosis can be assisted by radiographs or gastric endoscopy, although both techniques require relatively sophisticated equipment and in most instances are not practical except in a referral hospital. Most commonly, diagnosis is made at necropsy.

TREATMENT

- There is no worthwhile treatment.

Gastroduodenal Ulcer Disease

Gastroduodenal ulceration in foals is discussed in Chapter 9. This section discusses ulcer disease in adult horses. Gastroduodenal ulceration has been recognized more commonly in adult horses in recent years, particularly since the advent of long, flexible fiberoptic endoscopes suitable for insertion into the stomach. As with ulcer disease in foals, stress appears to play an important role in the genesis of this disorder. As a result, the incidence of ulcers is reported to be much higher in horses undergoing training than in those living more sedentary lives, although this may reflect the differences in feeding/grazing management. Ulcers occur with greatest frequency on the squamous gastric mucosa.

NSAIDs promote gastric ulceration by inhibiting local mucoprotective mechanisms. Toxicity with these agents also results in small and large colonic mucosal (and renal) lesions.

HISTORY AND PRESENTING SIGNS

- Horses confined and being "hand" fed
- In many cases ulcers are "silent," producing no overt signs of disease
- Poor athletic performance, recurrent colic, weight loss, inappetence, and diarrhea
- Perforation, although rare, results in rapid deterioration due to fulminant peritonitis
- Prior history of nonsteroidal anti-inflammatory medication

CLINICAL FINDINGS AND DIAGNOSIS

- The signs may be nonspecific, mild recurrent colic or capricious appetite being most common.
- Some horses show signs of depression, and there may be weight loss and a rough hair coat.

- Pain may occur after eating, although this sign is not found in every case.
- In younger horses (6 months to 2 years of age), bruxism (teeth grinding) and increased salivation may be indicators of disease.
- Moderate gastric reflux may be found.
- Abdominal fluid changes are mild and nonspecific but may indicate mild inflammation.
- Discolored "coffee grounds" appearance of gastric reflux occurs in some cases, which is due to hemorrhage from the ulcers.
- Tentative diagnosis is often based on suspicion, clinical findings, and response to treatment. Diagnosis is confirmed antemortem by gastroscopy. However, equipment to perform this procedure is currently only available in veterinary teaching hospitals and referral practices. Contrast radiography may be of value, but results are difficult to interpret. It is worthwhile noting that gastroduodenal ulcers are commonly identified as an incidental finding at necropsy.

TREATMENT

- Decrease any factors producing stress and eliminate NSAIDs, if possible. This is vital to optimize the effects of antiulcer medications.

KEY POINT

Use of histamine (H2) receptor antagonists such as cimetidine (Treatment No. 26) (20 mg/kg PO q8h or IV q6h) or ranitidine (Treatment No. 100; 6.6 mg/kg PO q8h or 2 mg/kg IV q6-8h) for at least 2 to 3 weeks is the principal method of medical therapy. Ranitidine seems to give superior results to cimetidine.

- New drugs designed to block the proton pump and therefore inhibit acid secretion (e.g., omeprazole 1 mg/kg q24h) are effective in horses and will become more widely used in the future when registered for use in this species.
- Less expensive are the antacids composed of aluminum/magnesium suspensions (Milanta II, Stewart Pharmaceuticals, Wilmington, DE, or Maalox TC) which also have been prescribed (250 mL/500 kg PO q8h) and may assist in ulcer therapy or to reduce the rate of recurrence.
- The mucoprotectant sucralfate (Treatment No. 102; 2-4 g/500 kg PO q6-12h) adheres to damaged gastric mucosa and stimulates local blood flow and mucous secretion. There is divided opinion as to the efficacy of this preparation. However, it is considered to be beneficial in horses with gastrointestinal signs from NSAID toxicity.
- If gastric emptying is delayed, use of bethanechol (Urecholine, Merck & Co., Inc., Rahway, NJ; 0.25 mg/kg SC q4h for several doses) is of benefit in some cases.
- Some horses may need to be managed by allowing them to graze all day or by being given constant access to hay because fasting or intermittent feeding of high-concentrate diets predisposes to gastric ulceration.

Gastric Parasitism

Infection with *Gastrophilus* spp. ("bots") is the most common cause of gastritis in adult horses. Infection with *Habronema* spp., *Draschia megastoma*, and *Trichostrongylus axei* also occurs. Although infection with any or all of these parasites is common, clinical signs are rare.

HISTORY AND PRESENTING SIGNS

KEY POINT

Clinical effects are rare, although ill thrift, colic, and gastric dilatation/rupture have been ascribed to these parasites, particularly "bots."

- Bot eggs seen on horses legs or bot maggots in feces

CLINICAL FINDINGS AND DIAGNOSIS

- Generally no signs are shown. In some cases, nonspecific signs (e.g., poor body condition) or signs of recurrent low-grade colic may be noted.

TREATMENT

- All gastric parasites are effectively treated with ivermectin at a dose rate of 0.2 mg/kg orally (Treatment No. 62).

Diseases of the Small Intestine

Anterior Enteritis (Duodenitis-Proximal Jejunitis, Proximal Enteritis)

Anterior enteritis is the result of a hemorrhagic/necrotic lesion in the duodenum and proximal jejunum. This significant inflammatory lesion allows for transudation and secretion of large volumes of electrolyte-rich fluid into the intestinal lumen. Severe pain, gastrointestinal reflux, dehydration, and electrolyte abnormalities are common

features of the disorder. The cause of this disease is not known, although lesions are similar to a disease occurring in humans due to infection with *Clostridium perfringens.*

HISTORY AND PRESENTING SIGNS

- Adult horses (>3 years old) are most frequently affected.
- Possible recent history of dietary change, particularly an increase in the energy content of the diet.
- Sudden onset of acute severe colic and signs of depression.

CLINICAL FINDINGS AND DIAGNOSIS

- Clinical signs of the disease are related to the severity of the small intestinal lesions.

KEY POINT

Severe pain is evident initially with large-volume, fetid, sanguineous nasogastric reflux. Pain and tachycardia often temporarily abate after gastric decompression. Signs of depression are often profound.

- Fever, injected mucous membranes, tachycardia.
- Ileus.
- Small intestinal distension due to intestinal inflammation may be detectable per rectum.
- Dehydration (often severe), hyponatremia, hypochloremia, hypokalemia, hypocalcemia, and mild metabolic acidosis together with prerenal azotemia and shock are classical features.
- Hematology commonly reveals the presence of neutrophilia.
- Mild peritonitis (total nucleated cell count 5000-10,000 x 10^6/L [5000-10,000/(ML]) with an elevated protein concentration of 30 to 45 g/L (3-4.5 g/dL) is common.

KEY POINT

Diagnosis of anterior enteritis is particularly difficult because the clinical signs are similar to those occurring with other forms of small intestinal obstructive disease. Unlike strangulating obstructions of the small intestine, horses with anterior enteritis frequently have a significant reduction in pain after gastric decompression (strangulating obstructions do not) but still show signs of profound depression.

- Response to repeated gastric reflux and medical support can be useful in assisting the clinician in making a tentative diagnosis.
- Definitive diagnosis can be made at surgery, although there is questionable benefit to affected animals if subjected to surgical intervention, and the prognosis may be worsened. However, given the similarity of signs to those occurring with obstructive small bowel diseases, many horses with anterior enteritis may undergo exploratory surgery.

DIFFERENTIAL DIAGNOSIS

- Small intestinal obstruction/strangulation
- Undifferentiated enteritis/colitis
- Large intestinal diseases/obstructions
- Acute peritonitis
- Acute liver failure, cholelithiasis
- Other causes of abdominal pain (e.g., nephrolithiasis)

TREATMENT

- Rapid institution of aggressive supportive therapy is the key to a successful outcome. Therapy is often time consuming and expensive because many cases require treatment for more than 3 to 4 days and some as long as 8 to 10 days until damage to the intestine is sufficiently repaired to prevent transudation of fluid into the lumen.

KEY POINT

Repeated gastric reflux (every 1-2 hours) or placement of an indwelling nasogastric tube is vital to assist in the control of pain and to reduce the risk of gastric rupture.

- In many cases, the nasogastric tube should be left indwelling, being sutured into the nostril or taped to the halter. Volumes of 6 to 10 L commonly can be obtained during initial gastric decompression.

KEY POINT

Massive intravenous fluid administration is indicated using polyionic sodium-containing fluids (see Fluid Therapy, Chapter 18) at initial rates of 10 to 15 L/h for a 500-kg horse, later reduced to 2 to 5 L/h after hydration is restored.

- In the maintenance phase of treatment, addition of potassium chloride to the fluids at a rate of 15 to 20 mmol/L (mEq/L) is indicated to assist in partially replacing deficits. Note: Reflux will often become more voluminous after induction of fluid therapy. Calcium supplementation also

may be indicated. Appropriate clinical response is reflected by improved demeanor, decreased heart rate, increased pulse pressure, and a return of serum electrolyte, urea nitrogen, creatinine, and acid-base values toward normal.

- Low-dose NSAID therapy such as flunixin meglumine (0.25 mg/kg IV q6-8h; Treatment No. 52) is useful to reduce pain, ameliorate the effects of endotoxemia, and as a prophylactic measure for laminitis, which is a common sequel to the enteritis.
- Intravenous administration of DMSO (0.5-1.0 g/kg IV slowly as a 10-20% solution; Treatment No. 34) has been suggested to assist in reducing the inflammatory bowel lesion and decrease complications such as thromboembolism and laminitis. Recently, some clinicians have used nitroglycerin patches applied to the pasterns over the digital vessels to encourage vasodilation and improved blood flow to the laminae as a prophylactic measure against laminitis. However, there have been no controlled studies to show the efficacy of this treatment.
- Infusions of lidocaine hydrochloride (1.3 mg/kg as a slow IV bolus followed by 0.05 mg/kg/min) may improve intestinal motility in refractory cases.
- Antimicrobial therapy with procaine penicillin (15,000-22,000 IU/kg [15-22 mg/kg] IM q12h; Treatment No. 84) has been prescribed on the basis that the disease may be the result of infection with *Clostridium* spp.
- With aggressive medical therapy, greater than 80% of horses with anterior enteritis recover.

Small Intestinal Obstruction

Obstruction of the small intestine is categorized either as simple, where there is intraluminal blockage without infarction, or strangulating, where the blood supply to the intestine is compromised. Simple small intestinal obstructions commonly cause severe pain due to accumulation of fluid proximal to the lesion and the discomfort of the obstructive lesion itself. In the case of strangulating lesions, the signs of discomfort frequently are magnified, because compromise of circulation to the bowel and liberation of toxins increases the sensitivity of pain receptors in the affected region. After complete obstruction, there is a cascade of events, including pain, toxemia, dehydration, cardiovascular compromise, and shock that is often irreversible if untreated.

KEY POINT

As a general rule, simple obstructions have a slower progression of signs and may be amenable to medical therapy, whereas strangulating obstructions are likely to be fatal without surgical correction.

CAUSES OF SMALL INTESTINAL OBSTRUCTION

1. ***Simple***
   - *a. Intestinal stricture*
   - *b. Adhesions due to previous surgery or peritonitis*
   - *c. Impaction (commonly at the ileum)*
   - *d. Thickened/dysfunctional ileocecal valve*
   - *e. Intussusception*
   - *f. Foreign bodies*
   - *g. Impaction with ascarids*
   - *h. Muscular hypertrophy of the ileum*
2. ***Strangulating***
   - *a. Volvulus*
   - *b. Herniation through the epiploic foramen*
   - *c. Mesenteric herniation*
   - *d. Strangulating umbilical hernia*
   - *e. Inguinal hernia*
   - *f Pedunculated lipoma*

HISTORY AND PRESENTING SIGNS

- Inappetence/anorexia
- Pain varying from mild with signs of depression to severe and uncontrollable, depending on the lesion
- Sweating, rolling, pawing

CLINICAL FINDINGS AND DIAGNOSIS

- Simple obstructions tend to cause the least severe signs and progress more slowly than strangulating lesions. Some simple obstructions correct spontaneously. However, if there is complete obstruction and no treatment, affected horses will demonstrate a progressive increase in severity of pain and/or cardiovascular compromise.
- Physical examination may reveal abnormalities such as distended loops of bowel in umbilical or inguinal hernias.
- Elevation in heart rate is usual (>50 beats/min), although some horses with substantial areas of infarcted bowel have normal heart rates.
- Obstructions commonly result in decreased intestinal sounds. In contrast, an increase in gut sounds may be heard early in cases of enteritis.
- Rectal temperature is usually normal or slightly elevated if the horse has been overtly active in its response to pain. In contrast, horses suffering from acute colitis and anterior enteritis that may show similar signs initially but then are affected

by severe diarrhea are more likely to have elevations in rectal temperature.
- Evidence of dehydration and cardiovascular compromise usually are found. There is decreased skin elasticity, diminished pulse pressure, increased capillary refill time, dry gums, and increased hematocrit and total plasma protein (see Table 7-3 for a summary of likely alterations occurring in PCV and TPP and their severity). If the disease involves bowel compromise, there usually is progressive endotoxemia and fluid derangements resulting in shock. This is reflected by injected mucous membranes, cold extremities, progressive increases in heart rate, weak pulse pressure, and signs of central nervous system depression.
- Nasogastric reflux is usually positive (>3—5 L).
- Rectal examination may reveal distended loops of small intestine.

KEY POINT

Failure to detect abnormalities per rectum or retrieve nasogastric reflux does not preclude the possibility of small intestinal obstruction.

- The technique for performing a thorough rectal examination is outlined in Figures 7-1 to 7-9. Horses with anterior enteritis have distended loops of bowel; however, these are not likely to be as tightly distended as those occurring with small intestinal obstruction. In horses afflicted with enteritis, distension temporarily abates subsequent to nasogastric reflux.
- Abdominocentesis is useful in assisting in decision-making processes (see Table 7-1). As a general rule, there will be progressive increases in the total nucleated cell count, protein content, and serosanguineous appearance of the fluid as bowel compromise becomes more severe.

DIFFERENTIAL DIAGNOSIS

- Small intestinal simple or strangulating obstruction
- Anterior enteritis
- Peracute enteritis/colitis (i.e., diarrhea)
- Large bowel obstruction, displacement, or strangulation
- Peritonitis
- Other causes of colic (e.g., urolithiasis, nephrolithiasis, liver disease/cholelithiasis)
- Laminitis
- Pleural effusion

TREATMENT

- Mild or partial simple obstructions may respond to medical therapy, including nasogastric decompression, restoration of fluid balance and pain control (see earlier section). This type of lesion is rare.
- Complete obstructions (simple and strangulating) require surgical intervention or euthanasia if surgery is not an option. Affected horses should be referred or subjected to exploratory surgery as soon as possible to avoid prolonging the time that bowel is compromised. Such compromise increases the potential for detrimental effects of endotoxemia, shock, and bowel rupture to occur. Medical stabilization of the patient with fluids and analgesics before surgery or transport for surgery may be indicated.

Diseases of the Large Intestines

LARGE INTESTINAL OBSTRUCTION

Diseases of the large colon constitute one of the most common causes of abdominal pain, and it is fortunate for both the affected horse and the veterinarian that many of these disorders are responsive to medical management. The large colon is a relatively mobile organ that, on a volume basis, constitutes the major proportion of the gastrointestinal tract of the horse. Given its size, mobility, and role in fermentation and water absorption, disorders of the large colon can result in significant pain, alterations in fluid balance, and gas accumulation. Definitions are similar to those applied to the small intestine; that is, obstructive lesions are referred to as simple or nonstrangulating obstructions, caused by intraluminal blockages (e.g., feed or sand impactions, foreign bodies/fecoliths/enteroliths) and displacements (e.g., left or right dorsal displacement). Strangulating obstructions involve vascular compromise and are commonly due to volvulus of the ascending colon.

Feed Impaction

Impaction of feed in the large colon constitutes the most common form of colonic obstruction and is therefore a frequent cause of colic. It is not well established what causes the impaction. Factors thought to contribute include motor dysfunction that disrupts bowel motility, stress, diet (e.g., poor-quality roughage) or dietary change, poor dentition, parasites, and possibly decreased water intake.

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HISTORY AND PRESENTING SIGNS

- Inadequate parasite management
- Feed spilling from mouth during eating ("quidding")
- Poor-quality feed
- Recent change in environment/weather conditions (e.g., onset of cold weather)
- Mild to moderate abdominal pain
- Inappetence

CLINICAL FINDINGS AND DIAGNOSIS

- Mild to moderate abdominal pain is usual. This progresses to severe pain if left untreated. However, this progression may occur over several days, unlike the more rapid succession of events with small bowel obstruction.
- The heart rate may be low initially. There may be a progressive increase in heart rate (50-65 beats/min) if the obstruction is not resolved.
- The degree of dehydration is variable.
- There is moderate to severe decrease in gastrointestinal sounds, which progressively worsens over 1 to 2 days.
- Abdominal distention may be noted in association with gas accumulation.
- Abdominal fluid may be normal, although with impactions of longer duration there may be increases in total nucleated cell count and a mild increase in protein concentration.
- Rectal examination may reveal a firm mass in the pelvic flexure or the right dorsal colon, although the anatomic location of the impaction (anterior abdomen) may preclude its detection. It is common to find a gas-filled large colon.
- Hematology and plasma biochemistry show few changes, apart from an increase in total protein. Acid-base status usually is normal.

DIFFERENTIAL DIAGNOSIS

- Other causes of large colon impaction (e.g., enteroliths, sand)
- Large colon displacement or volvulus
- Small intestinal obstruction or strangulation
- Acute colitis
- Liver disease/cholelithiasis
- Peritonitis
- Pleuropneumonia
- Rhabdomyolysis

TREATMENT

- Analgesics to control pain (see Medical Management of Colic).

KEY POINT

Fluid therapy is essential to correct dehydration and to soften the impaction.

- If gastric reflux is not present and there is evidence of gut sounds being present, fluids can be given by nasogastric tube (see Medical Management of Colic). Intravenous fluid therapy is also valuable. Isotonic polyionic fluids (65-100 mL/kg/day IV) should be considered (see Fluid Therapy, Chapter 18).
- Because foreign-body impactions are difficult to distinguish from food impactions, laxatives, such as mineral oil (Treatment No. 77) and dioctyl sodium sulfosuccinate (DSS) (Treatment No. 35), often are used (see Medical Management of Colic). However, frequent administration of fluids per nasogastric tube (e.g., water) are likely to be equally effective.
- Medical therapy can be continued for up to 5 to 7 days if pain is limited or easily controlled, the horse voluntarily consumes fluids, there is no deterioration in the animal's metabolic status, and there is no significant increase in abdominal fluid total nucleated cell count or protein concentration. Deterioration in any of these signs constitutes an indication for referral for surgery.

Foreign-Body Obstruction

This is a problem most common in young horses because of their less discriminating eating habits. Ingestion of rope, baling twine, straw bedding, shavings, plastic, and feed bags are common causes. These materials combine with ingesta and often result in obstruction of the gastrointestinal tract. Because of the reduction in luminal size at the junction of the right dorsal and transverse colon, obstruction at that site is common. However, obstructions at more proximal sites occur.

HISTORY AND PRESENTING SIGNS

KEY POINT

Moderate abdominal pain initially, progressively becoming more severe.

- Inappetence/anorexia
- Often seen in younger horses (<3 years old)
- Abdominal distension
- Decreased fecal output

CLINICAL FINDINGS AND DIAGNOSIS

- See section on large colon feed impactions, because the signs are often difficult to distinguish from that disorder.

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- Diagnosis is usually based on the history of abdominal pain, physical examination findings (e.g., lesion palpable per rectum), and response to management. Specific diagnosis may not be possible without surgery.

DIFFERENTIAL DIAGNOSIS

- Other causes of large colon impaction (e.g., feed, enteroliths, sand)
- Large colon displacement or volvulus
- Small intestinal obstruction or strangulation
- Acute colitis
- Liver disease/cholelithiasis
- Peritonitis
- Pleuropneumonia

TREATMENT

- Because foreign-body impactions are difficult to distinguish from food impactions, treatment with fluids via nasogastric tube with or without laxatives is used. Mineral oil (Treatment No. 77) and DSS (Treatment No. 35) are good choices. Fluid therapy should be regarded as the first line of treatment (see Chapter 18).
- Expulsion of the foreign body and resolution of signs will occur spontaneously in some cases or in response to medical management. In other cases, there is a progressive deterioration in signs requiring referral for surgical correction or euthanasia of the horse.

Sand Impactions

Horses maintained in sandy locations and that eat from the ground inevitably ingest sand. Alternatively, sand may constitute part of what the horse eats because of its inclusion in hay. Younger animals with indiscriminant eating habits at times voluntarily consume sand. Consumption of sufficient sand results in accumulation in the pelvic flexure and right dorsal and transverse colon. Sand induces a physical colitis. This inflammatory response, associated with accumulation of sufficient volume of sand, can result in colonic rupture.

HISTORY AND PRESENTING SIGNS

- Living in a sandy environment (e.g., Arizona, California, and Florida are common locations for horses with this disorder)
- Eating off the ground in sandy environments
- Pain and presenting signs similar to those described for feed impactions

CLINICAL FINDINGS AND DIAGNOSIS

- Many signs similar to those referred to for obstructions of the large colon.
- Rectal examination may reveal sand impactions in the ventral colon, which in some cases can be massive (>30 kg). Failure to detect sand or an impaction on rectal examination does not preclude the possibility of sand accumulation, because it may be deposited in a cranial portion of the gastrointestinal tract and is therefore out of reach. "Grit" may be detected when palpating or may be found on the rectal sleeve after removal of the veterinarian's arm from the rectum.
- Auscultation of the ventral abdomen is reported to assist in the diagnosis of sand impaction. A gritty or grinding sound may be heard as the sand moves within the large colon.

KEY POINT

Dissolving feces in water in a bucket or rectal sleeve and observing for sand on the bottom of the bucket or sleeve may provide evidence of the likelihood of sand impaction.

- Small amounts of sand may be found in feces and do not necessarily reflect sand impaction. However, large amounts of sand provide strong evidence for impaction. Note: If in doubt, compare the amount of sand in the affected horse's feces with that from other normal horses in the herd/region.
- Abdominal radiography can reveal sand impaction in foals, ponies, and miniature horses. Similarly, ultrasound examination can provide images consistent with accumulations of coarse sandy material in the large colon.

DIFFERENTIAL DIAGNOSIS

- Other causes of large colon impaction (e.g., feed impaction, enteroliths)
- Large colon displacement or volvulus
- Small intestinal obstruction or strangulation
- Acute colitis (diarrhea)
- Liver disease/cholelithiasis
- Peritonitis
- Pleuropneumonia

TREATMENT

- Laxatives to lubricate the gastrointestinal tract and assist in the movement of sand (see Medical Management of Colic). The laxative of choice is psyllium hydrophilic mucilloid (Treatment No. 98, 0.5-1.0 g/kg q6-24h; mixed in 4-8 L

water and pumped rapidly into the stomach via nasogastric tube before the mucilloid turns to gel). This treatment is maintained for several days to a week depending on the severity of the case. After initial therapy, the psyllium can be fed to the horse in sweet feed. Many practitioners also use repeated doses of mineral oil (10 mL/kg q12-24h for three to four doses) to assist in movement of sand from the large colon.

KEY POINT

Feces should be monitored for the rate of expulsion of sand.

- Therapy (e.g., with psyllium) may need to be continued for weeks to ensure that sand is fully removed from the colon.
- Severely affected and unresponsive cases may require referral for attempted surgical correction. Surgery involves a pelvic flexure enterotomy and flushing of sand out of the large colon.
- Prevention of recurrence is important. This requires ensuring that horses eat their feed from a manger or bucket to avoid ingestion of sand. Alternatively, eating food off rubber mats or out of troughs (tires cut in half make cheap troughs) is indicated. Removal of hay containing sand from the horse's diet is important. Intermittent administration (1 g/kg PO q24h) of psyllium mucilloid for several weeks to remove any accumulations of sand also may be indicated. Longer-term administration is likely to result in an increased rate of degradation of the mucilloid by colonic microbes and a decrease in laxative effects. A commonly recommended preventative program is to feed 1 g/kg psyllium daily for the first week of every month. Feeding diets high in fiber (e.g., grass hay) is advisable also.

Enteroliths

Enteroliths are mineral concretions that form in the large colon. They create partial or complete obstructions when passed into the small colon. Enteroliths are more common in horses in certain geographic regions (e.g., California and the southern states). Common anatomic sites for obstruction include the ampulla of the small colon and the transverse and small colons. Diets rich in nitrogen, magnesium, and phosphorus are thought to contribute to the formation of enteroliths, because these chemicals are integral components of mineraloliths. Alfalfa (lucerne) hay has a high nitrogen and possibly magnesium content and may play a role in the development of enteroliths. Alfalfa also may allow intestinal pH to rise, thereby promoting the potential formation of mineraloliths.

HISTORY AND PRESENTING SIGNS

- Adult/older horses
- Alfalfa diet
- Presenting signs are similar to those for feed impactions of the large colon.
- Pain may be intermittent because the enterolith temporarily lodges in the ampulla of the small colon and then discomfort abates when the enterolith passes back into the right dorsal colon.
- If the enterolith causes complete obstruction and vascular compromise, pain may be more intense.

CLINICAL FINDINGS AND DIAGNOSIS

- Abdominal pain occurs. This is low-grade and similar to that occuring in other obstructive diseases of the large colon.
- There are usually only small amounts or an absence of feces. If there is only partial luminal obstruction, limited amounts of feces, gas, and mineral oil can still be passed.
- Progressive abdominal distension occurs in many cases.
- A progressive decrease in gut sounds occurs. In severe cases there is an absence of gut sounds.
- Rectal examination may reveal gaseous distention of the large colon. Enteroliths are palpable in some cases. If rectal palpation of the horse is performed with its forequarters elevated, retrograde movement of the gastrointestinal tract may increase the chance of the mineralolith being felt. In smaller horses, radiographs may aid in the diagnosis.
- Abdominal fluid is usually normal. However, if there is vascular compromise at the site of obstruction, there will be progressive changes in abdominal fluid constituents, including increases in total nucleated cells, protein, red cells, and blood pigments.

DIFFERENTIAL DIAGNOSIS

- Other obstructive diseases of the large colon
- Large colon displacement or volvulus
- Small intestinal obstruction or strangulation
- Acute colitis (diarrhea)
- Peritonitis
- Liver disease/cholelithiasis
- Pleuropneumonia

TREATMENT

- After diagnosis of an enterolith, surgical removal is the therapy of choice. However, at times, enteroliths will not be diagnosed on the initial workup and an exploratory celiotomy is

performed, at which time the mineralolith is identified.
- On farms or in areas with a high incidence of enteroliths, feeding of vinegar (250 mL PO in feed q12h) has been recommended. It is thought the vinegar decreases colonic pH and reduces the potential for formation of mineraloliths.

STRANGULATING OBSTRUCTIONS OF THE LARGE COLON

Colonic Torsion

Severe torsion (270 degrees) of the large colon, causing vascular compromise and tissue devitalization, is a potent stimulus for induction of signs of abdominal pain in the horse. Although less severe torsions do occur, correction almost invariably requires surgical intervention. Average duration of survival after surgery remains low (approximately 30% of horses are alive 12 months postsurgery), but success can be greatly improved if there is a short interval between initiation of signs of colic and performing a laparotomy to correct the torsion. The etiology remains controversial. Brood mares are more commonly affected around the time of parturition.

HISTORY AND PRESENTING SIGNS

- Variable abdominal pain. When severe torsions are present there is often extreme uncontrollable pain.
- Most common in adult horses, particularly brood mares
- Highest incidence in summer
- Possibly related to parturition
- Inappetence/anorexia, signs of depression
- Reduced fecal output

CLINICAL FINDINGS AND DIAGNOSIS

- Variable often severe abdominal pain.
- Elevated heart rates are noted, usually greater than 75 beats per minute.
- Progressive abdominal distension occurs over several hours.
- Dehydration is common, with signs of cardiovascular compromise and shock.
- Rectal examination reveals a distended large colon. The colonic wall may feel thickened (edema).
- Results of abdominal fluid analysis are variable. However, in most horses there are few changes, and those that exist do not reflect the extent of tissue devitalization that has occurred. Where there is a profound increase in total nucleated cells, protein, and blood pigments, tissue ischemia is usually advanced and indicates a poor prognosis.
- Diagnosis may be confirmed on the basis of clinical signs and rectal examination in most cases. In others, diagnosis is not confirmed until an exploratory celiotomy or necropsy is performed.

DIFFERENTIAL DIAGNOSIS

- Other obstructive diseases of the large colon
- Large colon displacement
- Small intestinal obstruction or strangulation
- Acute colitis (diarrhea)
- Peritonitis

TREATMENT

- Initial therapy involves pain control, reconstitution of the circulating volume with intravenous fluid administration, and therapy for endotoxemia (see Medical Management of Colic).
- Surgery is required to correct the torsion, and horses with suspected torsion should be referred to a surgical center as early in the course of the disease as practical.
- Euthanasia constitutes a rational alternative in cases where owners do not wish to have surgery performed.

Colonic Displacement

Displacement of the colon dorsally over the nephrosplenic ligament (left dorsal displacement) and right dorsal displacement, where the colon rotates around the cecum, are the most common forms of colonic displacement. The cause of these displacements is not known, but it is thought to be the result of alterations in gut content (e.g., accumulation of gas or ingesta and alterations in motor activity of the colon).

In *left dorsal displacement*, the left portion of the large colon becomes lodged between the body wall and left kidney, with the nephrosplenic ligament supporting the colon in its displaced position. Two mechanisms are thought to contribute to colon entrapment at this site. One involves the pelvic flexure migrating over the top of the nephrosplenic ligament. The alternative mechanism requires splenic contraction or ventromedial movement, allowing migration of the colon between the spleen and body wall. When the spleen subsequently expands or returns to its normal position, the colon is trapped in this dorsal location.

Once lodged in the nephrosplenic space, the colon becomes occluded, causing a partial obstruction.

In *right dorsal displacement,* the most common form occurs when the large colon rotates (180 degrees) around the mesenteric attachment, resulting in the colon being located between the cecum and body wall on the right. The cause of this rotation is not known.

HISTORY AND PRESENTING SIGNS

- Most common in large horses
- Mild to moderate abdominal pain
- Inappetence, decreased fecal output

CLINICAL FINDINGS AND DIAGNOSIS

- Normal or slightly elevated heart rate. In severe cases the heart rate is often higher.
- Variable degree of pain.
- Variable degrees of abdominal distension are found, depending on the duration and degree of obstruction.
- Dehydration and cardiovascular compromise are mild in early cases. With progression, there is increased gas and fluid accumulation, tissue compromise, and potential for shock.
- Rectal examination. With left dorsal displacement, there is gaseous distension of the large colon and displacement of the spleen away from the body wall and toward the midline of the abdomen. In some cases, the colon lodging in the nephrosplenic ligament is palpable. Horses suffering from right dorsal displacement have a gas-distended colon with the pelvic flexure not palpable because it is relocated in the cranial abdominal region because of the rotation of the colon on the mesenteric stalk.
- Abdominal fluid is often unchanged, except in severe cases where tissue devitalization has occurred, which would be reflected by increases in total nucleated cells and protein and, possibly, blood staining. Inadvertent puncture of the spleen occurs more commonly with left dorsal displacement of the colon because of the ventromedial displacement of the spleen. The PCV of the fluid in this situation will frequently be higher than that of peripheral blood (approximately >0.6 L/L or 60%). Where results of rectal examination are equivocal for a diagnosis of nephrosplenic entrapment, transcutaneous ultrasound examination dorsal in the left caudal abdomen using a 2.5-MHz sector scanner transducer may assist in making a definitive diagnosis. Visualizing the left kidney immediately adjacent to the spleen rules out a left dorsal displacement of the large intestine. In contrast, where the left colon is displaced into the nephrosplenic space, the intraluminal gas causes classical shadows adjacent to the left kidney.

DIFFERENTIAL DIAGNOSIS

- Other obstructive diseases of the large colon
- Large colon displacement
- Small intestinal obstruction or strangulation
- Acute colitis (diarrhea)
- Peritonitis
- Liver disease/cholelithiasis
- Pleuropneumonia

TREATMENT

- Medical management involves therapy to control pain and fluid derangements.
- In some mild cases of left or right dorsal displacement, correction will occur spontaneously if feed is withheld. The reduction in gut fill allows the colon to shrink and possibly relocate in a more normal anatomic location. Recently, phenylephrine has been reported to dramatically decrease the size of the spleen, thus improving the chances of success with conservative therapy such as rolling. Administration of phenylephrine at 3 μg/kg/min for 15 minutes will reduce spleen size to about 25% of original size for about 30 minutes.
- Attempts to roll the horse have been described to correct left dorsal displacement. The horse is anesthetized, placed on its right side, and is then lifted into the air by the legs with a hoist. The horse is then lowered onto its left side and allowed to recover, hopefully with the colon replaced in its normal anatomic location. Corrections may be aided by an assistant directing colonic manipulations rectally. This may be a worthwhile procedure where economic considerations prevent surgery as an option.
- In severe cases of left or right displacement, where conservative therapy fails or if the horse's condition deteriorates, referral for surgical exploration, confirmation of the diagnosis, and correction of the problem is indicated.

Other Gastrointestinal Diseases

Acute Diarrhea (Acute Colitis)

In most cases of acute diarrhea, substantial inflammatory involvement of the large colon and

cecum occurs, with affected horses showing signs of abdominal pain, dehydration, and shock. The diarrhea may be severe, and clinical progression of the disease may be rapid. Given these considerations, the clinician's main efforts should be directed at maintaining fluid and electrolyte balance, treating shock, and preventing untoward sequelae. The etiology of individual cases of colitis frequently remains obscure despite vigorous diagnostic efforts. Even if a diagnosis is made, the cause of the diarrhea is rarely known at the time of onset of clinical signs. A number of causes for acute colitis have been described.

Salmonella spp. are frequently implicated in this syndrome. Salmonellosis most often occurs in horses living in crowded conditions and in association with concurrent disease or stress (e.g., after surgery). Salmonellosis has a much higher prevalence in hospital environments. Diarrhea results from increased fluid secretion due to a toxin released by the organism, inflammation of the bowel mucosa, and malabsorption/maldigestion due to villous destruction.

C. perfringens type A ("colitis X," edematous bowel syndrome, peracute diarrhea syndrome) produces a syndrome that often resembles peracute salmonellosis, although signs are more acute, and horses may die of shock and complications before diarrhea occurs.

Equine monocytic ehrlichiosis ("Potomac horse fever") is caused by *Ehrlichia risticii*, resulting in typhlocolitis. *E. risticii* is likely to be spread by an insect vector, although none has been identified positively.

Other causes of acute diarrhea/colitis include excessive NSA1D therapy, antibiotic administration (particularly high doses of trimethoprim-sulfa combinations and tetracyclines), heavy-metal toxicosis, plant toxicoses, cantharadin (blister beetle; *Epicauta* spp.) toxicosis, peritonitis, and parasites.

HISTORY AND PRESENTING SIGNS

Salmonellosis

- Horses of all ages are affected, but it is most common in younger horses.
- Stress (e.g., training, transport, overcrowding, anesthesia, and/or surgery) and recent dietary change predispose horses to salmonellosis.
- Outbreaks may occur, with large numbers of horses affected.
- There is growing evidence, at least in some populations, that probably most horses are carriers of the organism in their mesenteric lymph nodes and intermittently shed salmonella in their feces at times of stress. A limited number of horses subsequently become clinically affected.
- Signs of depression, inappetence, and colic are common, often before the onset of diarrhea.
- Acute, profuse, foul-smelling, and possibly bloody diarrhea.

Clostridium perfringens *Type A*

- Signs of profound depression and inappetence.
- Severe pain frequently unresponsive to routine analgesia.
- Death may occur before the onset of diarrhea.

Equine Monocytic Ehrlichiosis

- Horses of all ages are affected.
- Sporadic cases or outbreaks can occur.
- Endemic areas exist. The disease is more common in, but not restricted to, horses living near large waterways.
- Highest incidence in late spring, summer, and fall. Most cases occur in July through September.
- Signs of depression and inappetence are common.

Other Causes of Acute Colitis

- Prior administration of antibiotics. Agents most commonly implicated include tetracyclines, trimethoprim-sulfamethoxazole, erythromycin, and lincomycin. However, administration of any antibiotic may result in diarrhea.
- Administration of nonsteroidal anti-inflammatory agents.
- Exposure to heavy metals (e.g., arsenic, lead).
- Ingestion of alfalfa contaminated with blister beetles.
- Ingestion of plants known to cause colitis (e.g., acorn or oak, oleander, Japanese yew).
- Weight loss and other evidence of a heavy internal parasite burden.

CLINICAL SIGNS AND DIAGNOSIS

Salmonellosis

- Fever and signs of depression together with abdominal pain are typical. After early colic-type signs, a profuse watery diarrhea develops.
- Diarrhea may be voluminous, fetid, bloody, and contain mucosal tags.
- Elevated cardinal signs (increased heart rate; respiratory rate; temperature) and injected mucous membranes. Gut sounds may be decreased or absent early in the course of the disease.
- Septicemia and endotoxemia are features of the disease.

- Dehydration often is profound and is accompanied by hyponatremia (in horses that are drinking), hypochloremia, hypokalemia, and azotemia.
- Metabolic acidosis may occur because of bicarbonate loss, lactacidemia, and changes in strong ion balance. In some severe cases, plasma bicarbonate concentrations may be as low as 10 mmol/L (mEq/L).
- Leukopenia (with or without left shift) often is present before the onset of diarrhea.
- Hypoproteinemia is a common finding in cases where diarrhea has been present for a few days. Early in the disease, an increase in total plasma protein concentration may occur.
- Proximal enteritis can accompany colitis.
- Milder forms exist and some cases show initial evidence of colonic impaction followed by diarrhea.
- Complications associated with *Salmonella* septicemia include laminitis, thrombophlebitis (disseminated intravascular coagulation [DIC]), hepatitis, nephritis, and chronic colitis resulting in persistent diarrhea.
- Diagnosis is based on clinical findings and isolation of organisms from feces (see Chapter 16). At least three cultures using at least 10 g of fecal material are advised. Concomitant culture of rectal mucosal biopsies improves the likelihood of a positive culture. Postmortem cultures of colonic mucosa and mesenteric lymph nodes also may yield organisms. Recently, detection of *Salmonella* has been reported using polymerase chain reaction (PCR).

Clostridium perfringens *Type A*

- Extreme uncontrollable pain requiring euthanasia often is a feature of this disease.
- Important findings are signs of toxemia, including fever; injected mucous membranes; and a weak thready pulse.
- Hemoconcentration is common, with an elevated hematocrit (often >60%) and rapidly progressive cardiovascular collapse.
- Some horses are found dead with few prior signs.
- Diagnosis is made by increased fecal concentrations of *C. perfringens* type A being found on fecal culture.

Equine Monocytic Ehrlichiosis

- A number of syndromes can be attributed to infection with *E. risticii.*
- The most common form results in biphasic fever, signs of depression, decreased borborygmi, and mild abdominal pain.
- Moderate diarrhea lasting 24 to 72 hours may ensue.
- Horses that are severely affected develop profuse watery diarrhea lasting up to a week if supportive treatment is provided.
- Some horses develop ileus, profound toxemia, and DIC and die.
- Laminitis is a relatively common sequel to the disease (up to 25% of cases in some situations). The severity of laminitis can vary from mild to life threatening.
- Leukopenia often occurs early in the clinical course, commonly followed by a leukocytosis.
- Diagnosis is tentatively based on clinical findings and evidence of seroconversion (useful in approximately 50% of cases). An indirect immunofluorescence antibody test is the current test of choice. Titers increase before the onset of signs or within a week of their occurrence.

KEY POINT

To optimize effectiveness of serologic testing, an initial sample should be collected at the onset of signs.

- Horses with titers greater than 1:160 (that have not been vaccinated) and with signs similar to those described above are likely to have the disease.

KEYPOINT

A fourfold increase or decrease in the titer is strongly suggestive of infection with E. risticii.

- Unfortunately, more than 50% of cases remain undiagnosed.
- Serology to identify endemic areas is important, because horses showing early signs suggestive of equine monocytic ehrlichiosis can be treated aggressively.

Other Causes of Acute Colitis

- Antibiotic-induced colitis can result from overgrowth by *Salmonella* or *Clostridium* spp. There is variable severity of diarrhea and signs of depression. Inappetence, fever, and initial abdominal pain are likely.
- NSAIDs can cause colitis that produces signs of depression, hypoproteinemia, anemia, diarrhea, and secondary septicemia.
- Heavy-metal intoxication produces colitis, signs of depression, inappetence, diarrhea, possibly ileus in terminal stages, and circulatory failure.

- Cantharadin toxicosis produces signs referable to irritation in the gastrointestinal and urinary tracts. Signs include abdominal pain, signs of depression, anorexia, frequent attempts to urinate, and shock. Pertinent laboratory findings include evidence of dehydration, hypocalcemia and hypomagnesemia, and evidence of renal failure.
- Acorn *(Quercus* spp.) toxicosis produces abdominal pain, signs of depression, dysentery, shock, and possibly sudden death. Acorn husks may be found in the feces. Urinary dysfunction is also a feature of the disease.

DIFFERENTIAL DIAGNOSIS

Salmonellosis
Clostridiosis
"Colitis X"
Equine monocytic ehrlichiosis
NSAID-induced diarrhea
Diarrhea secondary to antibiotic therapy
Plant toxicoses (e.g., acorn poisoning)
Blister beetle poisoning
Internal parasitism (strongylosis, cyathostomiasis)
Heavy-metal intoxication
Bowel obstruction/strangulation
Peritonitis
Liver disease

TREATMENT

Salmonellosis

KEY POINT

Aggressive fluid and electrolyte administration is required. This involves attention to losses of total body water, which may approach 8% to 12% of the body weight in severe cases.

Electrolyte administration usually requires attention to losses of Na^+, Cl^-, K^+, and HCO_3^-. Although fluid therapy is described elsewhere (see Chapter 18), a subjective method for estimation of dehydration in horses with diarrhea is summarized in Table 7-8.

From the information provided in Table 7-8, it can be estimated what the likely fluid deficit for a horse might be.

It is important to provide the horse with sufficient fluids for ongoing maintenance and anticipated losses. For example, a 450-kg horse that has severe diarrhea and is about 10% dehydrated will require 45 L of fluid for replacement of the deficits. Maintenance is usually estimated to be 30 to 40 mL/kg/day, which amounts to an additional 15 to 20 L. If the diarrhea is severe, the ongoing losses could amount to 30 to 40 L/day. Thus, in this example, the horse may require more than 100 L of fluid during the first day of therapy.

Balanced polyionic fluids such as Multisol-R (see Fluid Therapy, Chapter 18) are good choices in the early part of the disease, followed later by maintenance fluids containing lower concentrations of sodium.

TABLE 7-8. Degree of Dehydration, Clinical Findings, and Fluid Requirement in a 450-kg (1000-lb) Horse with Acute Diarrhea

Degree of Dehydration	Body Weight Loss (%)	Fluid Deficit (L)	Clinical Findings	Fluids Required, Rate and Method of Administration (mL/kg/h)
Mild	5-7	23-32	skin turgor, moist gums, PCV 45-50%	5-10, PO or IV
Moderate	8-10	36-45	Depressed, sunken eyes, tacky gums, | HR CRT >2 sec, PCV 55-60%	10, IV
Severe	>10	>45	Cold extremities, | HR, profound depression, CRT >3 sec ± recumbency, PCV >65%	15-20 (up to 40 in the first 1-2 hours in cases of severe shock), IV

HR, heart rate; CRT, capillary refill tints

- These fluids can be administered while awaiting results of laboratory data (e.g., PCV, TPP, serum urea nitrogen (SUN), serum creatinine (SCr), electrolytes, bicarbonate).
- Losses of sodium (2000-6000 mmol or mEq) and potassium (500-3000 mmol or mEq) occur, and the estimated deficits and likelihood of correction of these by the fluid therapy need calculating (see Fluid Therapy, Chapter 18).
- Addition of potassium chloride to replacement fluids such as Multisol-R or 0.9% NaCl at a rate of 20 to 40 mmol/L (mEq/L) is very helpful. Administration of potassium in the form of KC1 via a nasogastric tube (30-60 g in 2 L water q6-8h) or as a paste mixed in yogurt or corn syrup also can be a useful way to assist in repletion of potassium losses.
- Based on the measurement of the bicarbonate value or base deficit in the plasma, estimates for the bicarbonate requirements can be made using the formula 0.3 x body weight (kg) x base deficit.
- In cases where severe bicarbonate loss has occurred, bicarbonate can be replaced intravenously or via nasogastric tube or as a paste. Oral administration is frequently the safest, with doses of 150 to 200 g/450 kg in 6 to 8 L water being given 12 or 6 hours during the acute phase of the disease, provided there are no signs of ileus.
- In extremely compromised patients, intravenous fluids can be administered by pump systems at rates of up to 1 L/min. Use of large-bore catheters (Teflon and polypropylene are the least thrombogenic) in several sites and administration via gravity from fluid bags can be used to deliver fluids at rates of 20 to 25 L/h.
- Use of hypertonic fluids (1.8% NaCl or double-strength Ringer's) has become more popular for the treatment of horses with severe diarrhea, particularly those experiencing substantial sodium losses and significant falls in plasma osmolality. These fluids should be used judiciously but are a valuable adjunct to therapy.

KEY POINT

Oral fluid administration may be of great value in horses with acute diarrhea, after correction of initial losses and stabilizing the circulatory status.

- Fluids can be administered via nasogastric tube, and the horse also can be offered fluids by free choice with provision of supplemented water. Horses can be provided with a bucket (6-8 L) of fresh water and one containing 60 to 80 g KC1. A third bucket containing 1 to 5 g/L of bicarbonate also may be provided.

KEY POINT

When offering supplementary electrolytes, it is vital that horses are always given free-choice water, because some animals will not drink electrolyte-containing fluids.

- Plasma transfusions are necessary when plasma protein values become low (albumin < 20 g/L [2 g/dL]; total plasma protein < 40 g/L [4 g/dL]), because of fluid transudation from the extracellular fluid compartment.
- The requirement for plasma is usually 5 to 10 L for a 450- to 500-kg horse, although even volumes of 2 to 4 L appear to have favorable clinical effects.
- Use of commercially available antisera (1.5-4.0 mL/kg IV once) is reputed to be of value (Polymune J, Veterinary Dynamics, Inc., Chino, CA; Endoserum, Immvac, Columbia, MO) in horses with acute colitis.
- NSAIDs such as flunixin meglumine (Treatment No. 52) are recommended to reduce pain and ameliorate some of the clinical effects of endotoxemia. The dose rate of flunixin used is recommended to be 0.25 mg/kg every 8 hours.
- Antidiarrheals/protectants such as bismuth subsalicylate (1-2 L [32-64 oz] q8-12h) via nasogastric tube are useful. These agents also may have a local anti-inflammatory effect and thereby decrease fluid secretion. Kaopectate and activated charcoal also have been recommended, although their efficacy is not proven.
- The use of antibiotics in acute diarrhea, although common, should be considered carefully. In many cases of salmonellosis, antibiotic use precedes the onset of the disease. Foals or debilitated adults may benefit from the use of antibiotics, because these agents may reduce the chance of organisms seeding to other organs if the horse is septicemic.

KEY POINT

Antibiotics do not appear to reduce the duration of salmonella diarrhea.

- Additional caution is indicated because several studies have shown rapid development of resistance to antibiotics by *Salmonella* organisms involved in hospital outbreaks.
- DMSO (0.5-1.0 g/kg slowly IV q24h as a 10-20% solution IV; Treatment No. 34) is thought to be useful because of its purported extensive

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therapeutic effects, including anti-inflammatory, antibacterial, and oxygen free-radical scavenging properties.
- Nitroglycerin patches over the digital vessels in the pasterns may be helpful in prevention of laminitis, which is a common complication of acute colitis. However, good clinical studies to prove efficacy of this treatment have not yet been reported.
- An isolation protocol should be adopted for horses with acute colitis that includes confinement to an individual stall and use of gowns/coveralls and gloves, overboots, and footbaths.

Clostridium perfringens *Type A ("Colitis X")*

KEY POINT
Many principles of therapy are similar to those described for salmonellosis, particularly voluminous fluid replacement.

- Pain is often severe in "colitis X," requiring potent analgesics. Xylazine (0.3-0.5 mg/kg IV as required; Treatment No. 109) or detomidine 5-20 µg/kg IV as required are useful agents, although they do have some adverse cardiovascular effects, causing a decrease in arterial pressure (see Table 7-5) and therefore should not be used until there is adequate circulating blood volume. Butorphanol (0.03-0.06 mg/kg IV as required; Treatment No. 15) can be combined with xylazine or detomidine for more potent analgesia (see Table 7-5).
- *Clostridium* spp. are sensitive to penicillin; therefore, administration of Na or K benzyl penicillin (22,000 IU/kg IV q6h; Treatment Nos. 85 and 86) may be of value.

Equine Monocytic Ehrlichiosis

- Clinical signs dictate the degree of therapy required. Mild cases require only appropriate nursing care.

KEY POINT
Severe cases require aggressive therapy, particularly fluids (see Salmonellosis). Also E. risticii *is sensitive to a number of antibiotics, especially tetracyclines.*

- Oxytetracycline (6.6 mg/kg IV q24h; Treatment No. 81) is the antibiotic therapy of choice. Early in the disease there may be a dramatic response to antibiotic and supportive therapy, whereas established disease responds more slowly. Antibiotic therapy should be continued for 7 to 10 days.
- Complications (e.g., laminitis) may still occur despite apparent good early response to therapy.
- Recurrence of the disease in horses in a geographic region where the disease has been diagnosed does occur.
- Prophylaxis with bacterins gives short-lived protection (3-6 months), necessitating repeated vaccinations.
- Vaccination in the face of an outbreak is often of limited value.

Other Causes of Acute Colitis

- Remove the inciting cause (e.g., discontinue NSAID therapy or treat for internal parasites).
- Provide appropriate supportive therapy as required (see Salmonellosis).

Chronic Diarrhea

Chronic diarrhea is defined as an increased fluidity of feces that has been present for 3 weeks or more. Affected horses usually have feces with a consistency more commonly associated with bovine feces. Although a wide variety of diseases can result in chronic diarrhea, a precise diagnosis is often elusive. However, the chances for successful diagnosis and therapy are greatly increased if a systematic approach is used and combined with judicious use of appropriate investigative procedures and laboratory tests. Despite the advent of new and particularly effective anthelmintics, parasitic causes of diarrhea due to *Strongylus* spp. or *Cyathostomas* are encountered relatively frequently.

DIAGNOSTIC APPROACH TO THE HORSE WITH CHRONIC DIARRHEA

- Complete history
- Thorough physical examination, including rectal examination
- Fecal examination for parasite ova or sand
- Serial bacteriology (for Salmonella)
- Hematology and serum or plasma biochemistry (e.g., full blood count, total protein and albumin concentrations, and plasma fibrinogen concentration; serum AP, GGT, AST activities, and bile acids; and plasma Na^+, K^+, Cl^-, HCO_3^-
- Abdominocentesis
- Urinalysis
- D-Glucose or D-xylose absorption tests
- Rectal biopsy
- Liver and/or abdominal ultrasound
- Liver biopsy if indicated

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HISTORY AND PRESENTING SIGNS

- Increased fecal volume of more than 3 weeks' duration.
- Affected horses have a loss of body condition.
- Appetite is often normal to increased.
- History of poor parasite management or repeated dosing with anthelmintics, with limited or no response.

CLINICAL FINDINGS AND DIAGNOSIS

- Vital signs are usually normal.
- Diarrhea with possible excoriation of skin on perineum.
- Loss of weight is common.
- Dehydration is not common, although some animals with more severe signs demonstrate significant fluid deficits.
- Total leukocyte counts are often normal.
- Hypoproteinemia (hypoalbuminemia) is a common finding and is indicative of protein loss or inflammation of the gastrointestinal tract.
- Increased numbers of parasitic ova in feces are associated with chronic diarrhea in horses that experience poor management, infrequent administration of anthelmintics, or show a lack of response after repeated anthelmintic administrations. Severe strongylosis/cyathostomiasis causes anemia, hypoproteinemia, poor coat quality, weight loss, and diarrhea.

KEY POINT

Low egg counts do not rule out the possibility of strongylosis/cyathostomiasis because the infection may be prepatent or egg production may be suppressed by previous anthelmintic administration.

- Sand in the feces often is reflective of increased sand ingestion and a secondary colitis due to its irritant nature.
- Pertinent rectal examination findings may include thickening of the bowel, possibly reflective of inflammatory bowel disease, roughened peritoneal surfaces indicative of peritonitis, enlarged mesenteric lymph nodes, abdominal abscessation, and abdominal masses.
- Increased activities of liver enzymes can be found, and there may be increased concentrations of bile acids reflective of a hepatopathy. A liver biopsy may assist in confirming a hepatopathy.
- Abdominocentesis is often normal but, depending on the cause of the diarrhea, may reveal inflammatory or neoplastic cells. Bacteria may be present in cases of chronic peritonitis.
- D-Glucose and/or D-xylose absorption tests may show decreased absorption of D-xylose or D-glucose, indicating small intestinal malabsorption (see Figs. 7-20 and 7-21). However, definitive diagnosis requires histopathology, usually performed on bowel biopsy samples or samples obtained after euthanasia. If the bowel disease is generalized, a rectal mucosal biopsy may be helpful in determining the diagnosis.

DIFFERENTIAL DIAGNOSIS

- Strongylosis (e.g., cyathostome or *Strongylus vulgaris* infestation)
- Chronic salmonellosis
- Inflammatory bowel disease/malabsorption syndromes (granulomatous enteritis)
- Sand-induced colitis
- Abdominal abscessation
- Alimentary lymphosarcoma
- Chronic liver disease/septic cholangitis
- Gastric neoplasia
- Idiopathic chronic diarrhea
- Chronic peritonitis

TREATMENT

- Strongylosis is best treated with ivermectin (0.2 mg/kg PO; Treatment No. 62) or moxidectin (Treatment No. 78) or if larval forms of strongylosis are suspected, fenbendazole (10 mg/kg PO for 5 consecutive days or 60 mg/kg once; Treatment No. 51) may be used. Fenbendazole is reputedly more effective against these forms of the parasite than ivermectin. Treatment regimens often need to be repeated because encysted larvae are resistant to the anthelmintic. Altering management to decrease exposure to parasite larvae is also important.
- Chronic salmonellosis is difficult to treat regardless of the medication used. Antibiotic therapy is useless, and one must wait for resolution of the problem. Management is required to maintain fluid and electrolyte balance.
- Inflammatory bowel disease (e.g., granulomatous enteritis, malabsorption syndrome, protein-losing enteropathy), alimentary lymphosarcoma, and gastric neoplasia respond poorly to therapy. Some types of inflammatory bowel disease respond to systemic corticosteroid therapy, but the response tends to be of limited duration.
- Sand ingestion is managed by reducing access to sand and by feeding of laxatives and diets high in fiber (see earlier section on Sand Impaction).
- Chronic liver disease may improve with good

supportive care and dietary management (see Liver Disease).
- Chronic active hepatitis may respond to antibiotic and prednisolone therapy (see Liver Disease).
- Abdominal abscessation is responsive to therapy on occasion. This is covered under the section dealing with peritonitis.

KEY POINT

Most cases of chronic diarrhea fall into the category where no specific diagnosis is made and are referred to as "idiopathic" chronic diarrhea. This makes a rational therapeutic approach difficult.

- These cases may show temporary response to iodochlorhydroxyquin (10 g/500 kg PO q24h; Rheaform, Squibb, Princeton, NJ). If there is response within 48 hours, the dosing should be continued for another 3 to 4 days, after which the dose is reduced to a level that controls the diarrhea. Those animals not responding in the first 48 hours are unlikely to respond and should have the drug discontinued.
- An additional treatment includes administration of a cecal contents liquor. Cecal contents are collected from a dead horse, and if they are not to be used immediately, they should be kept in a bucket covered with a 2- to 3-cm layer of mineral oil. The liquor is then diluted with water to a consistency that passes through a nasogastric tube, and 5 to 6 L of the preparation is administered via this method. Daily administration for a few days may be required. Response is quite variable but, in some cases, quite favorable.
- Change of diet to grass hay is also thought to be of value. Antibiotics are unlikely to be of benefit in the treatment of chronic diarrhea.

Internal Parasites

Internal parasites are ubiquitous, with horses being continually exposed throughout their lives. Infestation with *Cyathostome* and *Strongylus* spp. are the most common and are usually regarded as producing the most significant pathogenic effects. Administration of anthelmintic agents and management strategies designed to limit parasite burdens constitute one of the most important aspects of veterinary preventive medicine practiced today. Anthelmintics for the treatment of internal parasites are outlined in Chapter 18.

STRONGYLOIDES WESTERI

- This is a parasite that lives in the small intestine, commonly affecting young foals. The most frequent method of transmission of the parasite is via consumption of milk from an infected mare. Ingestion of infective larvae when eating is also a potential source of infection.
- Infections reach peak patency by 3 to 6 weeks of age, after which fecal egg counts rapidly decline. In most cases, foals have developed sufficient resistance to maintain infections at minimal levels by 12 to 16 weeks.
- Patent infections are diagnosed by fecal flotation.

ASCARIDS

- Infection with the roundworm, *Parascaris equorum,* is common in foals. This parasite has progressively less significance with increasing age and is almost clinically irrelevant in horses 2 years of age and older. Strong immunity to the parasite develops in the first year of life.
- Adult *P. equorum* live in the small intestine. Eggs are passed in the feces with foals ingesting embryonated eggs from the previous foal crop. After hatching in the intestine, larvae migrate through the liver and lungs for 17 days and then return to the small intestine. Patent infections occur by 3 months of age.
- Clinical signs include intestinal blockage and rupture often associated with recent administration of anthelminthic. Other signs reported include intussusception, ill thrift, diarrhea, and possibly respiratory signs.
- Diagnosis is confirmed by demonstration of characteristic thick-shelled eggs in the feces.

PINWORMS

- *Oxyuris equi* are regarded as the most common pin worms, with adults living in the large and small colons. Females migrate to the anus, rupture, and deposit their eggs on the perineum, in the feces, or on surrounding bedding. Infective eggs are ingested, molt, and develop into adults in the colon.
- Primary clinical manifestations relate to perineal irritation from rupture of the females, resulting in tail rubbing and a "rat-tailed" appearance.
- Diagnosis is based on examination of transparent tape that is briefly adhered to the perineum and then examined under a microscope.

STOMACH WORMS

- *Habronema* spp. and *Draschia megastoma* are spiurids that live in the stomach. Larvae may

invade skin wounds, causing "summer sores" and the eyes, inducing conjunctivitis. Flies act as intermediate hosts and transfer the larvae to these sites. Adult worms may cause gastric inflammation.
- Diagnosis of gastric infection is difficult. Cutaneous infestations are confirmed by demonstration of larvae in scrapings or biopsies.

TAPEWORMS

- *Anoplocephala perfoliata* and *Anoplocephala magna* are the tapeworms of the horse. *A. perfoliata* live in clusters around the ileocecal valve and are the most common tapeworm. In contrast, *A. magna* is a bigger parasite that is less frequently encountered.
- Clinical signs are not common, although in some cases mucosal ulceration, weight loss, colic, diarrhea, and rupture of the gut have been ascribed to *A. perfoliata.*
- The life cycle involves orbatid mites that live in pasture as intermediate hosts.
- Diagnosis is based on the demonstration of typical angular eggs in fecal flotation preparations.

BOTS

- Infestation with *Gastrophilus* spp. (stomach bots) is almost routine for horses living in temperate climates. Bots live in the stomach over winter and are then passed in the feces. Adult botflies deposit eggs on body hairs. These hatch after an increase in moisture, temperature, and the action of the lips of the horse. Larvae hatch and then progressively develop as they progress from the mouth to the stomach. Larvae produce deep pits in the stomach mucosa and may at times penetrate the stomach wall, resulting in peritonitis. Ulceration of the stomach is common.
- Diagnosis is difficult, although the presence of bot eggs on the hair coat provides strong circumstantial evidence of infection.

SMALL STRONGYLES (CYATHOSTOMES)

- Over 50 species of small strongyles affecting horses exist.
- The life cycle is direct, with females laying eggs that are passed in the feces. Here they develop into infective larvae and are eaten by grazing horses. Once in the gut, infective larvae invade the wall of the cecum and large colon, develop into the next larval stage, and remain in the gut wall for 1 to 2 months. Larvae then emerge into the gut and mature into adults.
- Cyathostomes induce pathology by larvae directly damaging the gut wall. In addition, the rapid emergence of encysted larvae in the spring can result in clinical signs of disease, including colic and diarrhea. Adult parasites produce decreased colonic motility, appetite, and weight gain. Encysted larvae have increased refractivity to anthelmintic therapy.
- Resistance by cyathostomes to anthelmintics has emerged as one of the major parasite problems in horse populations and warrants careful attention by equine practitioners.
- Diagnosis of infection is made by the presence of strongyle eggs in the feces; however, the extent of infection is not necessarily reflected by this procedure because of a variety of factors, including prepatency and previous anthelmintic administration limiting fecal egg shedding.

LARGE STRONGYLES

- This group of parasites is composed of *Strongylus vulgaris, Strongylus edentatus,* and *Strongylus equinus,* with the latter having few significant clinical effects. Large strongyles are regarded as the most pathogenic of all equine internal parasites, with *S. vulgaris* being regarded as the most injurious.
- The life cycle of large strongyles is similar to that described for small strongyles. Adults attach to the cecum and ventral colon. *S. vulgaris* larvae migrate from the gut, through the submucosal arterioles, to the cecal and colic arteries, reaching the cranial mesenteric artery within 4 months. They then return to the gut via the lumina of the arteries. The prepatent period is about 6 to 7 months.
- Larvae of *S. edentatus* have a broad migratory path, progressing via the portal veins, liver, peritoneum, and gut wall. The prepatent period is 11 to 12 months. As with small strongyles, migrating larvae of *S. vulgaris* and *S. edentatus* have increased resistance to anthelmintic therapy.
- *S. vulgaris* exerts its injurious effects by promoting inflammation in blood vessels along the course of migration. Vascular occlusion and aneurism of affected blood vessels, particularly the cranial mesenteric artery, can occur, resulting in inflammation, ischemia, and infarction, particularly of the distal small colon, cecum, and ventral colon.

KEY POINT

This process often results in thromboembolic colic (often recurrent), ill thrift, diarrhea, and possibly death in affected horses.

- *S. edentatus* larvae can damage the liver and produce peritonitis. Low-grade fever, signs of depression, inappetence, colic, constipation, and diarrhea may all be features.
- Diagnosis of large strongyle infections can be assumed if eggs are found in the feces. However, because of the prolonged prepatent period of all species in this group, eggs will not be present in the feces in animals less than 9 to 12 months old. Clinical signs also may be useful indicators of infection.
- Management practices to reduce the potential for transmission of the parasites are important. Such practices are based on routine anthelmintic administration but, more importantly, management strategies to reduce pasture contamination.

KEY POINT

More than 99% of equine parasites live on the pasture with less than 1% in horses.

Rectal Perforation

Rectal tears are most commonly iatrogenic, occurring as a complication of rectal palpation. There is often little warning, with the veterinarian only suspicious that a tear has occurred after seeing blood on the rectal examination glove after the examination. On occasion, rectal tears may result from breeding accidents when the stallion's penis enters the rectum. Idiopathic spontaneous occurrence of rectal perforation also has been reported. Most rectal tears occur in the dorsal region near the mesenteric attachment.

KEY POINT

All rectal tears should be regarded as an emergency.

CLASSIFICATION

Rectal tears are classified according to the number of tissue layers disrupted:

Grade 1—Involves the mucosa and submucosa.

Grade 2—Only the muscularis is disrupted, resulting in a mucosal-submucosal diverticulum. Rupture is rare.

Grade 3—Perforation of the mucosa, submucosa, and muscularis layers. The serosa remains intact and is the only layer between the rectum and peritoneal and pelvic cavities.

Grade 4—All layers are perforated, with direct communication between the rectum and pelvic and peritoneal cavities.

HISTORY AND PRESENTING SIGNS

- Prior rectal examination
- Blood on the sleeve after examination
- Sweating and signs of abdominal pain
- After breeding

CLINICAL FINDINGS AND DIAGNOSIS

- Blood on the rectal examination glove is common after a tear. A few flecks of blood are likely to reflect mucosal irritation and possibly a grade 1 or 2 tear. More substantial hemorrhage is common with grade 3 and 4 tears.
- The most dramatic clinical signs occur in horses with grade 4 tears. These can include progressive elevations in heart rate and signs of pain and sweating, associated with endotoxemia and subsequent shock. Signs are most profound when rupture into the abdominal cavity occurs and peritonitis ensues, with serious signs evident within a few hours of rupture occurring.
- Tears greater than 15 cm (6 inches) cranial to the anus are likely to be craniad to the peritoneal reflection and are more likely to result in peritonitis.
- Grade 3 tears often will produce similar signs to those occurring with grade 4 tears; however, they may be more slowly progressive.
- Some indication of the severity of tears and the degree of abdominal contamination can be obtained by repeated analyses of abdominal fluid (see Table 7-1).
- Clinical signs are usually less acute if fecal material enters the pelvic (retroperitoneal) cavity. However, horses with retroperitoneal cellulitis may become extremely debilitated.
- Horses with grade 2 tears may show no overt clinical signs. In this case, the mucosal-submucosal diverticulum may only be identified at a subsequent rectal examination that is performed for another reason. Other horses with grade 2 tears may be presented because of signs consistent with mild peritonitis or related to straining and fecal impaction.
- Clinical signs often are limited in association with grade 1 tears. Those cases that do show systemic involvement may show inappetence and mild signs of depression for a few days.
- Diagnosis is confirmed by careful manual examination of the rectum. Some veterinarians perform this examination using a nongloved hand to improve digital sensitivity. This examination can be augmented by the use of tranquilization with acepromazine (Treatment No. 1), xylazine (Treatment No. 109), or detomidine (Treatment No. 28). If the horse continues to show evidence

of straining, propantheline bromide (Treatment No. 96) might be considered in an attempt to reduce tenesmus. Alternatively, 25 to 50 mL of lidocaine (Treatment No. 67) mixed with an equal volume of lubricant or saline administered as an enema also is useful. In horses that still do not cease straining sufficiently to allow a thorough examination, epidural anesthesia using xylazine and lidocaine (Treatment No. 67) is indicated (see Chapter 8).
- A speculum or endoscope also is useful when examining the rectum.
- Once a tear is identified, its size, position, depth, and distance from the anus should be carefully assessed.

TREATMENT

- Grade 1 and 2 tears often will heal spontaneously. Regular monitoring for deterioration in condition should be undertaken. Systemic antibiotics, such as procaine penicillin G (Treatment Nos. 85 and 86) and gentamicin (Treatment No. 56) and possibly metronidazole (Treatment No. 75) also are indicated to assist with control of any transmural pelvic or peritoneal contamination that may have occurred. Reduction in pain and signs of mild systemic toxemia may be provided by administration of flunixin meglumine (Treatment No. 52) and/or intravenous fluids. The feeding of lush green grass or high-quality alfalfa hay helps keep the feces watery and will assist in allowing mucosal healing to occur more rapidly.

KEY POINT

Grade 3 and 4 tears are to be regarded as emergencies and life threatening if not treated rapidly and aggressively.

- The plan should involve rapid transport to a surgical referral facility. Epidural analgesia should be induced. Xylazine (Treatment No. 109) alone or in combination with lidocaine or mepivicaine is the best choice for this because it produces less ataxia and weakness than does lidocaine or mepivicaine alone. Bupivicaine may extend the duration of action when compared with shorter acting forms of local anesthetics.
- The rectum should be carefully evacuated of remaining fecal material, and it should be packed craniad to the tear back to the anus with cotton soaked in povidone-iodine then towel clamps or pursestring sutures used to close the anus.
- A number of surgical procedures have been described for treatment of rectal tears. The choice of procedure depends on the extent and size of the tear and the experience of the surgeon. Regardless of technique used, attempted correction of rectal tears is expensive, with the prognosis for grade 3 and 4 tears being poor.

Peritonitis

Peritonitis is relatively common in the horse and is classified relative to the origin of the inciting cause (primary or secondary), degree of involvement of the peritoneum (diffuse or localized), and in terms of the onset, severity, and duration of signs (peracute, acute, or chronic). The terms septic and nonseptic describe the presence or absence of bacteria, respectively. Most commonly, peritonitis occurs secondary to bowel compromise (e.g., colic), parasitism, and abdominal abscessation.

HISTORY AND PRESENTING SIGNS

- Poor management consistent with parasitism
- Trauma (direct to abdominal wall, known history of rectal perforation, previous surgery)
- Parturition or coitus resulting in uterine or vaginal perforation
- Colic with evidence of compromised bowel

KEY POINT

Signs of depression, inappetence and colic are common nonspecific signs of peritonitis.

- Weight loss in chronic cases
- Diarrhea

CLINICAL FINDINGS AND DIAGNOSIS

- Variable, ranging from almost no signs to severe systemic manifestations.
- Evidence of vaginal discharge or perforation, metritis, or a uterine tear in the mare postpartum or postcoitus.
- Rectal tear is a frequent cause, usually the result of rectal examinations and is determined by history and careful manual or speculum examination.
- Fever and elevated heart and respiratory rates are commonly present.
- Other signs include abdominal "splinting," reluctance to move, and ileus.
- Dehydration is common, with mild electrolyte derangements (especially hypocalcemia) being present.
- Shock is found in severe cases.
- Diagnosis is based on history and clinical and laboratory findings.

- Rectal examination may indicate roughened (gritty) peritoneal surfaces, thickened serosal surfaces on the bowel, pain, or evidence of an abdominal mass.
- Hematology may reveal a leukocytosis (neutrophilia), together with an elevated fibrinogen concentration. There may be anemia due to bone marrow depression in chronic cases.
- An increase in total plasma protein concentrations is found in some more chronic cases, which may be due to dehydration and/or hypergammaglobulinemia. Note: Hypoproteinemia occurs in some acute diffuse cases because of protein effusion into the peritoneal cavity.

KEY POINT

Abdominocentesis is vital for diagnosis of peritonitis.

- Changes in abdominal fluid may include increased total nucleated cell count (>50,000 x 10^6/L or 50,000/μL), increased protein concentration (>45 g/L or 4.5 g/dL), and increased turbidity (see Table 7-1).

KEY POINT

In addition to cytologic and biochemical analyses, samples should be subjected to Gram staining and bacterial culture and sensitivity testing.

- More than 60% of cases of septic peritonitis are due to mixed bacterial infections.

DIFFERENTIAL DIAGNOSIS

- Causes of peritonitis include abdominal abscessation associated with *Streptococcus* spp., *Rhodococcus* or *Corynebacterium* spp., *E. coli, Actinobacillus* spp., or anaerobes (e.g., *Bacteroides* spp.); neoplasia; parasitism; uterine perforation; gastrointestinal impaction/strangulation/rupture; enteritis; trauma to the abdominal cavity or foreign body; rectal perforation; postoperative (e.g., complication of castration or abdominal surgery); ruptured bladder, foals; associated with urachal infection, foals; and secondary to septicemia, foals.
- Laminitis
- Myopathies
- Pyelonephritis
- Pleuritis
- Colic

TREATMENT

- In secondary peritonitis, treatment of the primary disease is vital (e.g., management of internal parasites, rectal/vaginal tears, etc.).
- In acute cases, initial management includes fluid therapy to rehydrate the animal, control of cardiovascular compromise, and correction of electrolyte and acid-base abnormalities. Isotonic polyionic fluids (see Fluid Therapy, Chapter 18) should be given intravenously (10-20 mL/kg/h initially) as required. NSAIDs (e.g., flunixin meglumine 0.25-0.5 mg/kg IV q6h; Treatment No. 52) are given to control some of the effects of endotoxin production and provide mild analgesia. Endotoxin antiserum (1.5-4.0 mL/kg IV once, Endoserum, Immvac, Columbia, MO; Polymune J, Veterinary Dynamics, Inc., Chino, CA) is indicated in cases where endotoxemia exists.
- Nasogastric intubation for gastric decompression should be performed when required in cases with ileus or colic (see Figs. 7-10 to 7-13).
- Mineral oil (5-10 mL/kg PO q12-24h; Treatment No. 77) may be administered when no gastric reflux occurs and feces are dry or rectal findings are suggestive of an impaction.

KEY POINT

Appropriate antimicrobial therapy should be given based on results of culture and sensitivity.

- Initial introduction of antibiotic coverage directed against the most common pathogens implicated in peritonitis is indicated while awaiting culture results. Procaine penicillin at a dose rate of 15,000 to 22,000 IU/kg (15 to 22 mg/kg) IM every 12 hours (Treatment No. 84) in combination with gentamicin (6.6 mg/kg IV q24h; Treatment No. 56) are good choices. In cases where anaerobic bacteria (e.g., *Bacteroides fragilis,* implicated in approximately 10% of septic peritonitis cases) are suspected, addition of metronidazole (15 mg/kg PO q6h; Treatment No. 75) is indicated because penicillin may not be active against this member of bacterial species. Antimicrobial therapy often needs to be prolonged (>3 weeks), and the decision to discontinue therapy is based on clinical response and return of the leukogram, fibrinogen, and abdominal fluid constituents to normal or near normal. In cases where bacteria are susceptible to trimethoprim-sulfadiazine (15-30 mg/kg of the combined drug PO q12h; Treatment No. 108), this combination provides a useful alternative ther-

apy. Advantages include the following: the horse does not have to receive injectable antibiotics, owners can administer these agents for the long periods required, and the combination is relatively cost effective.
- Abdominal drainage through a teat cannula or an indwelling abdominal drain may be beneficial in some cases of acute septic peritonitis to assist in removal of offending bacteria and residual debris.
- Peritoneal lavage is advocated by some, particularly in cases of generalized peritonitis. Several liters of warm isotonic polyionic fluid are infused through a catheter in the paralumbar fossa, with the fluid drained from the ventral abdomen with an abdominal drain. Polyionic isotonic fluids (see Fluid Therapy, Chapter 18) are good choices because they are less irritant than saline.
- Heparin (20-40 IU/kg diluted in several liters of isotonic polyionic fluid IP or 10-50 IU/kg q8-12h IV; Treatment No. 59) has been advocated to reduce abdominal adhesions. This treatment is controversial.
- DMSO (0.5-1.0 g/kg IV q24h as a 10-20% solution IV; Treatment No. 34) is thought to be useful because of its purported extensive therapeutic effects, including anti-inflammatory, antibacterial, and oxygen free-radical scavenging properties, all of which are thought to decrease the incidence of abdominal adhesions.
- Referral of the horse for exploratory celiotomy can be considered, with the aim being to assist in defining the cause of the peritonitis or to physically remove debris/necrotic material from the peritoneal cavity.

Liver Diseases

Compromise of hepatic function in horses is probably more common than realized. However, such is the compensatory capacity of the liver that clinical signs of hepatic disease are uncommon. Hepatic failure/insufficiency usually occurs when more than two-thirds of liver function is compromised.

KEY POINT

Signs of hepatic failure include alterations in appetite, weight loss, icterus, dermatitis of unpigmented areas, derangements of central nervous system function, ascites, coagulopathies, hemolytic crises, colic, and pruritus.

The usual reason horses with liver failure are presented to the veterinarian for examination are icterus, weight loss, and deranged central nervous system function. Signs of dysfunction may be subtle, ranging from very mild behavioral alterations that may only be noted by those familiar with the horse to signs of depression, incoordination, aimless wandering, yawning, and head pressing. The cause of the hepatoencephalopathy is not clear, but it may be the result of low blood glucose concentrations, elevated blood ammonia values, or alterations in the ratios of amino acids in the central nervous system. This imbalance may lead to a failure of homeostasis in neurotransmitters or the production of false neurotransmitters. Accumulation of other toxic products that have not been cleared by the liver also may contribute to alterations in central nervous system function.

Acute Liver Failure

KEY POINT

The most common cause of acute hepatic failure is Theiler's disease (serum hepatitis).

Theiler's disease occurs only in adult horses and can involve an individual animal or there may be sporadic "outbreaks" over a matter of weeks. Most cases are reported in the fall. Administration of equine-derived biologic products has been the most frequently reported predisposing agent associated with the onset of this disease, hence the common name "serum hepatitis." The frequency of Theiler's disease has declined, probably as a result of a reduction in the use of these products. However, cases still occur where horses succumb to acute hepatic failure with no prior history of administration of biologies. Despite the epidemiology of this disease, no infectious agent has been identified. Affected horses are seronegative for human serum hepatitis antibodies. Other rare causes of acute hepatic failure include acute toxicosis with mycotoxins, pyrrolizidine alkaloids, rubratoxins, liver abscessation, suppurative cholangitis, and cholelithiasis. Hyperlipidemia and subsequent hyperlipemia with fatty infiltration of the liver in ponies also may result in fulminant acute hepatic failure.

HISTORY AND PRESENTING SIGNS

- Signs of depression
- Inappetence/anorexia
- Central nervous system derangements (hepatoencephalopathy)
- Possibly a history of administration of equine-derived biologic products (e.g., tetanus antitoxin)

- Pruritus/photosensitization
- Colic

CLINICAL FINDINGS AND DIAGNOSIS

- Signs of depression and inappetence are common.
- Icterus is normally a feature of acute hepatic failure.
- Central nervous system derangements (e.g., head pressing, aimless wandering, and yawning) due to hepatoencephalopathy may occur.
- Coagulopathies are rarely a feature of acute liver failure.
- Diagnosis is usually based on the history, clinical signs, and clinicopathologic derangements. Ultrasound examination of the liver may also provide useful information (e.g., enlarged bile ducts).

KEY POINT

The most common alterations in serum biochemistry values include elevations in the activities of AST, GGT, L-iDH (SDH), AP, LDH, and the concentrations of bilirubin and bile acids.

- Hypoglycemia is also a common feature of this disorder.
- In cases where hepatic failure occurs secondary to septic cholangitis or liver abscessation, there may be an indication of a systemic inflammatory response reflected by leukocytosis and hyperfibrinogenemia.
- A liver biopsy (Figs. 7-18 and 7-19) is valuable in confirming the diagnosis and assists with determination of the prognosis. There is usually evidence of acute hepatocellular degeneration. We have found that ultrasound examination greatly facilitates the collection of liver biopsies.

DIFFERENTIAL DIAGNOSIS

- Central nervous system diseases causing signs of depression/abnormal behavior (e.g., trauma)
- Gastrointestinal tract colic
- Systemic diseases causing inappetence/anorexia (e.g., endotoxemia)
- Primary photosensitization
- Chronic liver failure
- Chronic active hepatitis

TREATMENT

KEY POINT

Therapy for acute hepatic insufficiency should be directed at control of the abnormal behavior and support of liver function until hepatic compensation can occur. A low-protein high-energy diet should be fed.

- If the horse is anorexic, nasogastric intubation and provision of high-energy foods are recommended. Infusions with 5% dextrose (2 L/h IV) may provide some of the caloric needs of affected horses and boost blood glucose concentrations.
- Beet pulp (a good source of branched-chain amino acids) is a suitable supplement. Mixing beet pulp with molasses will improve palatability and the energy content of this supplement. The administration of branched-chain amino acids appears to reduce the severity of neurologic signs.
- Drugs such as xylazine (Treatment No. 109), detomidine (Treatment No. 28), romifidine, and chloral hydrate (all to effect) are useful for the control of manic behavior.
- Parenteral administration of B-complex vitamins is indicated.
- Efficacy of the supportive therapy is demonstrated by a rapid improvement of the horse's condition, a desire to drink and eat, and a substantial reduction in signs of central nervous system impairment.

Chronic Liver Failure

KEY POINT

Horses with this disorder usually are presented because of chronic weight loss or the onset of signs consistent with hepatic insufficiency.

Hepatic insufficiency occurs because hepatocellular compromise is such that the functional reserve capacity of the organ is surpassed.

KEY POINT

The most common cause of chronic hepatic failure is exposure to hepatotoxic plants, particularly those containing pyrrolizidine alkaloids.

Exposure to these toxins can occur directly by the ingestion of toxin-containing plants in the field or via the ingestion of contaminated hay. Recent studies show that horses need to consume more than 200 mg of the alkaloid per kilogram of body weight before severe (usually fatal) liver disease is induced. Chronic liver failure also may result from cholangitis, liver abscesses, or biliary obstructions.

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CLINICAL FINDINGS AND DIAGNOSIS

KEY POINT

Weight loss is often the most prominent feature of chronic liver failure.

- Anorexia or inappetence is common.
- A variety of behavioral changes can occur in severe cases, including yawning, head pressing, somnolence, dysphagia, dysphonia, and ataxia.
- Decreased gut sounds, colic, and watery feces occur in some cases.
- Dependent edema can occur if the plasma protein concentration becomes sufficiently low. However, reduction in total plasma protein concentration occurs less frequently than reported previously.
- Skin lesions (photosensitization/pruritus) may be seen.
- Some horses also demonstrate polydipsia and polyuria.
- Profound icterus need not be a feature of chronic liver disease, although some degree of icterus usually is found.
- Diagnosis is based on the same principles as those described for acute hepatic insufficiency.
- Elevations in the activity of liver enzymes (AST, GGT, L-iDH, AP, and LDH) and the concentrations of bilirubin and bile acids in the plasma are all good indicators of liver disease. However, in long-standing chronic liver disease, activities of liver enzymes may be normal because active damage is no longer occurring.
- Hypoproteinemia (hypoalbuminemia) and low blood urea nitrogen concentrations may be a feature of chronic liver disease.
- A liver biopsy (Figs. 7-18 and 7-19) will confirm the diagnosis of pyrrolizidine alkaloid toxicosis.
- Pathognomonic histopathologic changes include hepatomegalocytosis and biliary hyperplasia.
- A response to therapy/rest and a downward trend in the enzyme activities and bile acids probably provide the best information regarding the prognosis.

TREATMENT

- Strategies similar to those described for acute hepatic insufficiency are prescribed. Plasma transfusions (6-10 mL/kg IV) may help by promoting temporary increases in plasma protein concentration and oncotic pressure.

KEY POINT

Horses with chronic liver failure have a poor prognosis for survival, and most therapies are designed to be supportive in nature while awaiting results and hoping that hepatic function returns to adequate levels. This does not often occur.

Chronic Active Hepatitis

Chronic active hepatitis describes a group of diseases that appear to involve a relatively active and progressive liver disease. Based on empirical observations, most cases seem to have an infectious or immune basis.

HISTORY AND PRESENTING SIGNS

- Depression, weight loss, and variable icterus
- Signs present for weeks to months

CLINICAL FINDINGS AND DIAGNOSIS

KEY POINT

Febrile episodes often accompany the clinical manifestations of liver disease.

- Physical examination usually reveals elevated cardinal signs, depression, and a variety of other neurologic signs. Signs consistent with abdominal disease also may be noted.

KEY POINT

Some affected horses have an exfoliative dermatitis. This is thought to be the result of an immune-mediated vasculitis.

- Diagnosis is based on the clinical findings and laboratory procedures. Affected horses often have substantial elevations in serum GGT and AP activities, serum bilirubin concentration, and bile acid concentrations.
- An increase in TPP due to hypergammaglobulinemia is common.

KEY POINT

Diagnosis is confirmed by liver biopsy revealing periportal cholangiohepatitis and, in some cases, septic cholangitis. Bacterial culture of a portion of the biopsy sample may assist in the diagnosis.

TREATMENT

- Successful treatment requires intensive efforts involving fluids, antibiotics, corticosteroids, and aggressive supportive care.
- Support includes control of abnormal behavior

(e.g., xylazine; Treatment No. 109), provision of high-energy diets, intravenous fluids (5% dextrose in water, 2 L/h IV), and systemic antibiotics. Trimethoprim-sulfonamide (15-30 mg/kg of the combination IV or PO q12h; Treatment No. 108) or procaine penicillin G (22,000 IU/kg [22 mg/kg]) IM q12h; Treatment No. 84) and gentamicin (6.6 mg/kg IV or IM q24h; Treatment No. 56) empirically provide relief of signs in some cases.

- Favorable response to corticosteroid administration also has been reported. Prednisolone (1.5-2.0 mg/kg PO ql2h for 3 days, then 1.0 mg/kg PO q12h for 3 days, followed by 1.0 mg/kg PO q24h for 5 days, and then 0.5 mg/kg PO q24h for 5 days or as required) has been a successful treatment in our experience (Treatment No. 93).
- Improved mental status, appetite, and behavior are signs consistent with response to treatment. Some horses have shown a slow response to therapy and have taken several weeks to return to normal. Others do not respond to therapy or the costs become prohibitive and require euthanasia.

Cholelithiasis

Calculi in the common bile duct are thought to be the result of ascending or hematogenous infection from the duodenum. Calculi can be single or multiple, and if biliary stasis occurs, clinical manifestations will usually result. The condition can occur without clinical signs being evident.

HISTORY AND PRESENTING SIGNS

- Usually in horses more than 5 years old
- Mild colic, often with repeated bouts
- Inappetence
- Signs of depression

CLINICAL FINDINGS AND DIAGNOSIS

- Signs of depression and alterations in behavior (hepatoencephalopathy) may be apparent
- Signs of colic (e.g., pain, increased cardinal signs)
- Fever in some cases

KEY POINT

Diagnosis may be assisted by clinical pathology findings, including hyperproteinemia, hyperfibrinogenemia, and leukocytosis, which are common.

- Marked increases in the activity of GGT and AP and in total bilirubin and bile acids are common. The ratio of direct-to-total bilirubin is usually greater than 0.25. Increases in the activity of AST and in blood ammonia also occur.
- Coagulopathies can be present but are rare.
- Abdominocentesis may reveal an increased volume of yellow-orange fluid with an increase in protein and nucleated cells consistent with chronic inflammation.
- Bilirubinuria, as detected by dipstick examination, is common.
- Ultrasonography is of value in revealing an enlarged liver, dilated bile ducts, and hyperechoic areas in ducts.
- A liver biopsy (Figs. 7-18 and 7-19) may provide evidence of the histologic changes and bacterial organisms involved. A liver biopsy should not be performed if the horse has evidence of coagulopathy. Results of the biopsy will not confirm the diagnosis but provide important prognostic information. Common histopathologic changes occurring in association with cholelithiasis indicating a poor prognosis include periportal fibrosis, bile duct proliferation, accumulations of bile pigment, and hepatocellular necrosis.

TREATMENT

- Therapy is often unrewarding and should be tempered based on the findings of clinicopathology, biopsy, and ultrasound examinations.
- Antibiotic therapy is prescribed in an attempt to reduce biliary infection. Trimethoprim-sulfonamide combinations (15-30 mg/kg of the combination PO q12h; Treatment No. 108) or procaine penicillin G (22,000 IU/kg ([22 mg/kg]) IM q12h; Treatment No. 84) and gentamicin (6.6 mg/kg IV q24h; Treatment No. 56) for several weeks are good choices.
- Dietary manipulation and fluid therapy as described earlier for horses with hepatic failure are useful, particularly in animals showing signs of depression or behavioral abnormalities.
- Referral to a specialist clinic for surgery may be indicated in valuable animals but the prognosis is poor.

Hyperlipemia/Hyperlipidemia

Hyperlipidemia is common in horses and ponies and is the result of exercise or reduced caloric intake (e.g., colic, surgery, etc.). Hyperlipidemia is usually readily reversible after resumption of normal ingestion of food. In contrast, hyperlipemia is a more severe metabolic disorder, most frequently reported in ponies. It is characterized by signs of depression, inappetence, fatty liver,

and cloudy serum. Hyperlipemia is often secondary to a primary disorder that induces decreased caloric intake (e.g., pregnancy, lactation, colic). This section addresses hyperlipemia.

HISTORY AND PRESENTING SIGNS

- Common in ponies, although horses can be affected
- Pregnancy or lactation
- Signs of depression
- Anorexia/inappetence
- Most common in winter

CLINICAL FINDINGS AND DIAGNOSIS

- Affected ponies are often fat. Examination reveals signs of depression and lack of interest in food.
- Diarrhea is common.
- Central nervous system abnormalities can occur and may be manifested by incoordination and weakness.

KEY POINT

Clinical pathology changes include cloudy serum with triglyceride concentrations of 5.7 mmol/L (500 mg/dL) or more and elevations in liver enzyme activities (AST, AP, GGT), bilirubin, and bile acid concentrations.

- Azotemia is common in ponies with hyperlipemia.
- Metabolic acidosis may occur in the terminal stages of the disease.
- Liver biopsy reveals fatty infiltration.
- Postmortem examination reveals cloudy serum and pale swollen liver and kidneys as a result of fatty infiltration. When cut, these organs have a greasy texture. In some cases the liver may rupture, resulting in death.

TREATMENT

- Remove or treat the primary disease in an attempt to promote an increase in intake of feed. Feeding or tubing with high energy gruels is recommended.
- Constant slow infusion of glucose (dextrose 5% in water 0.5 L/h IV for a 200 kg pony) is the usual first line of therapy. Protamine zinc insulin (15 IU/kg IM q12h) followed by glucose (0.5 g/kg PO q12h) to provide energy has been recommended.
- Heparin (40-100 IU/kg SC q12h; Treatment No. 59) also may be prescribed in an attempt to assist in reducing plasma triglyceride concentrations. However, although the plasma may become clear unless the underlying disorder is corrected, heparin will not assist in resolution of hyperlipemia. This dose rate of heparin and the effects of liver dysfunction may result in alterations in hemostasis.
- The prognosis for ponies with hyperlipemia is poor.

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CHAPTER 8

Reproduction

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Reproductive problems and general reproductive examinations for pregnancy diagnosis form an important part of clinical practice for many equine practitioners. Although the rectal examination glove still forms the basis for much equine stud farm practice, the availability of ultrasound equipment and a variety of hormonal assays have permitted much greater diagnostic accuracy than previously possible. New technologies, such as shipment of cooled semen and insemination with frozen-thawed semen, are now commonly used in veterinary practice. These techniques have given the owner greater flexibility and a larger genetic pool from which to choose, but they require more precision and time. Veterinarians need to have a thorough understanding of the estrous cycle and semen physiology in addition to knowing how to interpret ultrasonographic findings and how to handle semen properly, if they are to be successful.

KEY POINT

Breeding with cooled shipped semen or frozen-thawed semen requires precise timing of ovulation in relation to insemination.

Thoroughbred stallions are routinely being shipped from northern to southern hemispheres to complete two breeding seasons yearly. Many of these horses are bred to over 100 mares every 6 months. Owners and veterinarians must cope with limited availability of the horse because of excessive bookings and restricted daily breedings. Management must be excellent, and cooperation between the farm team and veterinarian are needed. Detection of estrus, knowing the length of estrus for particular mares, and timing of ovulation with judicious use of drugs become important tools for success. In this chapter we discuss significant aspects of reproductive physiology, methods to manipulate the estrous cycle, approach to examination and diagnosis of reproductive disorders, and aspects of therapy that can be applied in a nonspecialized practice setting.

EXAMINATION OF THE REPRODUCTIVE SYSTEM

The Mare

A logical series of steps in examination of the reproductive system should be undertaken. The degree to which specialized diagnostic aids are required will depend on the history and initial findings.

HISTORY

Although specific aspects of reproductive history are often given careful attention by veterinarians, other details such as vaccination history, diet, and medical problems may be relevant to reproductive performance.

KEY POINT

The ideal breeding history consists of a sequential year-by-year account of the mare's entire performance and reproductive career.

The following details are relevant:

- Time performance career ended
- Drugs received during performance career
- History of lameness, colic

- Age
- Number of foals delivered successfully
- Number of previous pregnancies
- Abortions, early embryonic death, twins, neonatal deaths
- History of dystocias/foaling injuries
- Details of previous estrous cycles
- Duration of estrus
- Manner in which bred—natural cover, artificial insemination with fresh, cooled or frozen, thawed semen
- Time of breeding in relation to time of year or ovulation
- Number of breedings before mare failed to return to estrus
- History of discharge from vulva
- History of fluid accumulation in the uterus after breeding
- Details of previous investigations and treatment
- Knowledge of stallion's fertility to which mare was bred
- Level of management mare has been subjected to

If the history suggests difficulties in the mare conceiving the previous year, a full medical history should be obtained that includes details of all investigations and therapy. In some cases this will be readily available if the mare is on a stud farm attended by the practice in previous years. However, if the mare is new to the particular stud farm, it may be necessary to obtain records from the veterinary practice previously attending the mare.

Equine stud-farm practice is a demanding life with long hours and a busy workload for 4 to 5 months of the year. Because of this, many veterinarians keep inadequate records. It is important to institute a record system for brood mares that is simple but allows quick referral to features such as state of the ovaries, size of follicles, appearance of the cervix, color of discharge, and so on. Mares that are difficult to get into foal may require examination many times during the course of a breeding season. In these cases, it is essential that accurate records be kept to allow comparisons to be made between examinations.

EXAMINATION OF THE EXTERNAL GENITALIA

It is best to examine the external genitalia during estrus, although this option is obviously not always available.

KEY POINT

One of the single most important steps in a reproductive examination is assessment of perineal conformation.

The vulva and the anus should be in the same plane; when the anus has a "sunken" appearance and the vulva is sloping forward, the mare is prone to infection, a result of pneumovagina and fecal contamination. The vulval lips should be examined for any signs of previous lacerations or evidence of Caslick's surgery.

KEY POINT

The relationship of the pelvic floor to the orientation of the vulva needs to be examined.

If the pelvic floor is palpated and more than 4 cm of the vulva lies dorsal to it (Fig. 8-1), the vagina is predisposed to cranioventral rotation leading to pneumovagina and contamination. These mares should have a Caslick's operation performed. The lips of the vulva are parted to determine the integrity of the vestibulovaginal sphincter. The sphincter is intact when the labia can be spread slightly without air entering the cranial vagina. The clitoris should be examined, and although it is seldom affected by clinical disease, it can act as a reservoir for contagious equine metritis (CEM) organisms. The clitoral fossa and clitoral sinuses are sites that have been shown to harbor the CEM organism, and these areas can be cultured if infection is suspected.

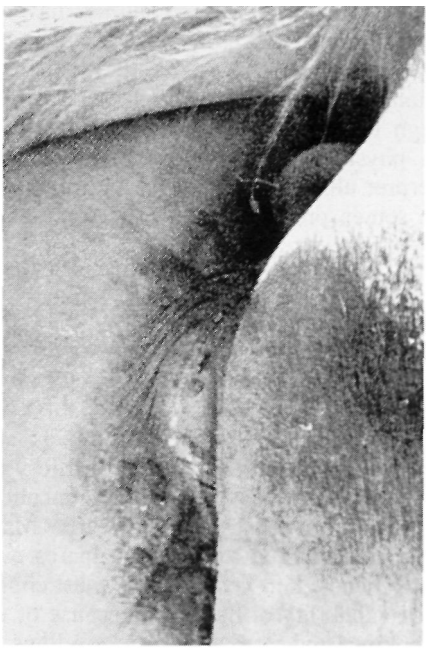


Figure 8-1. Cranial ventral rotation of the vulva results in aspiration of air and feces. If more than 4 cm of the vulva lies dorsal to the pelvis, a Caslick's operation should be performed.

They also harbor bacteria that cause endometritis. A veterinarian may induce an iatrogenic uterine infection by passing his or her arm repeatedly through the vestibule, especially if uterine therapy is performed after ovulation.

VISUAL EXAMINATION OF VAGINA AND CERVIX

Integrity of the cervix is vital for pregnancy to be sustained. Examination not only allows assessment of abnormalities but also permits evaluation of changes associated with various stages of the estrous cycle. Before internal examination, the vulva should be cleaned with disinfectant so that infection is not introduced at the time of examination.

KEY POINT

All disinfectants are potential irritants, and therefore care should be taken to prepare them at appropriate recommended concentrations.

The commonly used technique of splashing disinfectant into a bucket of water until the correct color appears is inaccurate and may lead to a concentration of disinfectant that is damaging to the sensitive mucous membranes.

The mare should be examined out of direct sunlight. After the labia are washed, they should be blotted dry and the inner edges of the labia wiped clean. The vagina may be examined with a sterile instrument such as a plastic, cardboard, or glass tubular speculum or a sterile duck-billed speculum. However, the technique of immersing the duck-billed speculum in disinfectant between mares can result in transmission of CEM. The light source should have a bright focally directed beam. The speculum is inserted into the vestibule by parting the labia with a hand. The speculum should meet resistance at the vestibulovaginal sphincter. If the speculum slides easily into the anterior vagina, the integrity of the vestibulovaginal seal, the second anatomic barrier against ascending infections, is lost. Once the speculum is in the anterior vagina, any purulent discharge, cervical lesion, or urine pooling should be noted.

KEY POINT

Urovagina, a cause of endometritis, may be observed only on the day before ovulation, when estrogen concentrations and perineal relaxation are highest.

It also may be noted during the first postpartum estrus when the vaginal structures are stretched craniad by the weight of the involuting uterus.

The cervix changes in appearance due to hormonal influence. In *diestrus,* the cervical and vaginal surfaces become pale and dry. The color of the membranes is typically gray, with a yellowish cast. The external cervical os projects into the cranial vagina from high on the wall and is tightly contracted, lending itself to the terms high, dry and tight, or "rose bud." In *estrus,* there is increased shinyness of the cervical and vaginal mucosal surfaces from mucus secretions. Near *ovulation,* the cervix is congested, moist, and lies on the floor of the vagina. Cervical and vaginal color in *anestrus* is blanched, almost white. The cervix becomes atonic and flaccid and often gapes open to reveal the uterine lumen.

KEY POINT

Physiologic hyperemia needs to be differentiated from hyperemia due to inflammation. The latter usually is brighter red in appearance and there may be cloudy fluid lying on the vaginal floor.

Artifactual reddening is produced quickly when air contacts the tissues, so evaluation of color must be made shortly after insertion of the speculum. Vaginoscopy can be used to identify rectovaginal fistulas, persistent hymen, and necrotic vaginitis after foaling. Mares with fistulas will have a green watery discharge. If a maiden mare has a complete hymen, the speculum cannot be advanced into the vagina. Necrotic vaginitis, a sequela to dystocia, can be life threatening. On vaginal speculum examination, the vaginal walls will be grayish black, necrotic, or granulomatous. There will be a foul odor when the speculum is passed into the vagina.

Direct palpation of the cervix using aseptic technique permits assessment of cervical integrity and identification of any adhesions. When the gloved hand is introduced into the mare's vagina, the labia should be parted with the fingers of the other hand to reduce contamination. The entire cervical canal should be palpated with the index finger in the canal and the thumb and third finger on the external os. Part of the cervical os may be torn or the cervical body may be ruptured such that it is permanently overstretched.

KEY POINT

Because of the importance of the cervical seal during pregnancy, if a lesion is noted, the cervix should be reexamined during diestrus to determine if it closes completely.

RECTAL EXAMINATION

Preparation for Rectal Examination. Rectal examination provides a technique whereby the

uterus and ovaries can be assessed and pregnancy either ruled out or diagnosed. Rectal examination is not a technique without some risk, because rupture of the rectum can occur, particularly in nervous or fractious horses. The examination should be performed with the mare adequately restrained and with a generous amount of obstetrical lubricant on one's hand and arm.

KEY POINT

If the mare strains excessively, 50 mL of 2% lidocaine can be added to 250 mL of lubricant and infused into the rectum to reduce the risk of a rectal tear. An alternative is propantheline bromide (Treatment No. 96) at a dose rate of 0.1 to 0.2 mg/kg.

Propantheline should be administered approximately 5 minutes before a rectal examination is performed. Some veterinarians have reported colic after propantheline administration. In some mares, chemical restraint may be needed. Xylazine or detomidine should be used only in combination with other tranquilizers or opioids when working near the hindlegs of a horse because some mares may experience hypersensitivity over the hindquarters and kick viciously.

KEY POINT

Some rectal examination gloves have a sharp edge along the seam. For this reason, we find that there is less likelihood of damage to the rectum if the glove is turned inside out before its use.

The hand should be introduced after application of a liberal volume of lubricant, and the fingers should be held closely together in a cone shape. Once the hand is in the rectum, a brief period should be given for the mare to relax. The arm should initially be advanced further into the rectum than the level required and then withdrawn, because this usually provokes less discomfort and straining. The feces present in the rectum are then removed with the palm of the hand facing dorsally. After application of further lubricant, the hand is reinserted, and any additional feces are removed. The arm is moved craniad, and if resistance or straining is encountered, the arm should not be advanced further. Most cases of rectal rupture occur when the hand is advanced cranially as the mare strains.

Systematic Exploration of the Reproductive Tract via the Rectum. A systematic examination should be adopted, commencing with one ovary (usually the side opposite the particular arm used) and proceeding along the uterus to the opposite ovary.

KEY POINT

The size and turgidity of follicles, uterine, and cervical tone need to be recorded and correlated with teasing records to allow prediction of the time of ovulation.

OVARIAN EXAMINATION. The ovaries are attached to the sublumbar region by the broad ligament and can be located by using the ilium as a reference point. The only normal structure that can be confused with an ovary is a fecal ball in the small colon.

KEY POINT

The size of the ovaries varies according to the interval in the breeding season when the examination is performed.

During winter to early spring, when mares are in anestrus, the ovaries are small, approximately 3 cm in diameter. During spring, with the mare cycling, the ovaries approximately double in size. An ovary can be held between the thumb and index finger to determine follicle size (range 1.5-6 cm) and turgidity. As follicles mature, they project above the ovarian surface (Fig. 8-2). A "shoulder" forms around the base of the growing follicle and increases its angle as ovulation approaches. The turgidity of the follicle generally changes with maturity, progressing from firm and thick-walled to fluctuant and thin-walled. On the first or second day of estrus, the dominant follicle will range from 25 to 30 mm in diameter. The follicle will increase in size by 3 to 5 mm daily until it ovu-

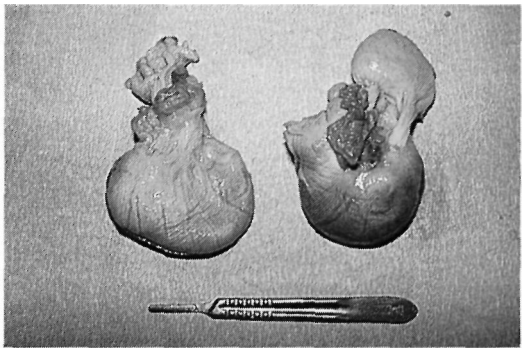


Figure 8-2. Ovary on the right (scalpel handle) has a large follicle on its concave surface. The surface of the follicle is smooth and fluctuant on palpation.

lates at which time it will be 38 to 60 mm in diameter. Accurate determination of follicle size, projection from the surface, and turgidity are important in determining the imminence of ovulation. None of these observations are absolute. Small ovaries, as found in young mares, are prone to ovulate smaller follicles than large ovaries of pluriparous mares.

Collapse of the follicle is generally assumed to be the time of ovulation. Typically, this collapse is felt as a crater or pit at the location of the follicle. When palpating this crater, the mare responds to the local pain involved by tensing the flank muscles on that side or raising the leg. The borders of the crater are sharp and the cavity feels empty for about 8 hours. As the hemorrhage of ovulation organizes into a clot to form the corpus hemorrhagicum, the mushy consistency of the cavity becomes progressively firmer until 24 hours after ovulation. After 24 hours, the crater may redistend with clot and serum, suggesting the presence of another follicle on palpation. It will be smaller, however, than the original follicle. These changes are best evaluated by repeated rectal examinations or by ultrasonography. Consideration of other estrual signs and the tone of the uterus are helpful if there is confusion on what is being palpated. The mature (5 days after ovulation) corpus luteum is seldom detectible by rectal palpation.

UTERINE EXAMINATION. The uterus is palpated to determine tone, size, and consistency. To locate the uterus, the examiner reaches 40 to 55 cm into the rectum and cups the hand by bending the wrist and fingers and gently pulls the relaxed rectal wall caudally and slightly ventrally. As the hand nears the pelvis, the uterus will be felt as a soft, pliable, and relatively flat tissue lying transversely in the caudal abdomen. It is generally 4 to 7 cm wide and 2 to 5 cm thick. In older pluriparous mares, the uterus will be found ventral to the pelvis, whereas in young nulliparous mares, the uterus may be located slightly dorsal to the pelvis. The initial impression on tone and size is best gained by cupping the fingers over the cranial brim of the uterus, with the palm on the dorsal aspect, and by moving the hand laterally and medially from the tip of one uterine horn to the other.

The uterus is tubular and feels compact on palpation when under the influence of progesterone. The tubularity of diestrus progresses to relaxation in proestrus and estrus. Estrogens produce uterine edema, which feels like a soft thickening of the uterus. There is also the impression that the thickened wall could be compressed easily between the fingers. In anestrus, the uterus becomes flaccid, thin walled, and often quite indistinct.

As early as 17 or 18 days postovulation, there are characteristic changes in uterine and cervical tone in pregnant mares that can be used to differentiate the pregnant mare from the mare in diestrus. In the gravid uterus the cervix elongates and is firm and tubular. The uterine wall is also toned and has the texture of a high-pressure water hose. Both margins of the vesicle are quite distinct, making an abrupt junction with the nongravid portion of the uterus.

KEY POINT

The presence of increased uterine tone and evidence of slight bulging on the ventral surface of the uterus indicate early pregnancy.

By 25 days the chorionic vesicle, approximately 2.5 cm in diameter, is palpable in one uterine horn. By 30 days, the vesicle is 4 to 4.5 cm in diameter. The bladder may be mistaken for a chorionic vesicle that is 45 to 90 days of age as uterine tone begins to diminish, especially at 45 days in the area of the vesicle. However, the bladder wall is much thinner than that of the pregnant uterus. By 90 days the pregnant horn is the size of a small football and has dropped over the pelvic brim into the abdomen.

CERVICAL EXAMINATION. The cervix is palpated by pressing it firmly using the finger tips against the pelvis. The effects of estrogen and progesterone on the cervix are similar to those on the uterus. Estrogens produce cervical softening, shortening, and edema. Late in estrus before ovulation, the cervix is so soft that it flattens readily against the pelvis. The diestrual cervix is long, tubular, and readily palpable. In anestrus, the cervix becomes soft and indistinct.

BACTERIOLOGIC AND CYTOLOGIC EXAMINATIONS

Taking a cervical or uterine swab is one of the most common procedures carried out in equine stud-farm practice. However, results are often misinterpreted and therefore misleading. Culture results must be correlated with cytologic findings. When cytologic findings are persistently positive and cultures are negative, the interpretation is that the cause of the inflammation remains to be determined. When cultures are positive and cytologic findings are repeatedly negative, contamination of the culture should be the usual conclusion.

KEY POINT

The caudal genital tract has a normal flora of bacteria, and isolation of bacteria from a uterine swab does not necessarily constitute infection.

Prolonged inflammation in the uterine lumen results in infertility. This inflammation can be a consequence of the deposition of semen, bacteria, urine, feces, air, caustic chemicals, or antibiotics into the uterus. Bacteriology identifies only bacteria. A smear taken from the uterus for cytologic examination identifies acute inflammation and is especially helpful in establishing a diagnosis in chronic or low-grade endometritis. There are a number of methods for collecting and examining uterine luminal cells. To be diagnostically useful and practical under field conditions, the examination must be rapid, simple, and produce consistent results.

The following technique is recommended. Samples of endometrium and luminal contents are obtained with the same instrument used to obtain uterine cultures, a disposable guarded culture instrument with a plastic cap (Kalayjian Industries, Long Beach, CA). The swab in this instrument is guarded by the cap and outer tube as it is passed through the cervix. Once in the uterus, the swab is exposed by pushing it against the cap that snaps open. The cap remains attached to the tube by a plastic stalk. After the swab is saturated for the microbiologic sample, it is retracted back into the tube. Then, while the tube is still in the uterine lumen, the entire tube is rotated briskly several times. This causes the cap to collect a sample of endometrium and uterine fluid. As soon as the instrument is withdrawn from the mare, the cap is cut off and a slide prepared from the drop of fluid and cellular material it contains. This is done by placing the open end of the cap on a slide and tapping it briskly with the index finger to transfer the sample to the slide. The sample is spread gently and allowed to air dry.

A simple three-step staining process (Diff-Quik) has proven practical and effective for field use. Staining takes a few minutes and slides may be examined wet or allowed to dry for later evaluation. Permanent mounting of a cover slip is recommended for long-term storage of slides.

A cytologic specimen that has been collected and prepared properly will have sheets of epithelial cells found throughout the slide (Fig. 8-3). If no epithelial cells are seen, there is no assurance that the uterine lining was sufficiently scraped. This problem tends to occur in older pluriparous mares whose uterus is located ventral to the pelvis. If many neutrophils are seen, inflammation is present (Fig. 8-4).

Interpretation of most samples processed in this manner is straightforward as being either positive or negative. Occasional samples show few neutrophils. In these cases, a ratio of neutrophils to epithelial cells should be calculated. If this ratio is more than 1 neutrophil to 10 epithelial cells, inflammation may be judged to be significant.

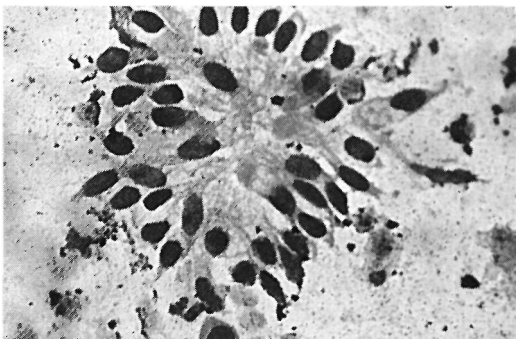


Figure 8-3. A cytologic specimen taken from the uterus should have sheets of epithelial cells present. If there are none, the sample may not have been procured properly.

KEY POINT

In the interpretation of cytologic findings, there should be sheets of epithelial cells on the specimen. Inflammation is present when there are greater than five neutrophils per high-power field.

Cultures are made either through a speculum or by manual insertion to the desired location. Samples should be taken from the uterus without contamination by extraneous bacteria. To eliminate these sources of contamination, one must select a clean site for examining the mare, prepare the animal for aseptic examination, and use an instrument that samples only from the uterus.

In our opinion, the best time to recover uterine

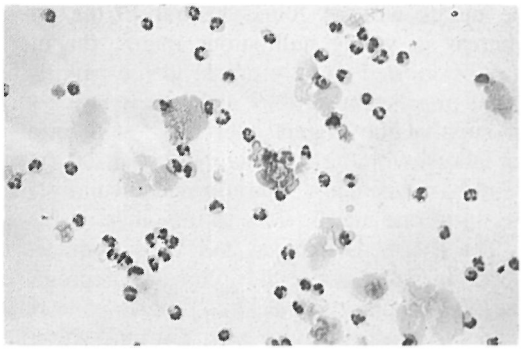


Figure 8-4. Greater than 5 neutrophils per high power field on a cytologic specimen taken from the uterus indicates inflammation. Inflammation may be caused from aspiration of air, feces, or urine or the presence of bacteria, fungus, or semen.

organisms is the first or second day of standing estrus, even if the cervix is not fully relaxed. At that time, secretions from the endometrium are increasing, making a moist swab easier to obtain, and the full flushing action of estrus has not yet developed. During diestrus, the endometrium is often dry, making bacterial recovery more difficult. Also, one may inoculate the uterus with bacteria carried on the hand from the vestibulum. The technique of culturing the uterus in the winter is questionable because the chances of not culturing bacteria associated with chronic endometritis are great.

Immediately after collection, uterine swabs should be placed directly on the final medium for culture. This gives positive correlation between numbers of organisms on the swab and numbers of colonies on the plate. Bacteria are lost as the swab dries; any method that delays drying such as refrigeration, adding sterile saline to the swab, or placement in a culture transport medium extends bacterial viability. When many mares are examined at one farm over a prolonged period, it may be advantageous to streak culture plates on location.

KEY POINT

Gross inspection of culture plates and making Gram stains enables the veterinarian to make initial therapeutic decisions.

Plating the sample on blood agar and one selective gram-negative medium is usually adequate. Plates are incubated at 37°C (98.6°F), preferably in a candle jar to provide microaerophilic conditions, and inspected daily. The number of colonies should be noted. Pure cultures are more significant than mixed ones.

ENDOMETRIAL BIOPSY

Endometrial biopsy is a valuable diagnostic tool for the problem mare. Candidates include barren mares with a clinically evident abnormality of the reproductive tract that fail to conceive after repeated breedings to a stallion of known fertility, mares with a history of early embryonic death, or mares with a history of acyclicity during the physiologic breeding season. Changes in the endometrium sometimes may be found on histologic examination when gross examination has not revealed any abnormalities. Experienced pathologists and veterinarians who do broodmare work are able to effectively classify endometrial changes into various grades, which permits a prognosis to be given for future reproductive performance. The biopsy should be collected during estrus or diestrus.

KEY POINT

The major evaluation is the assessment of glandular activity, severity of fibrosis, inflammation, and lymphangectasia.

A small sample of the endometrium may be collected easily from a mare using approximately 60 cm alligator-jaw biopsy forceps (Pillings biopsy punch). The forceps have basket-type jaws that are 2 cm long and 4 to 5 mm wide (Fig. 8-5).

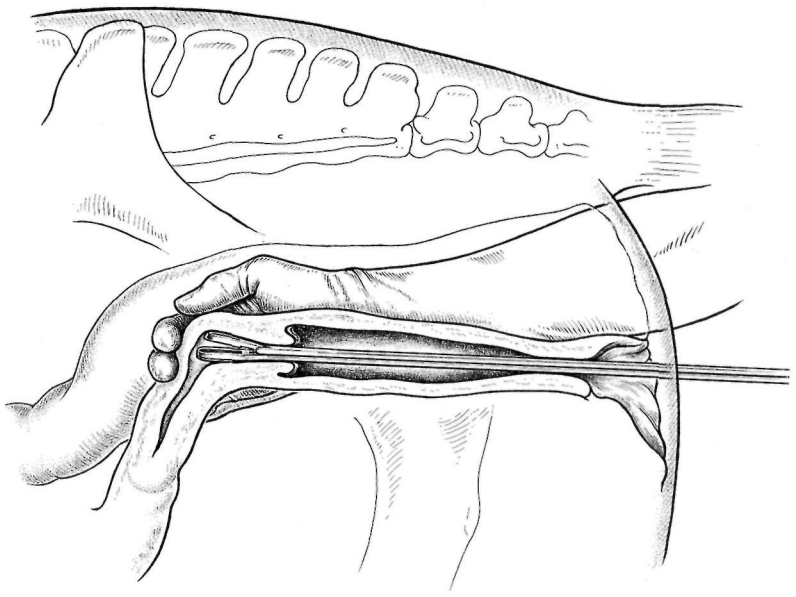


Figure 8-5. Uterine biopsy forceps shown being inserted into the uterus of a mare.

Although the size of the sample collected looks rather large, problems associated with endometrial biopsy are seldom encountered. If the sample size is too small, accurate interpretation is not possible. A useful method for sterilization of the forceps between mares is immersion in dialdehyde (Cidex; Treatment No. 31) for 10 minutes before use. If Cidex is used, the forceps should be rinsed carefully with sterile saline, because the chemical is very irritating to tissue. After thorough cleansing of the perineum and evacuation of the rectum, the sterile biopsy forceps are introduced into the vagina with a sterile-gloved hand. It is usual to do this with the left hand so that the right hand can be used to operate the jaws of the biopsy forceps. The index finger of the left hand is passed into the uterus via the cervix, and the biopsy forceps is passed along the finger into the uterus. The left arm is then withdrawn and placed in the rectum, where the uterus and forceps can be palpated. Using the hand in the rectum to guide the forceps, a piece of endometrium is pressed into the side of the open jaws of the biopsy forceps. The jaws are closed and the biopsy taken, after which it is placed in Bouin's solution for fixation before submission for histology. Within 24 hours, the fixative should be changed to alcohol or the tissue sample will become too friable for good sections to be cut.

Because of the changes in endometrial appearance that occur with ovarian activity, the stage of the estrous cycle during which the biopsy was collected needs to be recorded. Classification of the histologic findings has been suggested by Kenney and Doig (1986), because such a system has been found useful in predicting future reproductive performance. The crucial histologic assessments include the following:

1. Evaluation of the inflammatory response, particularly in the stratum compactum and luminal epithelium. Acute changes are characterized by polymorphs (neutrophils), whereas in chronic endometritis there are lymphocytes and a variety of other cells, including plasma cells.
2. Determination of endometrial fibrosis. Endometrial fibrosis is of particular significance because the findings indicate the likelihood of long-term problems in either conception or carrying a pregnancy to term.
3. Severe lymphangectasia indicates that the mare cannot quickly clear edema from uterine tissues, resulting in endometrial irritation and inflammation.

The classification scheme suggested by Kenney and Doig is as follows:

Category I—Essentially normal endometrium. Mares within this classification can be anticipated to have a foaling rate of up to 90%.

Category IIA—Moderate inflammatory changes that involve the superficial endometrial layers. Minor fibrotic changes associated with some of the glands also would allow classification. The anticipated foaling rate for mares within this classification is in the range of 50% to 70%.

Category IIB—Inflammatory changes are more widespread and involve deeper tissue. This also applies to any fibrosis that is more widespread and severe than in mares in category IIA. There may be widespread lymphangectasia. The anticipated foaling rate for mares in this category ranges from 10% to 50%.

Category III—Widespread and severe inflammatory or fibrotic changes. Lymphatic stasis is widespread and accompanied by palpable changes in the uterus. Endometrial atrophy or hypoplasia with gonadal dysgenesis may be found. Mares within this category have a very poor prognosis for future breeding, with foaling rates varying from 0% to 10%.

Care should be taken in pronouncing a mare sterile on the basis of single or even multiple biopsies. The occasional exception can be embarrassing.

ULTRASONOGRAPHY

Ultrasound examination of the reproductive tract of mares is a widely used technique in stud-farm practice. A 5-MHz linear-array probe is used most commonly. It is introduced via the rectum after removing the feces. Its use enables the operator to detect a conceptus on day 10 (day 0 is day of ovulation), follicles as small as 3 mm in diameter, and the presence of a corpus luteum through most of diestrus. A linear-array, sector or annular-array 2.5- or 3-MHz transducer is used to evaluate fetal viability by a transabdominal approach.

A systematic scanning technique needs to be developed to eliminate the possibility of missing structures or an embryonic vesicle. One method for examination is to move the probe from uterine body to left uterine horn, left uterine horn to left ovary, reexamine the left uterine horn and uterine body, evaluate the right uterine horn and right ovary, return to the uterine body, and then scan the cervix.

Normal Anatomy

The uterus is visualized as a cross-sectional image. With the probe held in a sagittal plane, the cranial aspect of the uterus is seen on the right of the ultrasound screen and the caudal aspect on the left of the screen. The uterus undergoes dynamic changes due to hormonal influences that can be visualized by ultrasonography. During *estrus,* uterine horns are well rounded, and both horns and

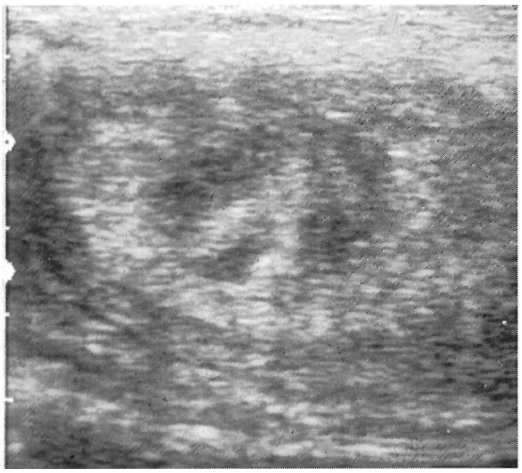


Figure 8-6. Ultrasound image of uterus during estrus. There is an interdigitating pattern of alternating echogenic and nonechogenic areas, similar to spokes on a wheel. This pattern is due to uterine edema.

body have an interdigitated pattern of alternating echogenic and nonechogenic areas similar to the spokes on a wheel or slices of an orange (Fig. 8-6). Endometrial folds generally parallel estrogen production and are visible at the end of diestrus, becoming quite prominent as estrus progresses and decrease or disappear 12 hours before ovulation. A small amount of fluid may be present normally within the uterine lumen during estrus. As ovulation approaches, the shape of the follicle changes from spherical to teardrop (Fig. 8-7). This change, in combination with loss of the endometrial folds, can be used to estimate time of ovulation. Endometrial folds are sometimes seen in early pregnancy, 16 to 28 days gestation, especially if large follicles are present on the ovaries.

During *diestrus,* the echo texture is more homogeneous, and the uterus is well circumscribed. The uterine lumen is often identified by a hyperechogenic white line in the area of apposing endometrial surfaces. During *anestrus,* the uterus is flat and irregular and may contour closely to surrounding abdominal organs. During *pregnancy,* the uterus appears similar to that of diestrus except that endometrial folds may again appear after day 16 and a vesicle will be present.

Follicles are nonechogenic and appear as black roughly circumscribed shaped images. Follicles may appear irregularly shaped due to impending ovulation, compression by adjacent follicles, or luteal structures. Within 24 hours of ovulation, a preovulatory follicle softens and becomes teardrop-shaped as the follicle progresses toward the ovulation fossa. After the follicle ruptures, a corpus hemorrhagicum forms. It has one of two appearances on ultrasound (Figs. 8-8 and 8-9). It is either a uniformly echogenic circumscribed image or it contains a centrally nonechogenic center. Echogenic lines attributable to clotting and fibrinization in the central nonechogenic center may be present. The corpus luteum is highly echogenic on the day of ovulation, and echogenicity decreases and then plateaus during the first 6 days and then increases over days 12 to 16.

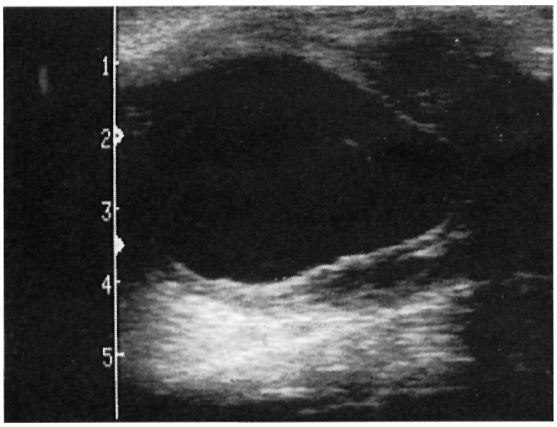


Figure 8-7. Ultrasound image of follicle near ovulation. Note its pear shape and the hyperechoic rim of the follicle.

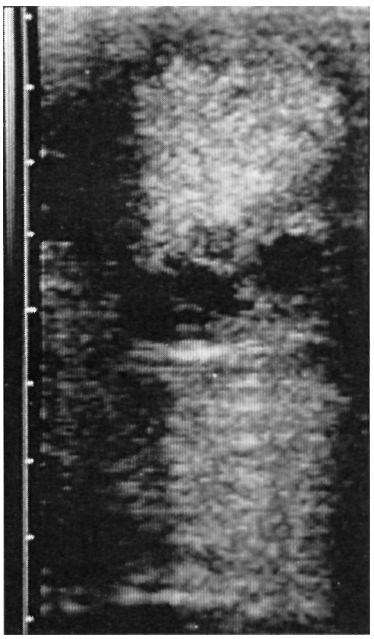


Figure 8-8. Ultrasound image of a uniformly echogenic corpus hemorrhagicum.

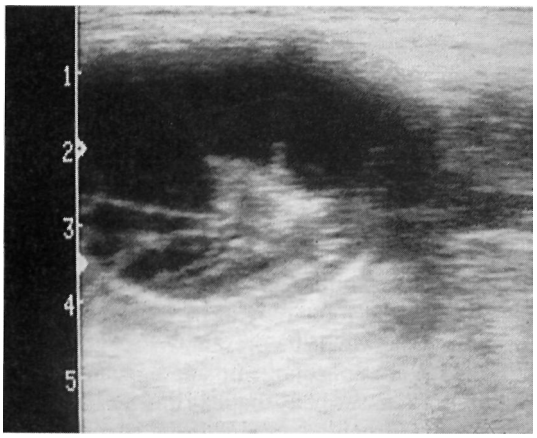


Figure 8-9. Ultrasound image of a corpus hemorrhagicum that has a centrally nonechogenic center. The echogenic lines represent fibrinization of the blood released into the cavity at ovulation.

Pregnancy

The first ultrasound examination for pregnancy determination is usually performed between 14 and 20 days after ovulation even though pregnancy status can be determined as early as day 10. The early equine conceptus is highly mobile within the uterine lumen and moves between the uterine horns and uterine body from the time it enters the uterus on day 5.5 until days 16 to 17. Because the vesicle is moving, it may be found anywhere within the uterine lumen from the tip of a uterine horn to the cranial aspect of the cervix. After day 17, the vesicle fixes at the caudal portion of one of the uterine horns near the bifurcation. Fixation of the early conceptus on days 16 to 17 is apparently caused by increased uterine tone and thickening of the uterine wall and rapid growth of the conceptus.

KEY POINT

The most important application of ultrasonography for pregnancy diagnosis is the early determination of twin pregnancy so that termination of one of the two pregnancies can be performed.

Twin or double ovulations are common in mares, with some reports suggesting a rate of up to 40% of ovulations. Despite this, the incidence of twin births is less than 1%, indicating a high rate of embryonic loss. Ultrasound examinations for the detection of twins should be performed before fixation on day 17 because two vesicles that are closely apposed cannot be separated easily after that time. Before day 17, the two vesicles are moving continually and will separate on their own. One of the two vesicles can be crushed during a second ultrasound examination performed 30 minutes to an hour after the first. If two vesicles settle in opposite horns, crushing one of the two should be performed before day 25. If the initial ultrasound examination is performed after day 17 and the two vesicles are closely fixed, there is a 50% to 60% chance that one of the two will reabsorb on its own by day 40 of gestation. The final decision on whether to abort the twin pregnancy with prostaglandin should be made before day 36, the time of endometrial cup formation. Once the endometrial cups are established, because of the high concentrations of equine chorionic gonadotropin (eCG) produced, the mare will not return to ovarian cyclical activity for 3 to 4 months, even if the pregnancy is terminated.

Until day 18, the equine vesicle is spherical and is rapidly increasing in size. Between days 17 and 26, the vesicle becomes irregular in shape and its rate of growth plateaus (Fig. 8-10). Increasing uterine tone on days 16 to 17 in combination with rapid growth of the vesicle may explain the change in vesicle shape. After day 26, the vesicle resumes growth but at a slightly slower rate.

KEY POINT

The embryo is first detected within the vesicle by ultrasound at days 20 to 22. The heart beat is commonly detected about day 24 and is an important indicator of embryo well-being.

The location of the embryo within the vesicle varies with gestational age. The embryo proper

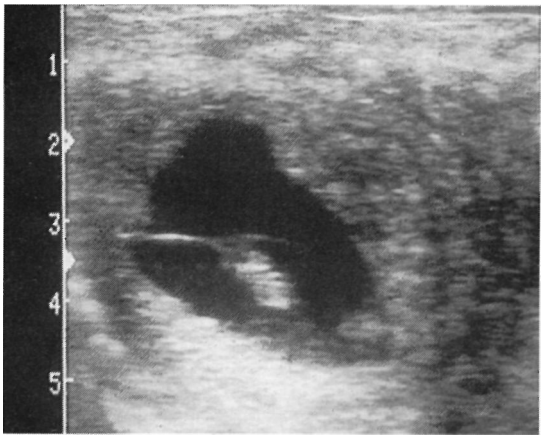


Figure 8-10. Ultrasound image of a 26-day embryonic vesicle. The echogenic line represents the division between the yolk sac and the allantoic sac. The embryo is the large white structure in the middle of the vesicle.

moves from the ventral (day 22) to the dorsal (day 40) aspect of the vesicle between days 22 and 40. This positional change is caused by expansion of the allantois with concurrent contraction of the yolk sac. After day 40, the yolk sac degenerates and the umbilical cord elongates from the dorsal pole, permitting the fetus to gravitate to the ventral floor where it is seen in dorsal recumbency from day 50 onward. Apposition of yolk sac and allantois results in a visible line that is normally oriented horizontally. At times, orientation of the yolk sac and allantois is abnormal and the line is oriented vertically. The abnormal orientation does not adversely affect embryo development, but it may be confused with twin vesicles.

Pathologic Changes Visualized by Ultrasonography

Uterine cysts, accumulation of intrauterine fluid, and air can be identified by ultrasonography. Uterine cysts indicate an ongoing degenerative process within the endometrium. Cysts will vary in size from 5 to 50 mm. There may be many small cysts that appear to be imbedded within the endometrium or just a single large cyst that contains many partitions. It is helpful to record the presence of intraluminal cysts visualized by ultrasonography because they may be confused with an embryo.

KEY POINT

Characteristics of the embryo that can be used to differentiate it from a uterine cyst include early mobility of the embryo (days 10-16), presence of specular reflection on the upper and lower surfaces of the vesicle, spherical appearance without partitions, and growth rate.

Fluid present within the uterus during diestrus and the early postovulatory period has been associated with early embryonic death, endometritis, and decreased conception rates. A small amount of fluid in the uterine lumen during estrus does not appear to adversely affect fertility. Air within the uterus appears as multiple hyperechogenic reflections. If present in mares during estrus or directly after ovulation, the mare should be closely examined for pneumovagina.

EXAMINATION OF THE UTERUS USING ENDOSCOPY

Flexible fiberscopes have proven useful in examination of the uterus. We use a flexible colonoscope to view the endometrium directly. The colonoscope is prepared by immersion of the viewing end and 60 cm of the distal end of the scope in gluteraldehyde (Cidex; Treatment No. 31) for 10 minutes. After the endoscope is removed from the Cidex, it is rinsed carefully with sterile saline before being inserted in a manner similar to the insertion of endometrial biopsy forceps. The perineum of the mare should be cleansed and disinfected before insertion of the endoscope, which is passed using sterile gloves to protect the tip of the endoscope. Once the endoscope is in position, the air-insufflation channel can be used to inflate the uterus to allow the endometrial surface to be visualized. Alternatively, saline can be used to inflate the uterus because it is less irritating than air. Endoscopic examination is useful in identifying uterine adhesions, removal of uterine cysts by laser or snare, and the presence of angiosis.

EPIDURAL ANESTHESIA

Epidural anesthesia is simply performed and is of great assistance in correction of some dystocias and for perineal surgery.

KEY POINT

The technique must be undertaken with the mare standing evenly on both hindlegs.

If the mare is resting one of the hindlegs, there will be uneven analgesia because more of the local anesthetic will run to one side of the epidural space. After clipping and routine surgical disinfection, the space between the first and second coccygeal vertebrae is palpated by lifting the tail. This usually coincides with the level at which the coarse tail hairs begin. A bleb of local anesthetic (1 mL of 2% lidocaine or mepivacaine; Treatment Nos. 67 and 72) is injected subcutaneously. After sterile gloves are applied by the operator, an 18- or 19-gauge, 3.75-cm (1.5-inch) needle is inserted to a depth of 2.5 cm (1 inch) (Fig. 8-11). The amount of 2% lidocaine or mepivacaine needed for epidural anesthesia varies, but no more than 5 mL/500 kg should be used initially and the effects assessed. The needle is left in place, and after approximately 5 minutes, additional local anesthetic can be injected into the epidural space if required. More than 8 mL of local anesthetic should not be used because instability of the hindlegs can occur. More recently, xylazine (Treatment No. 109) has been used for epidural analgesia at a dose of 0.17 mg/kg (100 mg/450 kg) xylazine diluted in 10 mL saline. It has the advantage of a longer duration of action, and mares are not as incoordinated as when local anesthetics are used.

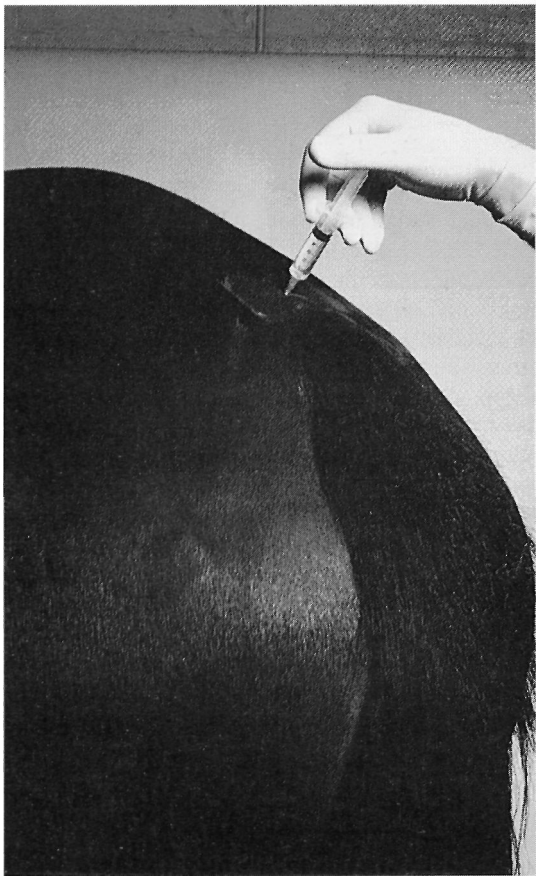


Figure 8-11. Epidural anesthesia. A needle (19-gauge, 1.5-inch, or 3.75-cm) is inserted into the epidural space between the last sacral and first coccygeal vertebrae. The space is located by moving the tail up and down and palpating for the space between the two vertebrae. After desensitizing the skin over the site with local anesthetic, the needle is inserted in the dorsal midline and directed in a slightly cranial direction into the epidural space. An initial volume of 1 mL/100 kg of 2% lidocaine or mepivicaine is injected slowly; there should be no resistance to injection. Alternatively, xylazine (100 mg/450 kg), diluted to a volume of 10 mL with saline, may be used.

However, it takes 45 minutes before anesthesia is attained. Therefore, it is recommended to administer xylazine with 5 mL of *2%* lidocaine instead of saline. Anesthesia is attained in 4 to 6 minutes and will last for 4 to 5 hours.

The Stallion

Examination of the genital system of the stallion is conducted to evaluate potential fertility or infertility and longevity of semen for shipment and its freezability. It is critical that the examination is thorough and results are properly recorded because findings are frequently used for sale purposes, for obtaining fertility insurance, or for making recommendations on breeding management.

KEY POINT

The examination should include evaluation of the external genitalia, libido, ability to mate, and collection of semen to determine sperm numbers, progressive motility, and morphology.

If pus, bacteria, or blood is present in the ejaculate or abnormalities such as azoospermia or cryptorchidism are noted, a rectal exam should be performed to assess the internal genitalia and inguinal rings.

GENERAL EXAMINATION

A history that details medical problems, injuries, vaccinations, and drug therapy the stallion has been receiving over the past 6 months needs to be taken. Previous breeding performance including first cycle conception rates, pregnancy rate for the year, libido, mating behavior, and any abnormalities encountered during breeding need to be recorded. The stallion must be positively identified, and a general physical examination should be performed at the initial evaluation. Mismanagement, inadequate nutrition, or a medical disorder may lead secondarily to deterioration in semen quality. The stallion should be observed moving freely in a small yard or at pasture so that any musculoskeletal disorders such as chronic degenerative joint problems, chronic laminitis, and back disorders or any ophthalmic abnormalities can be observed.

Examination of the Penis. Most stud farms wash the penis of the stallion before service, and this is a convenient time for examination.

KEY POINT

Use of disinfectants when washing the penis may favor the colonization of specific bacteria and lead to infection with bacteria such as Pseudomonas spp.

Use of promazine tranquilizers (e.g., acepromazine, promazine, chlorpromazine) should be avoided because of the risk of penile paralysis. In examination of the penis and testes, the veterinarian needs to move slowly and stay close to the stallion to avoid injury. A preferred technique is to approach the horse from the left just caudal to his shoulder, grasp the mane at the withers with

the left hand, and while keeping close contact with the stallion's body slide the right hand medially along the ventral abdominal wall until the penis is grasped proximal to the glans. The distal part of the penis, particularly the urethral process, can be examined for Habronema infection and evidence of trauma or neoplasia (e.g., squamous cell carcinoma). Examination further proximal allows assessment of scarring or injuries. Although injuries to the penis are not common, trauma can occur because of a kick from the mare or lacerations from barbed wire on fences.

Examination of the Scrotum, Testes, and Epididymides. Examination of the scrotum and testes is most easily performed after breeding. The scrotum itself is rarely affected by disease, and therefore examination is directed at the testes and epididymides. Both testes and attached epididymides should be freely movable within their respective scrotal pouches. There should be no free fluid within the scrotum (hydrocele) nor should there be any extraneous tissue within the sac (scrotal hernia). The testes and epididymides should be palpated carefully to assess tone and for abnormalities in size, consistency, or symmetry. Both testes should be of similar size and consistency, although it is common to find that one testicle is slightly smaller than the other.

KEY POINT

Stallions being examined for fertility potential should have their testes measured with either calipers (scrotal width) or ultrasonography (testes volume) because testicular size is related to daily sperm output.

Testicular width ranges in mature stallions from 9.5 to 12.5 cm. Although the head and body of the epididymides are often difficult to palpate, the tail of each epididymis is easily palpated at the caudal pole of the testicle. It is not uncommon for some stallions to have one of the testes rotated 180 degrees without the animal showing any pain or it having a negative effect on fertility.

Examination of Internal Genitalia. The accessory sex glands (vesicular glands, prostate, and bulbourethral glands) can be palpated during rectal examination at the level of the neck of the bladder. The glands are dynamic in nature in that they change shape and size due to sexual excitation. It can be difficult to accurately locate these structures by rectal palpation. If pathology is suspected, it is best to examine the glands using ultrasound via the rectum using a 7.5-MHz probe. Culture of the seminal fluids that pass through a catheter placed 15 to 20 cm into the urethra after the stallion has been exposed to a mare in estrus may be indicated.

BREEDING SOUNDNESS EXAMINATIONS

This examination is used to make recommendations on the number of mares that a stallion may mate successfully each season. Stallions are rated as unsatisfactory, questionable, or satisfactory breeders. Stallions that pass are expected to get 75% of 40 or more mares pregnant when bred naturally or 75% of 120 mares when bred artificially in one breeding season when given good management and fertile mares.

KEY POINT

The examination includes identification, physical examination, evaluation of genitalia, libido, mating ability, semen quality, and testes size.

Two semen samples are collected 1 hour apart after the stallion has had a week of sexual rest. If the two samples are representative, that is, the second ejaculate contains between 30 and 70% of the total sperm numbers of the first ejaculate, then the number of live, motile, normal sperm in the second ejaculate is a fairly accurate assessment of daily sperm output. The number of live, progressively motile, morphologically normal sperm needed for a stallion to be regarded as satisfactory ranges between 1.8 and 2.2 x 10^9 sperm, with higher numbers required in the summer.

Semen is collected by the stallion mounting a mare in estrus or a phantom. An artificial vagina, such as the Missouri model, is recommended. The sleeve of the artificial vagina is filled with warm water so that the temperature is between 42 and 45°C. The inside of the artificial vagina is lubricated before use with a nonspermicidal lubricant, and the collection bottle is warmed to body temperature and attached to the end of the device.

The stallion should be restrained from mounting the mare or phantom until full erection is attained. Once mounted, the penis is diverted to the side of the mare or phantom and into the artificial vagina. The artificial vagina is kept in place until the stallion dismounts. The water is then removed from the artificial vagina so that all the semen is collected in the collecting bottle. It is essential that the semen is kept warm and away from sunlight, air, or water. The semen should be stored at 37°C until it is assessed for concentration, motility, and morphology. The volume of the ejaculate also should be recorded.

Semen Evaluation

KEY POINT

Motility of raw and extended semen should be assessed within 5 minutes of collection. Five sperm per field are chosen and the number swimming across the slide represents progressive motility. A minimum of 10 fields should be counted.

Longevity of spermatozoal motility is determined on raw semen samples and on samples diluted in various extenders stored refrigerated (4-5°C) at 8, 12, 24, 36, and 48 hours after collection. Sperm morphology is evaluated using either stained smears or wet mounts using phase contrast microscopy. A minimum of 200 sperm should be evaluated. Normal sperm and those with acrosomal, head, midpiece, droplets, and tail defects need to be recorded. Sperm concentration can be measured with a hemacytometer or densimeter that is appropriately calibrated. A technique for measuring concentration with a hemocytometer is to add 50 |xL of raw semen to 5 mL of formal buffered saline and mix well. One drop of the solution is placed on the grid of the chamber and a coverslip applied. The fluid is allowed to settle for 3 to 5 minutes. The heads of the sperm in either five small squares in the large central squares of the hemacytometer or in all 25 small squares are counted. To determine the number of sperm per milliliter of ejaculate, the number of sperm counted is multiplied by five million if only 5 squares are counted and by one million if all 25 squares are counted.

BACTERIOLOGIC CULTURE

If swabs are taken for suspected infections of the penis and prepuce, it must be remembered that bacteria (including organisms such as *Pseudomonas* spp., *Escherichia coli, Staphylococcus* spp., and *Streptococcus* spp.) are normally isolated from healthy stallions. However, if a single organism is isolated together with inflammatory cells, pus, or blood in the semen, there may be an infection. The only organism that can be universally regarded as a significant pathogen is *Taylorella equigentalis*, the organism responsible for CEM. This is a microaerophilic organism that requires special culture conditions, and swabs should be handled appropriately (see Chapter 17). Swabs for bacteriologic examination should be collected routinely from the urethra before and after ejaculation and from the semen sample as part of the breeding soundness examination.

ASPECTS OF MARE AND STALLION MANAGEMENT AND REPRODUCTIVE PHYSIOLOGY

Artificial Lighting

It has been well established that in both the northern and southern hemispheres, the breeding season is not coincident with optimal ovarian activity of the mare. This is so because mares are long-day breeders with endocrinologic changes determined by day length. The peak of ovarian activity takes place late in the breeding season, and in the early part of the season, many mares are still in anestrus. During the early season, many mares also may be in a transition period in which there may be follicular activity but not ovulation because of low circulating concentrations of luteinizing hormone (LH). For this reason, various strategies have been tried to induce regular estrus activity in the early part of the breeding season.

KEY POINT

Artificial lighting is widely used on stud farms throughout the world to induce earlier onset of regular ovarian activity.

The pineal gland has been shown to be actively involved in the mechanism of action of photoperiod, although the exact mechanisms are still not clear. From a practical point of view, the following points are important. The light wavelength and intensity are critical, as is the duration of exposure. Although the exact wavelength and intensity of light to produce an effect are not known, either incandescent or fluorescent light bulbs, with an intensity of light greater than 10 footcandles, are satisfactory. Although this is usually done indoors, outdoor lighting also has been shown to be effective. This technique is practiced widely in the southern hemisphere. Studies from the University of Florida have shown that extension of day length by 2 to 3 hours is effective in advancing the breeding period. Artificial light needs to be added in the evening. Most stud farms use 16 hours of lighting a day in the late winter months (December 1 to March 1 or June 1 to September 1 in the southern hemisphere) to advance the onset of the breeding season.

KEY POINT

Mares have three to four waves of large follicles developing during a 60- to 90-day transitional period before they ovulate for the first time of the year. One cannot determine by

rectal palpation which of the follicles will ovulate.

Only the dominant follicle from the fourth follicular wave ovulates because it is the first follicle of the year that has the endocrinologic ability to do so. It is during development of this follicle that mares exhibit uterine edema. There are currently no drugs available commercially that effectively stimulate follicles to ovulate during the first three waves.

Endocrinology of the Estrous Cycle

- Knowledge of the hormonal events of the estrous cycle are of major importance because endocrine therapy is required in some reproductive disorders. As detailed earlier, increasing day length is a key trigger to the release of adequate quantities of gonadotropin-releasing hormone, which is a prerequisite for LH release and ovulation.
- Follicle-stimulating hormone (FSH) is released from the anterior pituitary. FSH results in development of ovarian follicles, which secrete estrogen. Estrogen release is responsible for the behavioral changes during estrus and the physical changes involving the vagina and cervix that are seen via a speculum examination.
- The high levels of estrogen act via a feedback effect to stimulate LH release from the anterior pituitary. The result of this is ovulation. Ovulation occurs approximately 24 hours before the end of estrus.
- After ovulation, the corpus luteum forms and produces progesterone. The progesterone level is maintained past day 18 if the mare becomes pregnant. Otherwise, prostaglandin release terminates diestrus by causing luteolysis, and the mare returns to estrus 17 to 19 days after ovulation. The result is that the blood levels of progesterone decrease to zero.
- With the abrupt decrease in progesterone, the anterior pituitary secretes further FSH, and the pattern of hormonal cyclical activity recommences.

Important Facts about the Estrous Cycle

- Length of diestrus is predictable in cycling mares (14-15 days). Length of estrus is not and ranges from 2 to 10 days.
- Diestrous ovulation is common in mares. These are potentially fertile ovulations. They also contribute to prolonged diestrus, a common condition in mares.
- Multiple ovulations are common; twins are not.
- Sperm survive in the mare for 48 hours or more.
- The ovum is fertile for only a few hours after ovulation (about 8-12 hours).
- The fertilized egg arrives in the uterine lumen in 5.5 days. Unfertilized eggs remain in the oviduct.
- Corpora lutea are responsive to prostaglandin greater than 5 days of age.
- Corpora lutea cannot be palpated rectally but can be visualized by ultrasonography.
- Although the description given is highly simplified, it is clear that a variety of hormonal aberrations can give rise to certain clinical problems. Prolonged diestrus may be the result of a persistent corpus luteum.
- Prostaglandin therapy (Treatment No. 53) may be indicated in such cases. Some mares demonstrate prolonged estrus possibly due to an apparent lack of feedback of estrogen to cause LH release. The use of human chorionic gonadotropin (hCG; Treatment No. 25) is sometimes effective in inducing ovulation in these circumstances.

Pregnancy Diagnosis

Most stud-farm managers will maintain records of service dates and number of services and indicate when estrus ceased, denoted by a lack of response to teasing. Although ultrasound examination has replaced the manual detection of pregnancy on larger stud farms, an experienced veterinarian can detect pregnancy via a rectal examination after 20 to 25 days. However, it is possible to be most accurate with rectal palpation after 30 days of gestation. Details regarding care that is necessary when performing a rectal examination are outlined earlier in this chapter. The earliest sign of pregnancy is the appearance of increased cervical and uterine tone. The diestrus uterus has little tone, and the presence of moderately increased tone is one indicator of pregnancy. At around 30 days, the characteristic enlargement or bulge in one of the uterine horns can be palpated. This enlargement continues so that at 42 days, when many mares are certified as being pregnant or nonpregnant, there may be some dorsal enlargement of the horn; the disparity in size between the pregnant and nonpregnant horns is quite apparent.

Ultrasound examination has permitted earlier establishment of a diagnosis of pregnancy. However, because of early embryonic loss, many mares that are pregnant at 20 days gestation may not be pregnant at 42 days. For this reason, repeat examinations are required to establish normal growth of the embryonic vesicle. If twin concepti are diagnosed, it is possible to manually crush one

of the vesicles between days 14 to 25 of gestation. This can be followed up with further ultrasound examinations to confirm the viability of the remaining embryonic vesicle.

In some smaller pony mares, rectal or ultrasound examination may not be possible. In these cases, tests to determine the presence of eCG may be used to establish the diagnosis of pregnancy. The simplest and most readily available of these tests is the mare immunologic pregnancy test, which is based on hemagglutination inhibition. The test is quite accurate between 40 and 120 days, but the main problem is false-positive results. These can occur as a result of early embryonic death, in which eCG levels can still be elevated despite death of the fetus.

Induction of Parturition

INDICATIONS

Foaling induction should be approached with great caution because of many possible complications. The most obvious problem is induction of the foal before full term. Rossdale and Ricketts (1980) also showed that although some pregnancies may be at full term in gestational length, the foal may still not be ready to be born. In such cases, induction of parturition at 340 days, for example, may lead to a foal that is not physiologically prepared for birth and therefore may suffer adaptation problems. The normal gestational length in the mare is an average of 340 days and ranges from 320 to 360 days. In pony mares, the gestational length is shorter.

KEY POINT

Mares that are candidates for induction are those that have experienced previous foaling problems or foaling injuries or those with injuries, increased gestational length, or medical problems. If gestational length is known accurately, it is preferable that induction be delayed until the mare is at least 335 days.

COLOSTRAL ELECTROLYTE CONCENTRATIONS

If an accurate service date is not available, some reliance can be placed on udder development and mammary secretion electrolyte concentrations to assess the proximity to foaling. In mares with known and unknown service dates, precolostral and colostral electrolyte concentrations are valuable in predicting if foaling is imminent.

KEY POINT

Colostral electrolyte concentrations are an accurate indicator that the foal is ready for birth, and we have found them to be extremely useful in making a decision for or against induction of parturition.

The mammary secretions in the weeks before foaling have concentrations of sodium and potassium similar to those of serum or plasma. The sodium values are usually in the range of 125 to 135 mmol/L (mEq/L) and potassium 7 to 12 mmol/L (mEq/L). Calcium values remain low and are usually less than 2 mmol/L (8 mg/dL) until the mare is within 12 to 24 hours of foaling. Sodium values in the mammary secretions decrease to less than 30 mmol/L and potassium concentrations increase to greater than 30 mmol/L within 24 to 48 hours before foaling. Calcium values increase more precipitously in the 24 hours before foaling, and values greater than 10 mmol/L (40 mg/dL) indicate that foaling is imminent. Therefore, if there is udder enlargement and evidence of "waxing" of the teats, the mare should have a sample of mammary secretion submitted for electrolyte analysis before a decision is made for or against induction. A number of commercial kits are available that assess calcium concentration in mammary secretions by a color change.

KEY POINT

Although it is useful to examine the cervix using a speculum, we have found that the cervical changes are not of great use in indicating whether induction should take place. A decision for induction should therefore be made on the basis of changes in the appearance of the udder and electrolyte alterations in the mammary secretions.

Either oxytocin or prostaglandin analogues, such as fenprostalene or prostalene, can be used to induce parturition. We prefer the use of oxytocin (Treatment No. 83) because most mares will deliver their foal within 45 minutes of drug administration, whereas mares take an average of 4 hours to deliver after prostaglandin treatment. Because most mares are induced to allow veterinary assistance, it is easiest to give the injection and remain on the farm while the mare delivers. Although large doses of oxytocin (>100 IU) have been recommended, we have found these to produce adverse reactions, including signs of colic and excessive straining.

KEY POINT

For induction, doses of oxytocin should not exceed 10 to 20 IU given intravenously every 30 minutes, and the total dose should not be greater than 60 IU.

In most cases, only one to two doses are necessary before the onset of stage II labor, and the amnion is presented in most cases within 20 to 40 minutes of the initial oxytocin injection. We have found this regimen to give reliable results, and viable full-term foals have been delivered successfully.

Artificial Insemination with Fresh Cooled or Frozen Semen

Although the Jockey Club does not permit the use of artificial insemination, it is possible in most other breeds, including Standardbreds. Almost all breed societies that permit artificial insemination also allow mares to be bred with cooled transported semen. Greater numbers are using frozen semen. The greatest advantages of these technologies is that the stallion can be used more efficiently, with more mares being mated each breeding season, and there is a decreased risk of disease. Breeding mares with cooled shipped semen or frozen-thawed semen increases the genetic pool and enables mares to be bred to stallions throughout the world while the mares remain at home with their foals.

For artificial insemination, most stud farms use Kenney's milk-based semen extender: powdered milk, 2.4 g; glucose, 4.9 g; sodium bicarbonate, 2 mL of 7.5% solution; gentamicin sulfate, 100 mg; and distilled water, added to make up a volume of 100 mL. Milk-based extenders that differ only in the antibiotic(s) used are commercially available from many companies. Amikacin, gentamicin, or potassium penicillin have the least adverse effects on sperm motility. The extender should be kept in a water bath or incubator at a temperature of 37°C until it is added to the semen that is to be shipped. Semen and extender are diluted at a ratio of 4:1 or diluted to a final concentration of 20 to 50 million sperm/mL. Inadequate dilution results in poor sperm motility after storage in a cooling tank for 24 to 48 hours. Longevity studies of semen should be conducted before its shipment, because only 50 to 60% of stallions have semen that tolerates cooling and storage. The maximum storage time is determined by progressive motility. First cycle conception rates are highest if mares are bred using semen with greater than 30% progressively motile sperm.

If the mares are to be bred on the farm where the stallion resides they should be inseminated with a minimum of 250 million normal motile sperm every second day during estrus until ovulation occurs. The alternative is to base insemination on findings from ovarian palpation. Mares should be inseminated within 24 to 36 hours of ovulation.

KEY POINT

To breed mares successfully with shipped cooled semen, there must be communication and cooperation between the stallion manager, mare owner, and veterinarians involved. Shipment of semen must be coordinated so that the mare is bred within 24 hours of ovulation.

First-cycle conception rates tend to be lower with shipped semen than with natural breeding or with artificial insemination when the stallion is housed at the same facility; however, breeding management needs to be more intensive, and veterinary costs are higher. The mare's reproductive tract should be examined every 24 hours after the second day of estrus. When the dominant follicle is 35 mm in diameter, semen should be ordered to arrive in the next 24 hours. A hormone to induce ovulation (hCG 2000 IU or desorelin implant) should be given when the order is placed because both drugs will induce ovulation within 36 to 48 hours of administration. The operator must identify that the semen is from the correct stallion for the mare involved when the semen tank arrives. All but 1 to 2 mL of semen is then drawn up into a syringe and the mare bred directly without warming the semen. Progressive motility is determined from the remaining sample after it is warmed at 37°C for 2 to 5 minutes before its assessment. Progressive motility should be more than 30%.

First-cycle conception rates are low with frozen semen. They range from 0% for some stallions to 60% with an average of 35% for most stallions. Freezing of sperm causes characteristic changes in the sperm cell membrane that interfere with sperm longevity and viability. Therefore, mares need to be bred close to ovulation. First-cycle conception rates are highest when the mares are examined every 6 to 8 hours and bred within 8 hours either side of ovulation. A management technique that we use is to palpate mares once daily during estrus until the dominant follicle is 35 to 37 mm in diameter. An ovulatory drug is given at that time. Mares are then palpated every 12 hours until 36 hours after drug administration. The mare is then bred with frozen-thawed semen. Rectal palpation and ultrasonography is performed every 8 hours

until ovulation. If the mare ovulates more than 8 to 12 hours after insemination, she is rebred.

KEY POINT

Some mares exhibit acute persistent inflammation in response to breeding with frozen-thawed semen and accumulate large amounts of fluid in their uterus.

Uterine lavage and oxytocin or cloprostenol 12 hours after breeding may alleviate the inflammatory response.

Ultrasonographic examination of the uterus can be used to time ovulation. During estrus, endometrial folds are visible on the ultrasound screen and appear as a "cartwheel, bicycle spokes, or tiger stripes." Twenty-four to 48 hours before ovulation, estrogen begins to decline, causing the image of the cartwheel to break up. Six to 12 hours before ovulation, the dominant follicle changes from a spherical to a "pear" shape. This is when most mares are bred if ultrasonography is being used to determine ovulation. Immediately after ovulation, the uterus has a homogenous gray appearance. The developing corpus luteum will either have a hyperechoic white rim with white strains through the space where the dominant follicle was or the space will be completely white as the luteal tissue takes the place of the blood clot.

Reproductive Disease in the Mare

Abortion

There are a wide range of causes for abortion in the mare, but in general there is a higher incidence in old mares. Bacterial endometritis or ascending placentitis may result in abortion. Persistent inflammation in combination with aging may produce chronic fibrosis, which subsequently will affect a mare's ability to carry a foal until term. Other causes of abortion include viral or fungal infections, twinning, and medical or surgical crises during pregnancy.

KEY POINT

The most common viral cause of abortion is equine herpesvirus 1 (EHV-1) infection, which generally causes abortion in the last trimester of pregnancy.

The other major viral infection that can cause abortion is equine viral arteritis.

There is considerable controversy as to whether endocrine factors play a role in abortion. In particular, debate has raged over the validity of exogenous progesterone administration.

KEY POINT

Most studies have not shown any beneficial effects of progesterone therapy in a mare with a history of an inability to carry the foal to term.

HISTORY AND PRESENTING SIGNS

- Return of the mare to estrus
- Discharge from the vulva
- Fetus found
- Retention of fetal membranes
- Dripping milk prematurely

CLINICAL FINDINGS AND DIAGNOSIS

- If the abortion occurs in the early stages of pregnancy, the only sign can be that the mare returns to estrus. In the later stages, a fetus may be found, and on occasions, the placenta may be retained.
- If the fetus is found, a necropsy should be performed, and samples collected for microbiology.
- Early embryonic loss after establishment of the endometrial cups may not be recognized for some time because the high eCG concentrations prevent a return to estrus.

DIFFERENTIAL DIAGNOSIS

- Bacterial abortion, caused by *Streptococcus, E. coli, Salmonella, Klebsiella, Actinobacillus,* and others
- Twinning
- Endometrial fibrosis
- Nutritional deficiencies, including malnutrition
- Viral abortion, caused by EHV-1, arteritis virus, and infectious anemia
- Fungal abortion, caused by *Aspergillus fumigatus* and other species
- Cervical tears
- Colic (endotoxemia)
- Surgery with mare in dorsal recumbancy for greater than 90 minutes during the last 3 months of gestation

TREATMENT

- If placentitis is the cause of the abortion, the uterus should be lavaged with saline and the

mare placed on systemic antibiotics (after results of culture and sensitivity tests).
- If inadequate progesterone levels are thought to be the cause of previous abortion, progesterone therapy may be given. Although no research work has been able to show a beneficial effect of progesterone, there is anecdotal evidence for its use. Progesterone therapy is beneficial in preventing abortion in mares that release prostaglandin systemically as a result of colic or endotoxemia. To maintain effective blood concentrations (>1 ng/mL plasma), progesterone in oil can be administered at a dose rate of 300 mg daily or oral allyl trenbelone (Treatment No. 3) can be given daily at a double dose rate of 44 mg/500 kg.

Cervical Problems

The cervix is the third physical barrier against external contamination of the uterus, after the labia and the constrictor muscles of the vaginal vestibule. It is a dynamic structure that must relax during estrus to allow semen in and contaminants to drain out and must close tightly during diestrus and pregnancy.

KEY POINT

Cervical defects, adhesions, tears, and incompetence will result in either endometritis or abortion. The cervix needs to be examined manually during diestrus to evaluate defects affecting its ability to close and during estrus if a maiden mare does not conceive after repeated breedings.

HISTORY AND PRESENTING SIGNS

- Fluid accumulation in uterus after breeding in maiden mare
- Infertility
- Abortion at 50 to 150 days
- Previous dystocia
- Discharge from the vulva
- Bacteria isolated on uterine or cervical swab

CLINICAL FINDINGS AND DIAGNOSIS

- Inadequate cervical dilation during estrus results in infertility. It occurs most commonly in young maiden mares or old maiden mares (>14 years of age) retired from performance careers. Two fingers should fit through the cervix during estrus.
- Two types of cervical lesions may occur after dystocia: laceration of the external os and a portion of the cervical canal or excessive stretching of muscle surrounding the canal. Mares with the latter lesion tend to abort after 5 to 7 months. The cervix should be palpated during diestrus to identify lesions. The cervical canal should be tightly closed, making it difficult to pass a finger through it. The rim of the external os should be about 1 cm thick.
- Intrauterine infusion of irritating compounds will adversely affect the cervix. On vaginal examination the cervix will be inflammed, hyperemic, and edematous. It may be ulcerated.

DIFFERENTIAL DIAGNOSIS

- Endometritis due to delay in uterine clearance
- Abortion due to placentitis, glandular fibrosis

TREATMENT

- Lacerations of the external os that include more than 25% of its circumference need to be repaired surgically. If the muscularis and the cervical mucosal layer are disrupted, the long-term breeding prognosis is guarded. The cervix usually tears again at foaling.
- If the cervix does not dilate at breeding, the mare may conceive if bred by artificial insemination. She can be given either oxytocin 10 to 20 IU IV or cloprostenol 250 μcg IM 4 to 8 hours after breeding to physically clear her uterus of inflammatory byproducts.
- Cervical tears identified at foaling should be treated daily with an antibiotic cream for 5 to 7 days to prevent cervical adhesions from forming.

Contagious Equine Metritis

Contagious equine metritis (CEM) was first recognized in Ireland and England during the 1977 breeding season.

KEY POINT

It is a highly contagious venereally transmitted disease that produces a copious mucopurulent vulvar discharge in infected mares.

The disease subsequently spread throughout the world. The organism involved was found to require microaerophilic culture conditions, which is why normal culture methods were not satisfactory. Subsequent work established the organism to be a new bacterium, which was named *T. equigenitalis.*

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HISTORY AND PRESENTING SIGNS

- Copious discharge from the vulva
- Infertility

CLINICAL FINDINGS AND DIAGNOSIS

- The signs are those of an acute endometritis, there being a profuse mucopurulent discharge from the vulva of the mare. However, some mares show no signs of the infection while carrying the disease.
- In most mares with CEM, the discharge ceases within 5 to 7 days whether treatment is given or not. Mares are asymptomatic carriers for long periods after infection.
- Although the stallion carries the organism, it usually produces no pathogenic effects.
- Diagnosis is by bacteriologic culture, and special chocolate agar media and microaerophilic culture techniques have to be used.

KEY POINT

Bacterial swabs must be taken from the clitoral fossa and sinuses to check for the presence of the CEM organism.

- The clitoral fossa seems to be a favorite site for localization of the organism, which can often be recovered even in pregnant mares.

DIFFERENTIAL DIAGNOSIS

- Bacterial endometritis
- Vaginal injuries

TREATMENT

- Treatment of mares infected with the CEM organism does not appear to be effective. The acute endometritis seems to resolve itself with or without antimicrobial therapy. About 20% of infected mares remain asymptomatic carriers regardless of treatment.
- In stallions, washing the penis with chlorhexidine (see Disinfectants, Chapter 17; Treatment No. 23) is quite successful in eliminating the organism. Care must be taken because overgrowth of other bacteria can occur.

Dystocia

Second-stage labor in the mare is an explosive event and is completed quickly, usually in 15 to 20 minutes. Because of this, unlike the situation in the cow, time to correct dystocias (difficult births) is limited. During first-stage labor, the foal moves into anterior and dorsosacral presentation. When labor is prolonged or there is a lack of progress of first- or second-stage labor, dystocia should be suspected. Early examination is essential if a live foal is to be delivered.

HISTORY AND PRESENTING SIGNS

- Prolonged discomfort and sweating
- Straining without appearance of the amnion
- Appearance of the amnion or a limb or head without further progress

CLINICAL FINDINGS AND DIAGNOSIS

- When there is lack of progress of delivery, careful examination of the position of the foal is essential. In some mares, this can be performed with minimal restraint, whereas in others, the use of minimum doses (0.2-0.4 mg/kg) of xylazine (Treatment No. 109) in combination with butorphanol (0.05-0.1 mg/kg; Treatment No. 15) may be used. Xylazine or detomodine should never be used alone when working around the back end of a mare because some become hypersensitive through their hindquarters and will kick out viciously. If initial examination indicates that extensive manipulation is required or the mare is fractious, general anesthesia may be necessary.
- The mare's perineal region should be washed thoroughly with soap and the vulval lips dried. After good lubrication of the arms of the operator, the position of the foal should be assessed and decisions made concerning attempted correction of the dystocia.

DIFFERENTIAL DIAGNOSIS

- Malposition of the limbs or head
- Incorrect presentation
- Oversize foal, dead foal
- Twins
- Premature placental separation
- Large colon torsion

TREATMENT

Correction of Presentation, Position, or Postural Abnormalities

KEY POINT

Most commonly only one forelimb is retained. If both front forelimbs are retained, the foal may have contracted tendons. Weak foals or dead foals may have the head retained in a

"poll" presentation (Fig. 8-12). Always correct the malaligned head before correction of malpositioned forelimbs.

Correction of a dystocia is difficult. One must make a plan on how the foal will be manipulated. If the dystocia cannot be corrected within 30 minutes, the situation should be reassessed. Anesthesia or a cesarean section may be required. Prolonged manipulation frequently results in metritis and laminitis. If a forelimb is retained, an obstetric chain or soft rope should be placed below the feltlock and gentle traction by one person should be placed on it. The foal may need to be repelled back into the uterus to manipulate the forelimb. If the head is retained, a snare or halter needs to be placed around the foal's ears and through its mouth and the head gently rotated back into the birth canal. Rarely are foals oversized; however, if both shoulders cannot be delivered through the vulva, it should be suspected. If delivery of the foal stops suddenly after the forelimbs, head, and chest are present outside the vulva, it may be a "dog sitter," a foal whose hind hooves have become caught on the pelvic brim because the limbs were folded under the pelvis. These foals are rarely delivered alive (Fig. 8-13).

Fetotomy. If the foal is dead and the malposition cannot be corrected, a fetotomy may be performed.

KEY POINT

There must be ample room in the birth canal and the cervix effaced completely to perform a fetotomy. The operator should never perform more than three fetotomy cuts if the mare is to remain a broodmare.

The vaginal mucosa swells rapidly if obstetric manipulations are prolonged (>30 minutes).

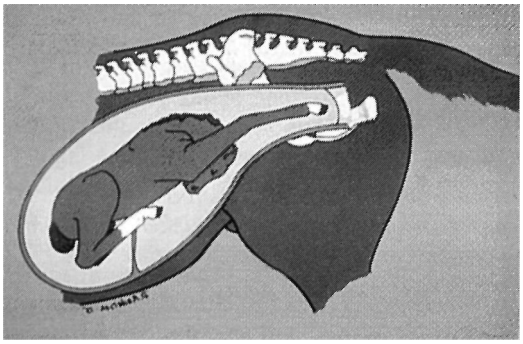


Figure 8-12. Poll presentation. Dead or weak foals may present in this fashion.

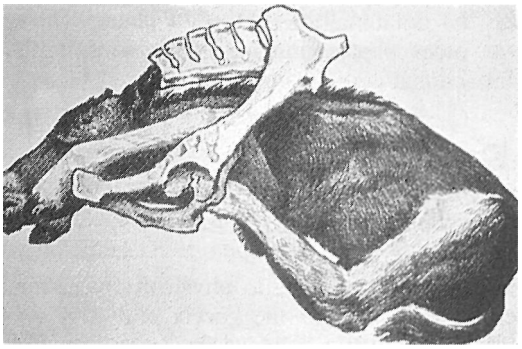


Figure 8-13. "Dog sitter" presentation of foal presentation.

These mares that have undergone prolonged manipulations do not make good candidates for fetotomy because the vaginal mucosa may be further damaged during the procedure, resulting in necrotic vaginitis. It is best to perform the fetotomy with the mare standing, if possible, while tranquilized. An epidural should be performed. It is essential that the fetotome be placed in the uterus and that the cervix is not cut inadvertently.

Cesarean Section. Cesarean section is required if the foal is alive and cannot be delivered in 20 minutes, if the uterus is torsed at term, or the mare has previous pelvic injuries. Surgery is best performed under general anesthesia, preferably in a surgical facility. A number of surgical sites have been used. A midline laparotomy provides the best exposure; however, uterine contents tend to be spilled in the abdomen and it is sometimes difficult to lift the foal out of the uterus. The flank approach enables the foal to be extracted more easily but there tend to be more complications with incisional healing.

Endometritis

Endometritis, or infection of the endometrium, is the most common disease necessitating treatment in the mare.

KEY POINT

Many factors predispose mares to infection, including genetics, parity, and perineal conformation.

Endometritis is categorized into four groups based on etiology and pathophysiology:

1. Persistent mating-induced endometritis. Semen induces a physiologic and transient inflammation in the uterus. Reproductively normal mares

clear the inflammation within 24 hours, whereas some older pluriparous mares or mares with a dysfunctional cervix cannot.

KEY POINT

Mares that accumulate fluid within the uterine lumen after breeding are infertile.

These mares are unable to physically clear intrauterine fluid through the cervix and may have impaired lymphatic drainage and excessive endometrial glandular secretions secondary to chronic inflammation. Excessive fluid within the uterine lumen after day 5 is detrimental to embryo survival.

2. Chronic infectious endometritis may be a sequela to persistent mating-induced endometritis. It also occurs when the mare's physical barriers to contamination, the vulva, the vestibular vaginal spinctor, or the cervix are compromised. The major pathogens involved include *Streptococcus equi* var. *zooepidemicus, E. coli, Pseudomonas, Klebsiella, Staphylococcus* spp., yeast, and fungi. The reason why infection becomes established is critical to the outcome of treatment, because treatment with antibiotics alone will not resolve the underlying problem.

3. Sexually transmitted disease (see Contagious Equine Metritis).

4. Chronic degenerative endometritis, commonly called periglandular fibrosis, is a byproduct of repeated pregnancies and uterine aging. Repeated stretching of the blood vessels in the broad ligaments and uterus results in degeneration of arterial and venous vessel walls (angiosis). Fibrosis may occur secondary to these abnormalities. Mares with periglandular fibrosis that conceive and carry the fetus past 7 to 8 months have hypoplastic microcotyledons and small fetuses for gestational age.

HISTORY AND PRESENTING SIGNS

Persistent Mating-induced Endometritis

- Most common in pluriparous mares greater than 14 years of age; may also occur in mares with cervical incompetence
- Failure to conceive
- Early embryonic loss

Chronic Infectious Endometritis

- All ages, but most common in older pluriparous mares with poor perineal conformation
- Secondary to overuse or misuse of antibiotics
- Mucopurulent discharge
- Short estrous cycles (<14 days)
- Failure to conceive

Chronic Degenerative Endometritis (Periglandular Fibrosis)

- Older pluriparous mares and older nulliparous performance mares
- Repeated early embryonic loss
- No vulval discharge
- Delivery of small thin foals
- Prolonged gestation

CLINICAL FINDINGS AND DIAGNOSIS

Persistent Mating-induced Endometritis

- The most common clinical finding is fluid accumulation in the uterus visualized on the ultrasound exam 24 to 72 hours after breeding.
- Usually no bacteria are isolated on uterine culture when performed early in the breeding season. If the mare has been bred repeatedly, bacteria may be recovered. Five or more neutrophils per field may be observed on cytology smears.
- If the mare has conceived, the embryo may appear abnormally shaped and may be lying in fluid on the 14- to 16-day pregnancy examination.

Chronic Infectious Endometritis

- There may or may not be signs of vulval discharge and, in some cases, failure to conceive or maintain pregnancy may be the only signs.
- Definitive diagnosis is made by collecting swabs for bacteriology from the cervix or endometrium. The use of guarded swabs limits the possibility of contamination from other sites. It is important to interpret bacteriologic findings in the light of cytology (see Chapter 17) to ensure that a clinically significant infection is present.
- Vaginal mucosa and cervix may be hyperemic. There may be a purulent discharge or urine pooling in the anterior vagina.
- Biopsy scores may be a Kenney II or Kenney III, depending on the severity of the insult and length of time the uterus has been infected.

TREATMENT

KEY POINT

Anatomic abnormalities such as pneumovagina, fistulas, or sunken perineum must be corrected surgically before any treatment.

Persistent Mating-induced Endometritis

- Mares should be bred only once each estrus during the 48 hours before ovulation. Treatment

is directed at rapid removal of intraluminal fluids 4 to 12 hours after breeding and administration of drugs that increase uterine contractions. The uterus can be lavaged with 1 to 3 L of saline, followed by 10 to 20 IU of oxytocin given either intravenously or intramuscularly. Prostaglandins may be helpful in mares with lymphangectasia (pooling of lymph within the uterine wall) because they cause the uterus to contract for 5 hours. Cloprostenol at a dose of 250 μg intramuscularly causes prolonged contractions and evacuation of intrauterine fluid. Treatment can be repeated 24 hours after breeding if fluid is visualized by ultrasonography.

Chronic Infectious Endometritis

KEY POINT

Intrauterine therapy is the technique used most consistently, although systemic antibiotic treatment is worthwhile. Misuse of antibiotics results in fungal and yeast infections.

- The antimicrobial agent selected depends on the sensitivity pattern of the bacteria that were isolated.
- Antimicrobials diluted in a volume of 60 to 150 mL of saline can be infused into the uterus with a disposable plastic uterine pipette.
- The usual dose of antibiotic per treatment is as follows: K penicillin, 3 g (5 million units); Na ampicillin, 3 g; ticarcillin, 6 g; chloramphenicol, 3 g; gentamicin sulfate, 3 g; Neomycin, 2 g; amikacin, 2 g. All aminoglycosides need to be buffered with either sodium bicarbonate or diluted in at least 150 mL of saline because they are extremely irritating to the endometrium.
- Some practitioners who report good results use weak antiseptic solutions (notably povidone-iodine) in a saline infusion. These agents are nonspecific and can be irritating, especially to the vaginal and cervical mucosa.
- The antiseptic of choice is povidone-iodine (Treatment No. 93), diluted 1:100. This infusion is repeated daily for 3 to 5 days, and after being left in place for 3 to 5 minutes, it should be drained and a saline solution used to decrease the irritant effects of the iodine.
- Fungal endometritis must be treated for a minimum of 10 days either locally or systemically for treatment to be effective. Rest from uterine manipulations appears to be as beneficial as drugs in many cases.
- In cases of chronic endometritis with evidence of glandular atrophy and fibrosis, there have been reports of the beneficial effects of uterine currettage.

Chronic Degenerative Endometritis

- There is no effective treatment.

Metritis-Laminitis Syndrome

If there is retention of the placenta or other factors leading to infection postfoaling, metritis may occur, followed by septicemia and laminitis due to endotoxemia. It should be considered a medical emergency.

KEY POINT

Metritis-laminitis is a common sequela to retention of fetal membranes.

HISTORY AND PRESENTING SIGNS

- Obstetric interference during delivery
- Retention of the placenta
- Vulval discharge
- Signs of depression, anorexia, pain
- Stall bound because of sick foal

CLINICAL FINDINGS AND DIAGNOSIS

- Signs of ill health may be noted as early as 12 to 24 hours after foaling.
- There may be a chocolate-colored fetid discharge from the vulva, and the mare will show signs of depression and pain.
- Heart and respiratory rates will be elevated. Temperature varies.
- Mare may show signs of low grade colic.
- Signs of laminitis may be found (see Chapter 4).
- Diagnosis is possible by a combination of history and clinical signs.
- Bacteriologic swabs from the uterus are useful to enable correct antibiotic selection.
- Complete blood count findings include white blood cell (WBC) counts less than 5000 X 10^6/L, neutropenia, and a left shift.
- Fibrinogen concentration can be as high as 5 to 10 g/L (500-1000 mg/dL).

DIFFERENTIAL DIAGNOSIS

- Colic due to various causes (e.g., large colon rupture, necrotic small colon)
- Uterine tear
- Peritonitis
- Pleuritis

TREATMENT

KEY POINT

Aggressive therapy is required. Treatment is aimed at decreasing bacterial growth and

eliminating toxins by removing fluid that has accumulated in uterus.

- Any mare with retained placenta or signs of metritis should be treated with systemic antibiotics, large volume uterine lavage, and a nonsteroidal antiinflammatory drug. Intravenous fluid therapy should be included if the mare is moderately to severely endotoxic.
- The uterus may be lavaged with warm salt water, using 6 oz of salt to 12 L of water or saline via a large-bore stomach tube. Before lavage, a rectal examination should be performed to assess fluid accumulation. Rectal examination should be repeated after the uterus is lavaged with 12 to 36 L of warm salt water to assess involution. If the uterus feels like a large flaccid sac, lavage should be performed twice a day; if it feels thick and corregated, uterine lavage can be performed once daily until the uterus is free from infection based on culture and cytology (<5 neutrophils/field) test results.
- If sensitivity results are not available, then the use of systemic penicillin and gentamicin for up to 5 days are probably the best choices.
- *Bacteroides fragilis,* an anaerobe that is not sensitive to penicillin, is often present. Metronidazole (Treatment No. 75) can be administered orally at a dose rate of 15 mg/kg every 6 hours.
- Flunixin meglumine (Treatment No. 52) is administered at a dose rate of 0.2 mg/kg to aid in preventing laminitis and controlling signs of endotoxemia.
- Mares that are showing clinical signs of endotoxemia should be treated with fluids intravenously. Flow rate depends on severity of condition. Spiking the fluids with 23% calcium gluconate at the rate of 125 mL/5 L appears to assist uterine involution. Oxytocin may be added to the fluids at a dose of 40 to 60 IU/5 L.
- If there are signs of laminitis, a range of treatment options is available, depending on the duration and severity of signs.

Ovarian Disorders

Ovarian abnormalities are usually found on rectal and/or ultrasound examination. The history is quite variable depending on the hormonal alterations induced by the ovarian disease. The major ovarian disorders include

- Tumors, most commonly granulosa cell tumors
- Gonadal dysgenesis (e.g., chromosomal abnormalities, particularly XO genotype)
- Persistent corpus luteum, resulting in prolonged diestrus
- Hemorrhagic follicles, causing failure of ovulation during the ovulatory season
- Ovarian atrophy (i.e., decrease or absence of ovarian activity in older mares)
- Ovarian hematoma. Hemorrhage can occur during ovulation and may result in quite dramatic ovarian enlargement.
- Large follicles with mares showing persistent estrus during transitional season
- Mares in persistent anestrus (i.e., no signs of estrus behavior during breeding season)

HISTORY AND PRESENTING SIGNS

- Prolonged diestrus
- Persistent estrus
- Changes in behavior—aggression, nymphomania
- Anestrus

CLINICAL FINDINGS AND DIAGNOSIS

- Mares with granulosa cell tumors (GCT) present because of changes in behavior, with anestrus or persistent estrous behavior most common. About 15 to 25% of mares will be aggressive.

KEY POINT

Diagnosis of ovarian abnormalities is by rectal examination and history. Ultrasonography and measurement of hormones are extremely helpful in some cases.

- Inhibin concentrations in the blood are elevated in about 85% of mares with GCTs, whereas testosterone is elevated in only 50% of afflicted mares.
- Hematomas may be confused with a GCT on rectal exam. Diagnosis of GCT and hematomas must be made on the basis of clinical signs (hematoma—normal estrous cycles), the rapidity of ovarian enlargement (hematomas—rapid), the presence (hematoma) or absence of the ovulation fossa, the size of the contralateral ovary (hematoma—normal size, follicles present), and the concentration of inhibin in plasma. On ultrasonographic examination hematomas appear uniformly echogenic. Some may appear as lucid areas separated by trabeculae, similar to those of a multicystic GCT. The GCT characteristically has a "honeycomb" appearance with multiple small nonechogenic areas separated by echogenic trabeculae (Fig. 8-14). However, the GCT can be highly variable in appearance. Some may contain one to two large cysts; others may be homogeneously dense throughout.
- Anovulatory follicles (hemorrhagic follicles) result when preovulatory follicles grow to an un-

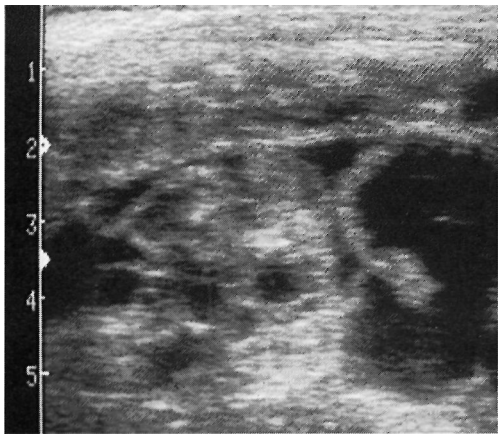


Figure 8-14. Ultrasound image of a granulosa cell tumor. There is a "moth-eaten" appearance to the ovary.

usual size (70-100 mm), fail to ovulate, fill with blood, and gradually recede. These follicles are distinctly echogenic on ultrasonography, with criss-crossing fibrin-like strands and some luteal tissue around the periphery. Mares will show erratic or persistent estrus.

- Mares with chromosomal abnormalities, ovarian atrophy, or mares in winter anestrus will show no estrus, will be passive when presented to a stallion, or will show some signs of estrous behavior. Ovaries will be small and firm. The uterus will be flaccid because there is no circulating progesterone. Definitive diagnosis is made by karyotyping the mare.

TREATMENT

Granulosa Cell Tumors. Although other ovarian tumors, notably teratomas, have been reported, GCTs are the most common.

KEY POINT

The affected ovary is removed by ovariectomy. Mares will return to normal cyclicity anywhere from immediately up to 24 months.

Unless the ovary is larger than a big orange, we have found that a flank approach provides the best access. However, it should be noted that subcutaneous seromas are common after flank laparotomy. In larger tumors, a midline or paramedian laparatomy is required because it is difficult to remove the ovary through the flank. Fertility of mares after surgery is good.

Persistent Corpus Luteum. Failure of normal luteolysis will result from persistent corpus luteum.

KEY POINT

Once persistent corpus luteum has been differentiated from early pregnancy, treatment with prostaglandin or an analogue will result in luteolysis.

Prostaglandin is given at a dose of 5 mg intramuscularly. Cloprostenol may also be used at a dose of 250 μg intramuscularly. Mares will show signs of estrus in 3 to 7 days with ovulation occurring 3 to 7 days later.

Anovulatory Follicles During the Transitional Season. Characteristic behavior in mares with large follicles is prolonged or irregular estrus. The endocrine machinery needed to induce ovulation early in the spring is not present so these follicles do not ovulate. During spring transition mares will develop three to four waves of follicles over 40 to 80 days. The follicles from the first two to three waves do not produce estrogen and do not respond to LH type drugs. However, the dominant follicle from the fourth wave produces estrogen and responds to LH drugs such as hCG (Treatment No. 25) at a dose of 2000 to 2500 IU given IV. Uterine edema visualized on the ultrasound is the only indication that the follicle is producing estrogen.

Anovulatory Follicles During the Ovulatory Season. Mares will show prolonged estrus and usually do go out of heat for 14 to 17 days. The follicle does not ovulate, but luteal tissue is laid down around the follicular wall so serum progesterone levels are elevated. Ovulatory drugs, such as hCG, do not appear to work. The cause is unknown.

Chromosomal Abnormalities and Ovarian Atrophy. In both conditions, there is no effective treatment. Mares with chromosomal disorders usually will have very small ovaries and a small uterus. Ovarian atrophy or senility usually occurs in mares older than 20 years of age.

Ovarian Hematomas. Ovarian hemotomas usually follow ovulation and may reach quite large dimensions. In most cases, the finding is incidental, because there are usually no abnormal behavioral signs. Although some hematomas may be persistent, there are usually no adverse effects, and no treatment is required.

Anestrus. Failure of mares to enter estrus is a common problem during the early part of the breeding season. As detailed in the section on management, the use of artificial lighting for 2

months before the start of the breeding season is useful to hasten the onset of regular estrus. Lactational anestrus may occur sometimes after foaling. Mares show estrus at the time of the "foal heat" and may ovulate, but then the ovaries regress and become small and firm. The cause is unknown, but it may be related to length of photoperiod because most mares that experience the problem foal early in the year. Removing the foal does not aid in resolving the problem. Many mares will resume cyclicity after 60 to 120 days. Behavioral anestrus occurs in some mares that are excessively protective of their foals even under the best of management conditions. Repeated rectal examinations, ultrasonography, and cervical exams can be used to determine when the mare is nearing ovulation. These mares usually can be covered by natural service, if they are tranquilized. It is best to inseminate artificially if the breed registry permits.

Perineal Lacerations

Because of the explosive nature of second-stage labor in the mare, if a foal's limb is positioned slightly abnormally, it may result in a tear in the dorsal vagina and through into the rectum. In some cases this tear is a localized communication (rectovaginal fistula), whereas in others a complete defect may result (third-degree perineal laceration). These injuries may be difficult to repair and can require one or more follow-up repairs after the original surgery.

HISTORY AND PRESENTING SIGNS

- History of dystocia
- Feces appearing at vulva
- Obvious communication between rectum and vagina

CLINICAL FINDINGS AND DIAGNOSIS

- The signs are obvious, and diagnosis is by visual examination to determine the type and extent of the perineal laceration.
- Injuries are classified into three grades:
  - **First Degree**—Superficial injuries to vaginal mucous membrane and rupture of the dorsal commissure of the vulva
  - **Second Degree**—Rupture of the perineal body
  - **Third Degree**—Penetration of the vaginal wall and rectum
- In addition, rectovaginal fistula, a fistulous tract allowing direct communication between the rectum and the vagina, is possible. In this case the perineal body is intact.

DIFFERENTIAL DIAGNOSIS

- Uterine rupture

TREATMENT

- First-degree lacerations do not require major treatment and can be repaired with a Caslick's operation.
- Second- and third-degree lacerations should be repaired surgically, although there is some debate as to whether immediate or delayed surgery is best.
- It appears that immediate surgery produces a higher rate of wound breakdown, although repair may be easier at this stage.
- If surgery is to be delayed, at least 3 to 4 weeks should elapse before reconstruction is attempted so that swelling and localized infection are under control.
- For third-degree perineal lacerations, we have found that standing repair using epidural anesthesia (see Fig. 8-11) is the technique of choice. In such cases, it is important that the mare be kept at pasture or fecal softening agents are used so that excessive tension on the suture lines does not occur. Feed should also be restricted for 24 hours before surgery.

Perineal Conformation Problems

For more than 50 years, veterinarians in stud-farm practice have been aware of the role of perineal abnormalities in the genesis of infertility.

KEY POINT

A sloping vulva, together with a sunken anus, allows pneumovagina with resulting endometritis in many cases.

The other major conformational problem is cranial displacement of the vestibule and urethral opening. This results in urovagina, which is commonly called "urine pooling."

HISTORY AND PRESENTING SIGNS

- Infertility
- Discharge from the vulva
- "Sucking air"

CLINICAL FINDINGS AND DIAGNOSIS

- The dorsal commissure of the vulva should be no more than 2 to 3 cm above the pelvic brim.

- The normal position of the vulva should be vertical. If the vulva slopes forward, this predisposes to fecal contamination and, combined with pneumovagina, may lead to cervicitis and endometritis.
- Examination with a speculum will indicate whether there is evidence of urine pooling in the anterior vagina.

DIFFERENTIAL DIAGNOSIS

- Rectovaginal fistulas
- Perineal lacerations

TREATMENT

Caslick Operation. The Caslick operation is widely used in stud-farm veterinary practice. It is the procedure of choice in mares that have a sloping vulva or those in which the dorsal commissure of the vulva is displaced dorsally above the pelvic brim.

Local infiltration of 2% lidocaine (Treatment No. 67) is used at the mucocutaneous junction of the vulva. The anesthetic is placed so that the vulva can be sutured to the level of the pelvic brim. This requires a total of approximately 30 mL of local anesthetic in most mares. Using a pair of sharp, curved scissors, a thin strip (2-3 mm) of the mucocutaneous junction is removed from the left and right sides, extending from the level of the pelvic brim to the dorsal commissure. Some clinicians prefer to make an incision at the mucocutaneous junction rather than removing the epithelium. The advantage of the incision technique is that less scarring and resultant skin thickening occur at the vulval edges. Suturing is performed using a nonabsorbable suture in a continuous interlocking pattern. If some tissue is excised, it is important that as little as possible be removed so that the vulval lips can meet without tension.

An episiotomy is required before foaling and also may be necessary before mating. In these cases, the episiotomy opening should be resutured as soon as practical.

Pouret's Operation. Because of the vulval damage that occurs and the reduction in the vulval opening, Pouret described an alternative operation that is useful to correct pneumovagina and also may be of value in correcting urine pooling. Pouret proposed that in older mares, the cranial displacement of the rectum and anus leads to similar displacement of the vagina and vulva because of the intimate soft-tissue connections between these structures.

KEY POINT

Surgery involves separation of the soft-tissue connections between the rectum and caudal vagina so that the vagina can move into a more normal caudal position.

This is performed with the mare in a standing position with an epidural anesthetic. The surgery involves a horizontal incision midway between the anus and the vulva, followed by blunt dissection for 10 to 12 cm cranial to the perineum. This frees up the attachments between the rectum and vagina so that the rectum can move cranially.

Urethral Extension. Mares that pool urine will do so during the third or fourth day of estrus just before ovulation. Rarely, will there be urine in the vaginal vault during diestrus. Mares will also pool urine after foaling if they have a large foal or delayed uterine involution. This second group will recover with time without surgical intervention. The urethra can be extended caudally by a number of surgical methods. About 30% of mares will develop a fistula in the extension. Most mares requiring an urethral extension have poor perineal conformation, which eventually results in the mare becoming infertile.

Postpartum Hemorrhage

Hemorrhage from a uterine artery is common in older mares and may or may not be fatal to the mare depending on how the hemorrhage progresses. If the hemorrhage slowly dissects into the broad ligament between the myometrium and the serosa of the uterus, it will form a hematoma. The resulting clot stops the arterial bleeding. If the broad ligament ruptures or the serosal surface of the uterus tears during formation of the hematoma, the mare quickly bleeds to death.

HISTORY AND PRESENTING SIGNS

- Pain
- Colic
- Anxiety
- Excitement or depression
- Cold extremities while sweating

CLINICAL FINDINGS AND DIAGNOSIS

- Mare may be found dead in stall after normal foaling.
- Mare may be found weak and unsteady on her feet.
- Diagnosis is made by rectal palpation of the uterus and broad ligaments. Palpation of the

broad ligament and uterine horn that are involved causes extreme discomfort.

DIFFERENTIAL DIAGNOSIS

- Pain associated with normal uterine involution
- Uterine rupture
- Colic from gastrointestinal involvement (large colon torsion, necrotic small colon, ruptured cecum or large colon, etc.)

TREATMENT

KEY POINT

The mare must be kept quiet. Confining the mare to a dark quiet stall, using mild sedation or opioids for pain if necessary, is the most successful treatment.

- Many therapies have been tried with limited success, including formalin, naloxone, hypertonic saline, plasma expanders, blood transfusions, and acepromazine.

Retained Placenta

Retention of the placenta in the mare is far less common than in the cow. The placenta is normally expelled rapidly, and if it is still present for longer than 3 hours after foaling, treatment for removal should be instituted. The cause of retention of the placenta is not always clear, and it is interesting that most placentas are retained in the nonpregnant horn. Retention of the placenta is a common sequela to abortion, especially if there are twins.

HISTORY AND PRESENTING SIGNS

- Abortion
- Normal foaling
- Premature delivery of foal
- Dystocia
- Twins

CLINICAL FINDINGS AND DIAGNOSIS

- The clinical signs are obvious, but if the membranes have been present for longer than 6 to 8 hours, it should be considered a medical emergency.
- Careful clinical examination including determination of heart rate, respiratory rate, temperature, and capillary refill time should be performed. Endotoxemia and metritis are common sequela if treatment is not instituted immediately.

DIFFERENTIAL DIAGNOSIS

- Uterine prolapse

TREATMENT

- Oxytocin at a dose of 20 IU given IM or IV may be used. This regimen is often sufficient to cause expulsion of the placenta within 30 minutes.
- If the placenta has not been expelled within 60 minutes, a further 20 IU may be given safely. A problem with large bolus administration (100 IU or more) of oxytocin is that it often causes abdominal discomfort, pain, and sweating.

KEY POINT

An alternative to intramuscular injection is to administer the oxytocin as an infusion.

- A 1-L polyionic IV solution is used, into which is placed 100 IU of oxytocin. This is infused slowly, and if the mare shows any signs of discomfort, the flow rate is decreased. A total of 500 to 750 mL usually is required. One hundred milliliters of calcium gluconate 23% can be added to the IV solution and administered slowly in stubborn cases. Heart rate and respiratory rate need to be monitored closely; if either increases greatly, the infusion should be stopped. Calcium is needed for smooth muscle contractions. Myometrial exhaustion can lead to retention of the placenta.

Manual Removal

If oxytocin is unsuccessful, systemic antibiotics should be given by 8 hours postfoaling, and the extent of attachment can be evaluated by internal exploration.

Manual removal of the membranes may be contraindicated because hemorrhage, endometrial scarring, and tags of placenta may be the result, especially if the placenta is pulled forcibly. If membranes are to be removed manually, they can be twisted around a sweat scraper or other linear object, which is held horizontally at the lips of the vulva (Fig. 8-15). The sweat scraper is then rotated clockwise or counterclockwise so that there is tension on the membranes. By slowly twisting, waiting for tension of the membranes on the sweat scraper to decrease (detachment), and then repeating the process, much or all of the placenta can be removed in the first attempt.



Figure 8-15. Retained placenta should never be pulled out forcibly. Wrapping the membranes around a sweat scraper and slowly twisting the placenta out does not appear to cause damage to the uterus. If the placenta does not loosen while it is being twisted, the operator should stop the procedure and try again later.

Antimicrobial and Anti-Inflammatory Therapy

Systemic antimicrobial therapy is required if the placenta is retained for more than 6 hours. The agents of choice are trimethoprim sulfa (Treatment No. 108) if the placenta is retained after a routine foaling; however, if it is retained after obstetric manipulations or abortion, procaine penicillin (Treatment No. 84) at a dose rate of 15-22 mg/kg (15,000-22,000 U/kg) every 12 hours and gentamicin (Treatment No. 56) at a dose rate of 3.3 mg/kg every 12 hours or 6.6 mg/kg every 24 hours are recommended.

KEY POINT

If there is a lack of response to the therapy that has been initiated, the uterus should be cultured. If there is predominate growth of one to two organisms, sensitivity patterns should be determined because certain strains of E. coli, *a common cause of metritis, are becoming increasingly resistent to gentamicin.*

The use of flunixin meglumine (Treatment No. 52) at dose rates of 0.25 mg/kg every 6 hours may be useful to aid in prevention of endotoxemia and laminitis.

Twinning

Twinning is a common noninfectious cause of abortion and accounts for 20 to 30% of all diagnosed abortions. Few twin pregnancies result in the birth of two live foals (14%) or the birth of one live and one dead foal (21%). Twin concepti must be identified early in gestation and one conceptus reduced to result in a singleton pregnancy before formation of the endometrial cups at day 38 of gestation.

HISTORY AND PRESENTING SIGNS

- History of double ovulations.
- Little in the history to indicate that twin pregnancies are present.

CLINICAL FINDINGS AND DIAGNOSIS

- Rectal palpation is extremely inaccurate for diagnosis of twins in the early stages of pregnancy and therefore is of little diagnostic use.
- Ultrasound examination provides the possibility of a definitive confirmation of the presence of twins, although care must be taken not to confuse embryonic vesicles with endometrial cysts.

KEY POINT

Diagnosis of twin pregnancy via ultrasound is best undertaken around 15 days after ovulation.

- If there is any doubt about the appearance of the embryonic vesicle, a follow-up examination must be performed before a decision about elimination of one or both fetuses.
- Embryonic vesicles may be side by side in one horn (unilateral) or in opposite horns (bilateral).

DIFFERENTIAL DIAGNOSIS

- Endometrial cyst

TREATMENT

- Twin pregnancies are eliminated by natural embryo death, manually induced embryo death, and spontaneous or induced abortion. Natural reduction of one of two embryos usually occurs between days 17 and 40 of gestation. About 70% of unilaterally fixed twins detected on days 11 to 15 are reduced to a singleton by day 40,

whereas embryo reduction is minimal when the vesicles are fixed bilaterally. Therefore, bilaterally fixed embryos must be reduced to singleton manually, preferably before day 25 of gestation. The best approach for unilateral twins that are not reduced by day 17 is to wait and evaluate the success of natural reduction. Before day 17, if the twin embryos are side by side, a second ultrasound examination is performed in 30 to 60 minutes. As both embryos are moving, they should be separated by the second exam and then one can be crushed manually. If twin vesicles are still present at day 36, the mare should be given prostaglandin (Prostin 5-10 mg) to induce abortion.

KEY POINT

The smaller of the vesicles is usually eliminated, and this is done by squeezing the vesicle or pressing it against the brim of the pelvis until it ruptures.

Uterine Prolapse

Uterine prolapse is uncommon in the mare, which is fortunate because it can be a difficult condition to correct. It usually occurs soon after foaling and is more common in mares that have had complicated deliveries. If not treated immediately, the mare may die from hypovolemic shock or from hemorrhage of the uterine artery.

HISTORY AND PRESENTING SIGNS

- Recently foaled mare
- Tissue mass protruding from the vulval lips
- Mare is straining severely
- Hypovolemic shock

CLINICAL FINDINGS AND DIAGNOSIS

- Diagnosis is not difficult because the prolapsed uterus appears as a red soft mass with a wrinkled surface. Depending on the duration of the exposure, there may be ulceration and the exposed surface may be desiccated.
- The other possible diagnoses that can be mistaken for uterine prolapse include rectal or bladder prolapse or premature separation of the chorioallantois in a foaling mare.

DIFFERENTIAL DIAGNOSIS

- Prolapse of the bladder
- Prolapse of the vagina
- Prolapse of the rectum
- Premature placental separation

TREATMENT

- Prompt treatment is essential.
- First aid measures include keeping the prolapsed mass moistened with warm saline and preventing the mare from lying down.
- Short-term general anesthesia (xylazine and ketamine, see Chapter 19) should be carried out to prevent straining and to ease replacement of the uterus.
- Medication to be administered should include tetanus prophylaxis, broad-spectrum, systemic antibiotics, intravenous fluids spiked with oxytocin (40-50 IU; Treatment No. 82), and calcium gluconate 23% (125 mL/5 L) to aid in uterine contraction.
- The uterus is rinsed with warmed normal saline containing K penicillin (1 million units per liter of saline). Massage of the uterus will permit replacement, but care must be taken that the endometrium is not torn because it can become extremely friable.
- Vulval retention sutures are not effective in preventing the uterus from prolapsing again.

Uterine Rupture

Uterine rupture can occur before or after parturition. Prepartum uterine rupture is associated with uterine torsion, whereas uterine rupture during parturition is associated with dystocia and manipulative procedures.

HISTORY AND PRESENTING SIGNS

- Colic
- Shock
- Depression
- Toxic

CLINICAL FINDINGS AND DIAGNOSIS

- Clinical signs depend on the extent of uterine rupture. If the rupture is large, the mare rapidly shows signs of hemorrhagic shock and may die. If the serosa remains intact, the mare may not show any signs until significant peritonitis has developed.
- Diagnosis is confirmed by rectal palpation, uterine palpation (via the vagina), and abdominocentesis.
- Inability to identify a tear by palpation does not preclude its presence because many are difficult to find.

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DIFFERENTIAL DIAGNOSIS

- Colic from gastrointestinal involvement
- Uterine artery rupture

TREATMENT

- Supportive therapy including appropriate systemic antimicrobials, intravenous fluids, nonsteroidal anti-inflammatory drugs, and peritoneal lavage.
- Surgical repair of tear through a ventral midline approach.

Uterine Torsion

Suspension of the equine uterus from the broad ligaments attached to the dorsolateral body wall makes torsion of the gravid uterus uncommon. Uterine torsion usually occurs from the seventh to ninth month of gestation.

HISTORY AND PRESENTING SIGNS

- Pregnant 7 months to term
- Colic

CLINICAL SIGNS AND DIAGNOSIS

- Colic signs may be mild to severe and appear to be related to tension on the broad ligaments or pressure necrosis of uterine wall.
- Diagnosis is made on rectal exam. One broad ligament will be stretched across the midline from left to right or right to left and will feel like a tight band. The mare will exhibit pain on palpation.
- Unlike the cow, vaginal signs of uterine torsion are not common unless the uterine torsion is more than 360 degrees.

DIFFERENTIAL DIAGNOSIS

- Large colon torsion

TREATMENT

KEY POINT

Although uterine torsion can be corrected by rolling the mare, surgical correction is best.

- In simple uterine torsions without uterine tissue necrosis, the uterus can be returned to normal and the pregnancy allowed to continue until term. Most torsions can be corrected through a standing flank laparotomy. The uterus is rolled back into normal position. The torsion is reduced by elevating from beneath and repelling the fetus.

Reproductive Disease in the Stallion

Castration

Castration is the most common surgical procedure performed by veterinarians in equine practice. Castration is performed mainly as a management strategy for horses that have no breeding future. In most cases, horses are presented for castration around 2 years of age. The main options are whether to perform the surgery with the horse standing or under general anesthesia.

SURGICAL TECHNIQUE

Standing Castration

- Standing castration is preferred by practitioners who work with Thoroughbred and Standardbred racehorses. These horses are usually well handled, and with adequate restraint, the technique is simple and has few complications. The standing technique for castration is most useful in horses less than 3 years of age. The main complication is injury to the operator from a kick by the horse due to insufficient desensitization.
- We prefer the horse to be tranquilized with 0.5 mg/kg xylazine (Treatment No. 109) and 0.05 to 0.1 mg/kg butorphanol (Treatment No. 15) given intravenously. An alternative to xylazine is detomidine (Treatment No. 28) at a dose rate of 10 to 20 (xg/kg given IV. This will result in profound sedation of the horse, at which time local anesthetic (10-15 mL of 2% lidocaine; Treatment No. 67) may be injected subcutaneously along the median raphe of the scrotum using a 23-gauge needle (Fig. 8-16). After allowing 2 to 3 minutes for the local anesthetic to take effect, 50 mL of 2% lidocaine is injected through the desensitized scrotal skin into the body of each testicle using a 19-gauge, 3.75-cm (1.5-inch) needle. The alternative is to use an 18-gauge, 8.75-cm (3.5-inch) spinal needle inserted up through the testicle into the spermatic cord (Fig. 8—17) and injecting 20 mL of 2% lidocaine. Local anesthetic injected into the testicle will diffuse into the spermatic cord and block local sensation.
- Surgery commences after appropriate skin disinfection of the scrotum, with a right-handed oper-



Figure 8-16. Standing castration. To desensitize the skin before desensitizing the spermatic cord, a 23-gauge needle is used to inject about 10 mL of 2% lidocaine along the median raphe of the scrotum.

ator standing on the right side of the horse, although some veterinarians prefer to stand on the horse's left side. The scrotum and testicles should be held in such a way that the incision is made away from the operator. The left testicle should be removed first, and the incision is begun at the cranial pole of the testicle, about 5 mm (0.2 inches) from and parallel to the median raphe of the scrotum. The incision should be at least 12 to 15 cm (5-6 inches) long.

KEY POINT

The most common complication from standing castration is excessive swelling around the penis and prepuce because the incision in the scrotum is too small and does not permit adequate drainage.

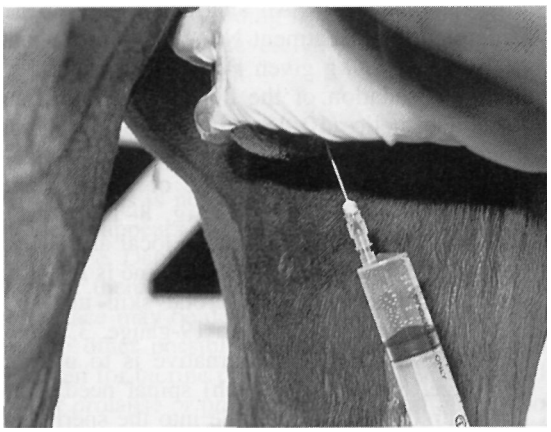


Figure 8-17. Standing castration. Desensitizing the spermatic cord can be done using an 18-gauge, 7.5-cm (3-inch) spinal needle directed through the desensitized skin of the scrotum into the spermatic cord. Before injection of the local anesthetic, the operator should ensure that the needle is not in a vessel.

- It is usual for the scrotum and parietal tunic to be incised together so that the testicle falls through the scrotal opening.
- For emasculation of the testicle, we prefer the use of a triple-crush emasculator and initial emasculation of the fibrous components (including the tunic) of the spermatic cord. This is followed by emasculation of the vascular pedicle, and the emasculators should be left in situ for at least 1 minute (Fig. 8-18).
- Before the emasculators are removed, a large hemostat is applied to the cord proximal to the emasculators. Thus, if there is hemorrhage when the emasculators are removed, the cord can be retrieved. A separate incision is made similar to the first, 5 mm (0.2 inches) from the median raphe on the right side, and the right testicle is similarly removed.
- To ensure that there is adequate drainage, we prefer to remove the median raphe between the two incisions.

Castration under General Anesthesia

- The combination of 1.1 mg/kg xylazine (Treatment No. 109) IV followed 3 to 5 minutes later by 2.2 mg/kg ketamine (Treatment No. 64) IV (see section on General Anesthesia in Chapter 19) is a simple combination that provides adequate time for castration.

KEY POINT

For right-handed operators, castration is most easily performed with the horse lying on its left side.



Figure 8-18. Standing castration. After incising through the scrotum and tunics, the testicle is removed using a triple-crush emasculator.

- A rope approximately 4 m (12-14 ft) long is tied around the pastern of the right hindleg and taken between the front legs and underneath the left side of the neck. The free end is then taken to form a half hitch around the right hind pastern so that the leg is flexed up and the foot is pulled cranially.
- Surgery is performed similarly to that described above, except that the lower (left) testicle is removed before the upper (right) testicle.
- Stallions older than 3 years should only be castrated under general anesthesia and the vascular pedicle ligated rather than emasculated.

POSTOPERATIVE CARE

- Procaine penicillin (15-20 mg/kg or 15,000-20,000 IU/kg; Treatment No. 84) is given IM before surgery. In a series of cases we did not administer penicillin and had an unacceptably high rate of local wound infections with *Streptococcus* species. A single dose appears to be adequate in controlling problems with postoperative infection.
- Administration of 4.4 mg/kg phenylbutazone (Treatment No. 89) is helpful in preventing excessive swelling at the castration site in the immediate postoperative period.
- If the horse has not been vaccinated for tetanus, tetanus antitoxin (3000 IU) is given subcutaneously.
- The horse should be observed carefully for signs of hemorrhage in the first few hours after surgery. Exercise is important in preventing excessive swelling and aiding in local drainage. Horses should be exercised twice daily for the first 5 to 7 days after surgery. Usually this involves lunging the horse for 10 to 15 minutes, followed by hosing the scrotal area with cold water, avoiding the wound.

COMPLICATIONS

Although castration is a simple operation, there are a range of complications that can cause problems for both the horse owner and the veterinarian.

Excessive Hemorrhage. Emasculation is a technique that is not foolproof for hemostasis. After most castrations, there will be minor degrees of hemorrhage, with blood dripping from the castration wound. In most cases, this stops within 30 to 60 minutes. However, if there is more severe hemorrhage, with blood coming from the wound in a steady stream, it is more serious and may require exploration of the site if the hemorrhage has not stopped within 30 minutes. To find the offending vessel, the horse is anesthetized and placed on its back. After appropriate skin disinfection and draping, the side where hemorrhage is evident is investigated to find the spermatic cord and ligate the particular vessel. In most cases it is not difficult to find the site of the hemorrhage, although it is usually necessary to explore some distance down the inguinal canal. If the source of the hemorrhage cannot be found, sterile gauze packing may be used to pack the region and apply pressure for hemostasis.

Excessive Local Swelling. This is the most common complication of castrations. It is more common after standing castrations, because when a castration is performed standing, it is more difficult to ensure that the scrotal skin incision is of adequate length. If the incision is too small, there will not be adequate drainage, and therefore excessive local swelling is the result. Under these circumstances, it may be necessary to enlarge the original incision so that drainage is effective.

KEY POINT

In most cases, the problem will resolve with 10 to 15 minutes of daily exercise, nonsteroidal anti-inflammatory drugs such as phenylbutazone (Treatment No. 89) at a dose rate of 2.2 mg/kg orally every 12 hours, and local hosing.

Local Infection. This is another common complication of castrations because in most circumstances the technique is carried out under conditions where sterility cannot be ensured. We have found that most local infections are due to *Streptococcus* species, and therefore IM procaine penicillin (Treatment No. 84) at a dose rate of 15 mg/kg every 12 hours is the treatment of choice. If sufficient drainage is ensured, most cases will respond to simple treatment. However, in some cases infection can ascend in the spermatic cord and result in scirrhous cord.

Scirrhous Cord. Scirrhous cord is a serious problem that results from infection ascending the spermatic cord. In some cases this is manifested as local swelling and purulent discharge from the scrotal wound. However, in some cases the infection may ascend and spread to the intraperitoneal part of the cord.

KEY POINT

Such cases may present many months after castration with signs such as hindlimb swelling and colic.

Cases of scirrhous cord must be treated by surgery, with removal of all infected cord. This can be very difficult when the infected cord extends through the internal inguinal ring. In these cases, marsupialization may be necessary so that the infection can drain externally. Appropriate samples (tissue and/or pus) should be taken at the time of surgery and appropriate antimicrobial therapy instituted.

Eventration. Eventration, with abdominal contents, usually small intestine, descending through the inguinal canal and out through the scrotum, is a rare but disastrous complication of castration. It usually occurs immediately after or within a few hours of surgery but in rare cases may occur several days later.

KEY POINT

Eventration is more common in Standardbreds than other breeds and should be anticipated in any colt that has had a history of a scrotal hernia as a foal.

If eventration does occur, early treatment is essential before contamination is severe and irreversible. To replace the intestine, a flank laparotomy is usually necessary.

Cryptorchidism

A cryptorchid is a colt or stallion with one or both testicles retained somewhere between the original embryologic site near the kidney and the scrotum. Cryptorchids are usually discovered when presented for castration, and many veterinarians have been embarrassed to discover that the horse they have anesthetized for a routine castration is a cryptorchid. For this reason, it is obviously essential to carefully examine any horse presented for castration before surgery. The most difficult situation is the horse presented with no testicles evident, no history available, but stallion-like behavior. If a testicle is retained, it is most commonly found somewhere in the inguinal canal. However, the testicle also can be intraabdominal.

HISTORY AND PRESENTING SIGNS

- Presence of only one testicle
- No testicles present but stallion-like behavior

CLINICAL FINDINGS AND DIAGNOSIS

- One or no testicles apparent on palpation of the scrotum.
- In some cases, deep palpation of the external inguinal ring with the horse under tranquilization will result in the testicle or epididymis being palpable.
- The inguinal rings are explored by rectal palpation to determine the location of a retained testis and to evaluate the contents of the inguinal canal in horses experiencing inguinal or scrotal hernia. Both the superficial and deep inguinal rings should be palpated to locate a retained testis. The superficial inguinal ring can be located by palpating the ventral abdominal wall between the penis and the medial aspect of the thigh. The opening of this ring is 2 to 3 cm in diameter and the canal is 10 to 12 cm long. The ring is directed laterally, cranially, and slightly ventrally from the edge of the prepublic tendon. The deep inguinal rings are identified on rectal palpation as slit-like openings ventrolateral to the pelvic brim. To locate them, all feces are cleared out of the rectum as far cranially as can be reached. The examiner then sweeps the hand medially and laterally while slowly retracting the hand caudally. During the procedure the hand will come into contact with either vessels entering the inguinal ring or with the ring itself.
- Rectal examination should be performed to determine whether some of the cord structures can be palpated traversing the internal inguinal ring. Although this is often difficult, in some cases of a retained intraabdominal testicle, the testicle can be palpated close to the internal inguinal ring.

KEY POINT

If there is doubt about the presence of one or both testicles in a horse showing stallion-like behavior, it is possible to perform plasma hormone analyses to aid in diagnosis.

- In horses older than 3 years of age, measurement of plasma estrone sulfate concentrations is useful, with cryptorchid horses having values greater than 400 pg/mL. In younger horses, a hCG response test is used, with plasma being collected for testosterone concentrations before and 24 hours after the administration of 10,000 IU of hCG. Cryptorchid horses will show a substantial rise in testosterone concentrations after hCG. It also should be noted that basal testosterone levels are also higher than in geldings.

DIFFERENTIAL DIAGNOSIS

- Castrated horse with adrenal production of testosterone

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- Castrated horse with epididymis removed and mistaken for testicle

TREATMENT

- Because of the likely hereditary nature of cryptorchidism, the horse should be castrated rather than attempts made to relocate the testicle.
- Most cryptorchid cases are unilateral, and the affected testicle is removed via an inguinal approach.
- In cases where the testicle is enlarged, the testicle may not be capable of being removed via the inguinal canal, and a paramedian laparotomy is the technique of choice.

Habronema Infestation

Larvae from *Habronema muscae* can burrow into the urethral process, which is attractive to the flies because of the moist surfaces. The resulting lesion can resemble a squamous cell carcinoma, and histopathology may be necessary to confirm the diagnosis.

HISTORY AND PRESENTING SIGNS

- Swelling around the prepuce
- Discharge from the prepuce
- Hemospermia

CLINICAL FINDINGS AND DIAGNOSIS

- In early cases there may be little to be found on clinical examination. Longer-standing cases usually demonstrate localized edema involving the urethral process, and there may be a granulomatous reaction.

KEY POINT

Lesions can occur on the prepuce but are more common on the urethral process.

- In chronic cases there is usually some degree of ulceration, and typical caseous lesions are spread throughout the affected site.
- If there is doubt about the diagnosis, a biopsy of material may be necessary at the time of treatment.

DIFFERENTIAL DIAGNOSIS

- Neoplasia
- Viral papillomatosis
- Balanoposthitis
- Trauma

TREATMENT

- Ivermectin administration cures most cases.
- In chronic cases of infestation of the urethral process, amputation of the process may be necessary. To prevent constriction, the urethral mucosa is then sutured to the skin around the process.

Hydrocele

An abnormal amount of fluid can accumulate between the visceral and parietal layers of the tunica vaginalis and surround the testes. The condition occurs most commonly during hot, humid weather.

HISTORY AND PRESENTING SIGNS

- Enlarged scrotum without pain
- Hot, humid weather

CLINICAL FINDINGS AND DIAGNOSIS

- Initial diagnosis is based on palpation of the scrotal contents. A hydrocele feels like a large compressible fluid-filled bag. The scrotum is generally not painful.
- Ultrasonographic examination reveals clear (black) to slightly speckled (white dots) fluid within the scrotum surrounding the testes.
- Definitive diagnosis is based on aseptic needle aspiration of fluid from the vaginal cavity.

DIFFERENTIAL DIAGNOSIS

- Scrotal hernia
- Scrotal trauma
- Epididymitis

TREATMENT

- Remove the underlying cause if it can be identified. Infectious conditions and malnutrition have been associated with hydroceles.
- Fluid usually reaccumulates when drained from the vaginal cavity by needle aspiration, unless an inciting factor for the hydrocele is identified and corrected.
- If a large amount of fluid accumulates (>200 mL), it may cause heat-induced testicular degeneration with a resultant decline in semen quantity and quality.

Neoplasia of the Penis

Neoplasia of the penis and/or prepuce is rare in the horse. Squamous cell carcinomas are by far the

most common tumors affecting the penis. Early diagnosis is important because metastasis to regional lymph nodes can occur. Other tumors that can affect the penis include melanomas, sarcoids, and hemangiomas.

HISTORY AND PRESENTING SIGNS

- Discharge from the prepuce
- Hemospermia
- Swelling around the preputial area

CLINICAL FINDINGS AND DIAGNOSIS

- Horses will vary in presenting signs, but many cases are presented because of discharge from the prepuce.
- Examination should be performed with the horse sedated with xylazine at a dose rate of 0.2 to 1 mg/kg of body weight.
- Squamous cell carcinomas appear usually on unpigmented skin and have an erosive appearance.
- Melanomas are most common in gray horses and may be found in the preputial area without causing any adverse clinical signs.
- If the lesion is localized, a biopsy should be taken to enable histopathology to be performed and a definitive diagnosis made.
- In more extensive lesions, it may be necessary to amputate the penis. This technique is obviously more useful in geldings than in stallions.

KEY POINT

In some cases where there is a squamous cell carcinoma that has been present for some time without causing adverse clinical signs, there may have been metastasis to regional lymph nodes. It is important to palpate the inguinal lymph nodes and to also palpate abdominal lymph nodes by a rectal examination.

- In cases where metastasis has occurred, there may be obstruction of local lymphatic drainage, resulting in local swelling around the prepuce. There also may be swelling of one or both hindlegs.

DIFFERENTIAL DIAGNOSIS

- Habronemiasis
- Trauma
- Balanoposthitis
- Viral papillomatosis

TREATMENT

- Where the lesions are found early in the course of the problem, local excision using cryosurgery appears to provide the best results.
- In some cases, radiation therapy using implants of ^{222}Rn has been found to be effective in treating localized lesions.

KEY POINT

Where the tumor is more extensive, amputation of the penis is required.

- This is a salvage procedure in a stallion and is used most commonly in geldings. Where there is more extensive disease, it may be necessary to ablate the prepuce and divert the penis caudally so that the urethra opens ventral to the ischium.
- If there is evidence of metastasis, the prognosis is very poor, and euthanasia is the only real option.

Penile Paralysis

KEY POINT

Penile paralysis is most common in stallions after receiving one of the phenothiazine-derivative tranquilizers, such as promazine (Treatment No. 96) or acepromazine (Treatment No. 1).

Since the advent of xylazine, the phenothiazine tranquilizers are used less commonly, and the incidence of penile paralysis has become less.

HISTORY AND PRESENTING SIGNS

- Administration of a phenothiazine-derivative tranquilizer
- Prolonged protrusion of the penis

CLINICAL FINDINGS AND DIAGNOSIS

- Diagnosis is not a difficulty, but an accurate history should be taken to determine whether other neurologic problems have contributed to the problem.
- The penis appears to be engorged and partially erect, and edema usually develops early in the course of the problem.
- There is congestion and stagnation of blood in the corpus cavernosum, which accentuates and aggravates the condition.

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DIFFERENTIAL DIAGNOSIS

- Neurogenic paralysis
- Traumatic paralysis
- Phenothiazine-induced paralysis

TREATMENT

- If the injury is the result of a kick or other trauma, local anti-inflammatory measures, including the application of ice packs, are useful.
- If the problem is drug induced, early support may prevent passive congestion and edema. Successful treatment of an acepromazine-associated penile paralysis has been reported using benztropine mesylate (Cogentin, Merck & Co., Inc., West Point, PA) given IV at a dose rate of 8 mg for a 500-kg horse.
- Local massage and support of the penis are essential to reduce the edema and congestion. Supporting the penis in some type of sling prevents the stasis and congestion of blood that occurs by virtue of the dependency. It is useful if the sling is made of a mesh material (usually nylon), which enables the stallion to urinate without restriction.
- If the penis can be returned to its position in the prepuce, using some type of skin emollient to prevent local trauma, the prognosis is improved.
- If there is marked inflammation and swelling, the use of systemic phenylbutazone at initial dose rates of 4.4 mg/kg twice a day for 2 days, followed by 2.2 mg/kg twice a day for 4 to 7 days, may be of assistance.
- If there are doubts about the horse's ability to pass urine, catheterization of the urinary bladder may be required.
- In cases that are nonresponsive to medical management, the reefing operation or, in more severe cases, amputation of the penis is required.

Seminal Vesiculitis

Infection of the accessary sex glands in the horse is rare. If there is blood, pus, bacteria, or neutrophils in the semen it should be suspected. Some stallions will be reluctant to breed or ejaculate with an acute infection, whereas others may show pain during ejaculation. Most stallions, however, exhibit normal breeding behavior.

HISTORY AND PRESENTING SIGNS

- Blood, pus, or white blood cells in ejaculate
- Pain on ejaculation
- Reluctance to breed

CLINICAL FINDINGS AND DIAGNOSIS

- It is difficult to selectively collect fluid from the seminal vesicles.
- Diagnosis is most often made on rectal examination and ultrasonographic findings. Infected glands may be enlarged and firm. Ultrasonographically, there will be echogenic fluid with white speckles in the fluid in the gland.

DIFFERENTIAL DIAGNOSIS

- Orchitis
- Epididymitis
- Urethritis
- Cystitis

TREATMENT

- Instillation of appropriate antibiotics (culture and sensitivity growth patterns of bacteria isolated from semen) into the lumen of the vesicular glands using a small-diameter (2-mm) catheter placed through the urethral opening of each vesicular gland using an endoscope. Stallions should be treated for 8 to 10 days. Systemic antibiotics diffuse poorly into the accessory genital glands.

Testicular Degeneration

Degeneration of the testes is an acquired condition that can affect one or both testes, depending on whether the causative factor is localized, as with a locally aggressive tumor, or generalized, as with fever. The condition can be temporary or permanent. Thermal injury is the most common cause.

HISTORY AND PRESENTING SIGNS

- Decreased conception rates
- Fever within the last 60 to 90 days
- Anabolic steroids given
- Scrotal trauma

CLINICAL FINDINGS AND DIAGNOSIS

- Testes can be either small and soft to small and firm when palpated.
- Sperm output is decreased from normal and there is a large number of morphologically abnormal cells.
- The stallion may still have clinical signs of infection or scrotal trauma.

DIFFERENTIAL DIAGNOSIS

- Sperm granuloma
- Blocked ampulla
- Testicular hypoplasia

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TREATMENT

- If the degeneration occurs secondarily to fever, sexual rest for 90 to 120 days is indicated.
- If anabolic steroids are being given, administration must be stopped.
- Degeneration may occur with aging for which there is no treatment.

Testicular Torsion

Torsions usually occur in descended testes and can be transient or permanent. Most spermatic cord torsions range from 180 to 360 degrees. Clinical signs depend on the severity of the torsion and the extent of circulatory disturbance.

HISTORY AND PRESENTING SIGNS

- A 180 degree torsion may be an incidental finding during examination of the external genitalia.
- Some stallions will exhibit pain during performance with a 180 degree torsion. A 360 degree torsion generally causes acute severe scrotal pain and scrotal enlargement from local edema and hemorrhage.

CLINICAL FINDINGS AND DIAGNOSIS

- In a 180 degree torsion, the cauda epididymis and the caudal ligament of the epididymis are palpated in the cranial scrotum.
- With a 360 degree torsion, the cauda epididymis and caudal ligament of the epididymis are palpated in their proper scrotal positions. Acute scrotal pain in conjunction with unilateral scrotal enlargement and edema are detected.

DIFFERENTIAL DIAGNOSIS

- Inguinal/scrotal hernia
- Scrotal trauma
- Orchitis
- Testicular neoplasia
- Testicular hematoma

TREATMENT

- Treatment of 180 degree torsion is not necessary unless the stallion exhibits pain during exercise. An orchiopexy can be performed.
- A 360 degree torsion is a medical emergency. Unilateral castration is recommended.

Venereal Diseases

Although a variety of viral, bacterial, and protozoal venereal diseases occur in the stallion, most of these are uncommon and unlikely to be encountered in clinical situations. However, many diseases have significance in relation to quarantine concerns. Some diseases no longer occur in North America, and many are unknown in the southern hemisphere.

HISTORY AND PRESENTING SIGNS

- Preputial discharge
- Infertility
- Anorexia
- No presenting signs

CLINICAL FINDINGS AND DIAGNOSIS

Dourine. This protozoal disease has been eradicated from North America and is found only in Africa, the Middle East, and South America. It has a slow incubation period of several weeks, and the horse may have a urethral discharge. Systemic involvement is evident, with elevated body temperature and plaques developing on the lower body. Later in the course of the disease, penile paralysis may occur, and neurologic signs such as ataxia and, in extreme cases, paralysis are found. Diagnosis is best made using a serum sample for a complement fixation test.

Herpesvirus. Herpesvirus 3 infection is known as *coital exanthema.* It is a self-limiting disease, but in the acute stages it can produce vesicles on the penis, which can ulcerate and become secondarily infected with local bacteria.

Equine Viral Arteritis. This disease is very commonly carried by stallions that frequently are asymptomatic. It is particularly common in Standardbred stallions. Only a small number of horses in an affected group may show clinical signs, and the signs can vary from ocular to abdominal. In mares, abortion is the most significant sign of viral arteritis. Confirmation of infection is best done using a viral neutralization test on blood.

Equine Infectious Anemia. This viral infection has important economic consequences and is spread by biting flies. However, because the virus has been found in semen, a venereal mode of transmission has been proposed. Diagnosis is made on serum samples using a Coggins test.

Bacterial Diseases. CEM was the most important bacterial venereal disease in recent years and resulted in outbreaks of infection in both the northern and southern hemispheres. Because of rigorous bacteriologic testing, the disease has now been brought under control. Other bacterial venereal diseases of sporadic significance include those

caused by *Klebsiella* and *Pseudomonas*. These infections are usually diagnosed because of endometritis in a number of mares that have been mated with the affected stallion.

DIFFERENTIAL DIAGNOSIS

- Contagious equine metritis organism
- Other bacterial venereal diseases
- Herpesvirus 3
- Equine viral arteritis

TREATMENT

- Most venereal infections are not responsive to treatment and must be controlled by sexual rest.
- Many viral diseases are self-limiting and will resolve with time.
- Bacterial venereal infections are particularly difficult to treat and control. Local washing and disinfection of the penis may lead to worsening of the infection. Local treatment with antibiotics, particularly gentamicin, may be worthwhile in some cases.

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CHAPTER 9

Pediatrics

Reuben J. Rose and David R. Hodgson

CLINICAL ASPECTS OF NEONATAL PHYSIOLOGY

In the change from intrauterine to extrauterine existence, remarkable adaptations occur as the foal moves from a fluid to a gaseous environment. If these adaptations do not follow in proper sequence or some fetal circulatory pathways remain open (e.g., ductus arteriosus, foramen ovale), signs of maladaptation may appear.

In the period immediately after delivery, gasping respiration occurs, and the alveoli open, with absorption of pulmonary fluid. Arterial blood gases reflect the changes in gas exchange across the lung, and over the first few days of life they show a pattern of decreasing tensions of carbon dioxide ($Paco_2$) and increasing tensions of oxygen (Pao_2), as demonstrated in Figure 9-1. The respiratory rate is high during the first 4 to 6 hours after birth, with values in the range of 50 to 90 breaths per minute. This rate will be influenced by environmental temperature, with higher respiration rates in colder conditions.

KEY POINT

Body position has a major influence on pulmonary gas exchange and is of major significance when managing the sick foal.

Even in healthy normal foals, the Pao_2 may be 10 to 30 Torr (mm Hg) lower when the foal lies in lateral recumbency as compared with sternal recumbency. Therefore, when there are signs of maladaptation or cardiorespiratory problems, foals should be placed in sternal recumbency to ensure that the arterial oxygenation is optimized.

In the immediate neonatal period, there may be extensive right-to-left shunting of blood because the various fetal circulatory pathways do not close immediately.

KEY POINT

The major fetal circulatory pathway of clinical importance is the ductus arteriosus, which usually closes within 12 hours of birth.

Most foals older than 24 hours do not have a patent ductus arteriosus, although it has been reported in normal foals for up to 5 to 6 days after birth.

KEY POINT

A systolic heart murmur is a normal finding in foals for up to 3 to 4 months after birth and

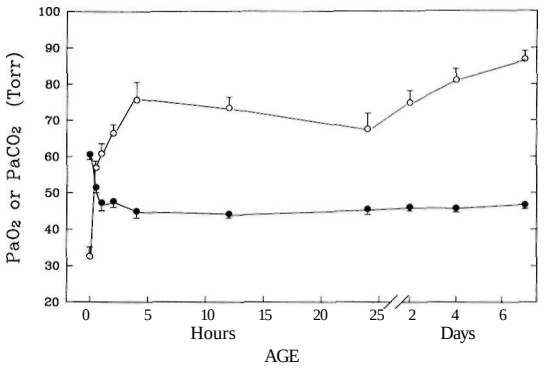


Figure 9-1. Arterial oxygen (Pao_2) (o) and carbon dioxide ($Paco_2$) (•) tensions in foals from birth to 7 days of age. (From Stewart, J. H., Rose, R. J., and Barko, A. M.: Respiratory studies in foals from birth to one week of age. *Equine Vet. J.* 16:323, 1984, with permission.)

should not be confused with the continuous "machinery" murmur of a foal with patent ductus arteriosus.

Immediately after delivery, the foal obtains up to 1.5 L of blood from the placenta, which is important for establishing normal blood volume.

KEY POINT

In some mares that foal standing or when the mare stands quickly after delivery, the umbilical cord may rupture and deprive the foal of vital blood.

This may result in hypovolemia and shock, which may be manifest in an affected foal as maladaptation, with a prolonged period before the foal gets to its feet and sucks. Acid-base measurements in these foals have shown quite a profound metabolic acidosis because of reduced peripheral perfusion and increased lactate production. Normally, there are no metabolic acid-base disturbances in newborn foals, the values for standard base excess and bicarbonate concentration being within the reference adult range.

KEY POINT

Most foals will stand within the first hour after birth and should suck from the mare by 2 hours after birth.

Foals that do not conform to these criteria should be examined carefully, because a range of congenital abnormalities and metabolic derangements can lead to the presenting sign of maladaptation.

During the first week after birth, there are gradual increases in the Pao_2 and improvements in cardiac function. This first week is the period when foals are most susceptible to any problems that result in cardiorespiratory dysfunction. The most common causes of cardiorespiratory abnormalities are bacterial infections that result in septicemia.

CLINICAL EXAMINATION OF THE FOAL

In Chapter 1 we discussed details of the general clinical examination, particularly related to adult horses. This approach also applies to examination of the foal, but there are some specific aspects of foal examination that need to be emphasized.

General Examination

HISTORY

Collection of the history is important with any condition, but in the case of newborn foals, it should also include questions relating to the health of the mare. Before foaling, details such as the presence of milk dripping from the teats, vulval discharge, and previous foaling difficulties (including foals born dead or dying shortly after birth) should be obtained. These aspects of history may indicate intrauterine infection.

DEMEANOR

The demeanor of the foal provides important clues to its state of health. Most sick foals look sick.

KEY POINT

Foals with metabolic derangements, sepsis, respiratory distress, and maladaptation usually stop sucking the mare, so the mare's udder becomes engorged and the mare may drip milk.

MUCOUS MEMBRANES AND PULSE

In taking the vital signs, attention should be paid to the mucous membranes, capillary refill, and peripheral pulses. The extremities should be warm, and a pulse should be palpable in the great metatarsal and brachial arteries.

AUSCULTATION OF THE HEART

The normal heart rate in foals from 24 hours to 7 days of age is usually in the range 60 to 110 beats per minute, with the resting heart rate becoming lower over the first week of life. A systolic heart murmur can be heard in a high percentage of foals for up to 2 to 3 months after birth. In most cases, the murmur is grades I to III (see Chapter 6) and is a normal finding.

RESPIRATORY SYSTEM

The respiratory rate is highest in the first hour after birth. The range of respiratory rates in foals up to 1 week varies from 25 to 60 breaths per minute. Rates in the range of 70 to 90 breaths per minute may be found during the first hour after birth. Because respiratory rate is affected by the state of excitement of the foal, care should be taken when recording and interpreting the respiratory rate.

KEY POINT

Auscultation of the chest can be misleading in some foals with severe respiratory disease, with no abnormal lung sounds despite severe pneumonia and atelectasis.

Foals with suspected pulmonary disease should have chest radiographs taken and, if possible, arterial blood gas analysis performed (see later section on Diagnostic Aids).

GASTROINTESTINAL SYSTEM

Auscultation of the abdomen is important to detect ileus, and palpation may help to determine some impactions. Foals will often show signs of severe abdominal pain, which may be difficult to localize. In general, a conservative approach to management is indicated, and exploratory laparotomy is seldom required. The major problem of the newborn foal is retained meconium, which is found more commonly in colts than in fillies.

RECTAL TEMPERATURE

Rectal temperature is usually in the range of 38 to 39°C (100-102.5°F). The temperature increases from birth to 4 days of age and thereafter plateaus.

KEY POINT

Newborn foals that have subnormal temperatures have a worse prognosis than those with normal or elevated temperatures.

In foals that are active or have been out in the sun, the temperature may be transiently elevated to greater than 39°C. However, temperatures greater than 39°C should be regarded with suspicion. It should also be remembered that many foals with bacteremia and septicemia do not have elevated body temperatures.

URINARY SYSTEM

Although urine may not be passed until the foal is 6 to 10 hours of age, frequent urination of dilute urine is normal in newborn foals.

KEY POINT

The most important area to examine is the umbilicus, which may be infected or more commonly involved in a patent urachus.

In this condition, there is a communication between the urinary bladder and the umbilicus so that urine dribbles from the umbilicus.

MUSCULOSKELETAL SYSTEM

Examination of the musculoskeletal system of the newborn foal can provide useful clues about the foal's maturity.

KEY POINT

In premature foals, there is often overextension of the fetlocks, and the foals will be weak and have difficulty in standing.

Radiographs of the carpus and tarsus may be useful to assess the degree of ossification. Evaluation for evidence of joint effusion is also important because localization of bacterial infection in joints ("joint ill") and epiphyses is a common problem.

PLACENTA

Where problems are found very soon after birth, examination of the placenta for abnormalities should be done. Weighing the placenta also can provide an indication of possible placental insufficiency. Normal weights for placentas in Thoroughbred horses are in the range of 4.5 to 7.0 kg.

UTERINE BACTERIOLOGY

If a bacterial infection is suspected in a newborn foal, it may be valuable to obtain uterine swabs from the mare (see Chapter 8) to determine whether infection is present.

RESTRAINT OF FOALS

In neonatal foals, restraint in lateral or sternal recumbency usually is required for venous catheterization, arterial blood collection, or intranasal oxygen administration. However, when nasogastric tubing, intravenous or intramuscular injections, or venous blood collection is necessary, foals can be restrained while standing. For injections and venous blood collection, it can be helpful to back the foal into a corner of a box stall and grasp both ears, as shown in Figure 9-2. Figure 9-3 shows restraint by holding one arm around the chest and the other arm pulling the tail up over the rump.

DIAGNOSTIC AIDS

A range of diagnostic aids will be useful to assess various diseases in newborn foals. Many of these (e.g., transtracheal aspiration, bronchoalveolar la-



Figure 9-2. Restraint of the foal. The foal is restrained by backing it into a corner of a box stall and grasping both ears.

vage, abdominocentesis, cerebrospinal fluid collection) have the same indications and technique as in adult horses, and the particular diagnostic aid is discussed under the appropriate body system. However, some techniques may be of more importance in the foal or may be somewhat different from those in the adult horse. The most commonly used of these techniques are discussed below.

Blood Culture

KEY POINT

The use of blood culture is a vital part of the workup in any foal with suspected septicemia.

In some cases, bacteria may even be seen on a blood smear and can provide useful information for commencing antimicrobial therapy. Details of blood culture methodology are given in Chapter 17. Particular care must be taken to avoid contamination with skin microorganisms, and therefore, attention must be paid to appropriate skin disinfection.

Arterial Blood Gas Analysis

KEY POINT

Arterial blood gas and acid-base analyses are essential aspects of the evaluation of newborn foals showing maladaptation or signs of dyspnea.

Venous blood may be used to assess acid-base status, but only arterial blood samples may be used to evaluate gas exchange. More specialized practices may have a blood gas machine in the practice laboratory. However, in most cases, arrangements can be made with a local hospital to process the occasional blood gas sample. Special blood gas syringes are available containing powdered heparin, but it is just as easy to use 2-mL syringes and fill the dead space of the syringe with sodium heparin (1000 IU/mL). It is important to ensure that there are no air bubbles in the syringe because the blood gas values obtained will be erroneous. Approximately 1.5 to 2 mL of blood should be collected and immediately capped by bending the needle over or inserting the needle into a rubber stopper such as the top from a Vacutainer tube. The sample should be placed in ice or an ice water bath before analysis, which should be completed within 4 hours of collection. Because the blood gas measurements are made at 37°C, it is important to note the foal's body temperature at the time of collection. Temperatures above 37°C will result in higher values for Po_2 and Pco_2 than uncorrected values at 37°C. Most machines perform the correction automatically if the patient's temperature is programmed in during the sample processing.

In foals up to 2 weeks of age, arterial blood samples are most easily collected with the foal restrained in lateral recumbency. After collection, digital pressure should be applied for 2 minutes



Figure 9-3. Restraint of the foal. For minor procedures, the foal can be restrained by holding one arm around the chest and pulling the tail up over the back.

to the site of needle penetration. The following sites may be used for arterial blood collection:

Great Metatarsal Artery—On the lateral aspect of the third metatarsal bone, the great metatarsal artery is easily palpated and is relatively fixed in position. We find that either 23- or 25-gauge, 15-mm (5/8-inch) needles can be used for blood collection. Most foals will struggle briefly when the needle is inserted, and therefore the hindleg should be restrained and the needle inserted quickly through the skin before connection of the heparinized syringe (Fig. 9—4). In foals showing signs of shock, it may not be possible to palpate a pulse in the great metatarsal artery.

Palmar (Digital) Artery—The palmar arteries are readily palpable on the abaxial surface of the fetlock. Sometimes a pulse will not be detected, but it is possible to insert a 25-gauge, 15-mm (5/8-inch) needle into the artery, as shown in Figure 9-5.

Brachial Artery—In newborn foals that have weak or absent peripheral pulses, usually it is possible to palpate a pulse in the brachial artery as it crosses the medial aspect of the proximal forearm near the insertion of the pectoralis descendens muscle. A blood sample can be collected using a 23-gauge, 25-mm (1-inch) needle.



Figure 9-5. Arterial blood gas collection from the palmar (digital) artery. The artery is easily palpable over the abaxial surface of the sesamoids. However, the artery is much more mobile and therefore slightly more difficult to penetrate than the great metatarsal artery.

Intranasal Oxygen Administration

Intranasal oxygen is used therapeutically in foals with respiratory distress if there is hypoxemia.

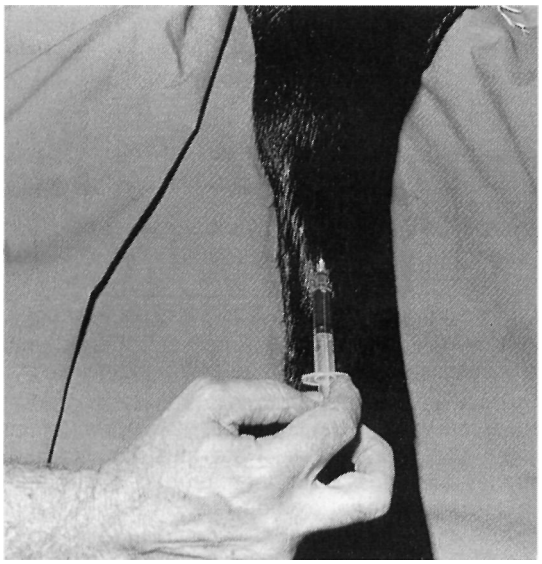


Figure 9-4. Arterial blood gas collection from the great metatarsal artery on the right hindleg. The artery runs along the lateral aspect of the metatarsus and is relatively immobile.

KEY POINT

Intranasal oxygen administration is also useful as a diagnostic tool to assess cardiopulmonary function.

With the foal restrained in lateral recumbency, a soft infant nasogastric catheter is placed via the ventral nasal meatus into the nasopharynx. An oxygen cylinder with a rotameter capable of delivering 10 L/min of oxygen is used, and the oxygen is bubbled through a container of water to humidify the gas (Fig. 9-6). In foals up to 1 week of age, a flow rate of 10 L/min is equivalent to administering 100% oxygen using a mask and valve system. Foals with normal cardiopulmonary function should at least double the arterial oxygen tension. The normal response of foals administered oxygen in lateral recumbency is shown in Figure 9-7, and it is apparent that 5 minutes of intranasal oxygen are sufficient to evaluate the foal's capability for elevating the arterial oxygen tension.

Thoracic Radiography

Although thoracic radiographs will not allow specific disease diagnosis, radiographs are essential to determine the degree of lung pathology.



Figure 9-6. Intranasal oxygen administration with the foal lying in a box stall. This may be used diagnostically to determine the elevation in arterial oxygen tensions or therapeutically to correct hypoxemia associated with pulmonary problems. Flow rates around 10 L/min of oxygen are used, and the gas is humidified. In foals with pulmonary disease, sternal recumbency is preferred to lateral recumbency.

KEY POINT

Using portable xray machines, satisfactory films can be obtained, particularly using rare earth screens.

The most common abnormality in infectious respiratory disease is an interstitial pattern or pulmonary infiltrate. In some cases, the infiltrate may be localized to certain regions of the lung fields (e.g., cranioventral or caudodorsal), suggesting inhalation or aspiration pneumonia. Some pulmonary diseases produce a typical radiographic appearance, for example, *Rhodococcus equi* pneumonia with large abscesses scattered through the lung fields.

Hematology and Biochemistry

The normal hemogram of the newborn foal is similar in many regards to that of the adult. Foals do not have fetal hemoglobin, and the red cell indices decrease rapidly after birth. The hemoglobin decreases from values in the range of 140 to 180 g/L (14-18 g/dL) at birth to 120 to 160 g/L (12-16 g/dL) at 24 hours of age. Thereafter, there are few changes in mean values, the red cell indices being a little lower than those in adult horses. Red cell size is smaller than in adult horses, with mean cell volume (MCV) values around 35 to 40 fL. Leukocyte values are also similar to adult values, with a predominance of neutrophils. The number of neutrophils gradually diminishes with age, and at about 6 months, foals have similar numbers of neutrophils and lymphocytes. During the first few days of life, the neutrophil-to-lymphocyte ratio is greater than 1.5, and values less than this may be found in foals with prematurity associated with low levels of plasma Cortisol. Studies in Newmarket, U.K., have shown that premature foals with neutrophil-to-lymphocyte ratios of less than 1.5 have a poorer chance of survival than those with higher ratios.

Enzymes in foal plasma or serum are often considerably higher than in adults, and little significance can be attached to elevations until the foal is more than 3 months of age. The most notable change is in alkaline phosphatase, where values in the first few days of life may be greater than 1000 IU/L. However, elevations in muscle-derived enzymes are of clinical significance.

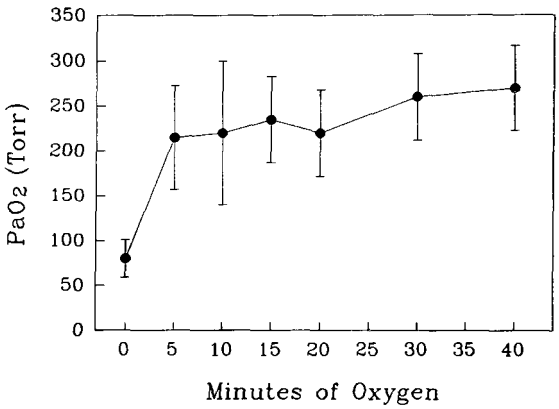


Figure 9-7. Administration of intranasal oxygen at a flow rate of 10 L/min to normal thoroughbred foals in the first week of life. Note that 5 minutes of intranasal oxygen is sufficient to produce a maximum elevation in arterial oxygen tension (Pao_2). (From Rose, R. J., Hodgson, D. R., Leadon, D. R, and Rossdale, P. D.: The effect of intranasal oxygen administration on arterial blood gases and acid-base parameters in spontaneously delivered, term induced and induced premature foals. *Res. Vet. Sci.* 34:159, 1983, with permission.)

Plasma or serum electrolytes are similar to values in adult horses except that values for potassium are slightly higher.

NEONATAL INTENSIVE CARE IN PRACTICE

Most equine practices will not have the range of sophisticated equipment necessary for full intensive care, such as may be found in specialized equine neonatal intensive care units. The key to the success of even the most basic intensive care is the availability of personnel with some nursing skills. In many diseases, foals require support for 1 to 3 days without a great degree of sophistication in resources. The important aspects of low-level intensive care in a practice environment are as follows.

Temperature Control of Environment

It is metabolically costly for a sick foal to expend energy in thermoregulation. On many stud farms, foals are kept in cold environments. The simple process of warming the foal and ensuring that environmental temperatures are in the range 25 to 30°C (77-86°F) will be beneficial.

Feeding the Foal

Nursing care to assist the foal to suck from the mare, bottle feeding, or, in some sick foals, nasogastric intubation is an important component of intensive care. Some of the important points in feeding a sick foal are discussed later in this chapter and in Chapter 18.

Assessment of Passive Immunity and Plasma Transfusion

Failure of passive transfer with resulting low IgG levels in the serum or plasma of newborn foals is one of the most common predisposing factors to infectious diseases in foals. For this reason, it is important to measure the IgG concentration in the plasma of foals admitted to a critical-care facility. Where the IgG concentration is low (<8 g/L, or <800 mg/dL), plasma transfusion is the treatment of choice. For this reason, a bank of frozen plasma should be maintained.

A Simple Facility for Oxygen Administration

Intranasal oxygen administration is easily performed with a cylinder of oxygen and a water bottle for humidification of the gas, as shown in Figure 9-6. In foals with respiratory distress, where the oxygen tension is critical, positioning in sternal rather than lateral recumbency should be done. This will optimize the arterial oxygen tensions. Premature foals often will have hypoxemia (Fig. 9-8), and intranasal oxygen administration at flow rates of 5 to 10 L/min will be beneficial in elevating arterial oxygen tensions (Fig. 9-9).

Basic Blood Chemistry

Because foals require attention over a 24-hour period, usually at inconvenient hours, it is important for the practice that is involved in neonatal intensive care to be able to perform total protein (refractometer) and hematocrit (microhematocrit) measurements. Additional measurements that are useful during intensive care of foals include plasma glucose, plasma or serum electrolytes, blood gases, and acid-base measurements. Some

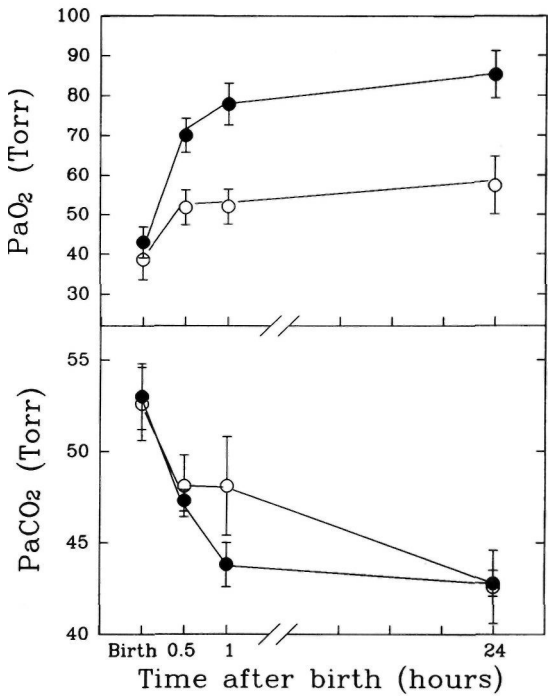


Figure 9-8. Arterial oxygen (Pao_2) and carbon dioxide ($Paco_2$) tensions in full-term (•) and premature (o) foals in the first 24 hours after birth. Note the lower arterial oxygen tensions in the premature foals. (From Rose, R. J., Rossdale, P. D., and Leadon, D. P.: Blood gas and acid-base status in spontaneously delivered, term induced and induced premature foals. *J. Reprod. Fertil. Suppl.* 32:521, 1982, with permission.)

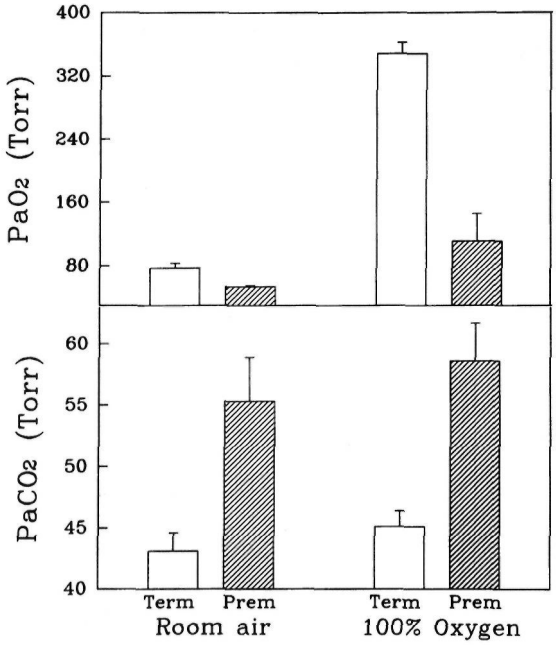


Figure 9-9. Arterial oxygen (Pao_2) and carbon dioxide ($Paco_2$) tensions in term and premature foals breathing either room air or 100% oxygen. (From Rose, R. J., Hodgson, D. R., Leadon, D. P., and Rossdale, P. D.: The effect of intranasal oxygen administration on arterial blood gases and acid-base parameters in spontaneously delivered, term induced and induced premature foals. *Res. Vet. Sci.* 34:159, 1983, with permission.)

of the equipment necessary, for example, for glucose and bicarbonate measurements is not expensive and could be cost effective within a practice.

NEONATAL THERAPEUTICS

Foals are not simply little horses, and appropriate adjustments to therapy need to be made when considering drug selection and dosage regimens. This is particularly important during the first week of life. It is best to avoid the anti-inflammatory drugs in young foals (<2 weeks of age), which carry the risk of gastrointestinal ulceration. There are a few important general considerations that will affect the approach to drug therapy in foals:

- Foals have a larger extracellular fluid volume than older horses and less body fat.
- There may be an increase in the permeability of the blood-brain barrier.
- The consequences are a larger volume of distribution of some drugs and prolongation of action of drugs that require redistribution to fat. For these reasons, barbiturate anesthetics may be best avoided and inhalation anesthetic induction used.
- Alterations in hepatic and renal function may affect the metabolism and distribution of a number of drugs, particularly during the first few days of life. However, most studies have shown that in the case of renal function, the glomerular filtration rate is similar to that in adults within a few days of birth.

In this section on neonatal therapeutics, we will consider two main areas: antibiotic and fluid therapy.

Antibiotic Therapy

The broad principles of antibiotic therapy, as applied to the adult horse, are also true for the foal. However, the use of oral antibiotics is possible in foals, and the mouth is often a convenient route of administration. Important aspects of the major antibiotics used in foals are discussed below. Details of dose rates, frequency, and routes of administration are given in Table 9-1.

PENICILLIN G

This is the drug of choice to treat infections caused by gram-positive bacteria and most anaerobes. It is bactericidal and relatively inexpensive. Penicillin is available in a short-acting form as sodium or potassium salt, which may be given IV or IM (Treatment Nos. 84 and 85). Alternatively, procaine penicillin can be used to obtain more prolonged blood and tissue concentrations. For details of brand names, see Treatment No. 84. Dose rates are given in Table 9-1.

SEMISYNTHETIC PENICILLINS—AMPICILLIN AND AMOXICILLIN

The "broad-spectrum" penicillins have a similar range of activity against gram-positive pathogens but are less effective than penicillin G. These antibiotics are effective against some gram-negative foal pathogens, but the range is quite limited. Therefore, if a gram-negative infection is suspected, ampicillin or amoxicillin is not the drug of choice.

KEY POINT

The greatest value of amoxicillin is that it can be administered orally in syrup form and therefore injections can be avoided.

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TABLE 9-1. Dose Rates of Commonly Used Antibiotics in Foals

Antibiotic	Dose Rate	Route	Frequency
Penicillin—Na or K	20,000-40,000 IU/kg	IV, IM	6 hourly
Penicillin—procaine	15,000 IU/kg or 15 mg/kg	IM only	12 hourly
Ampicillin—sodium	15 mg/kg	IV, IM	6 hourly
Amoxicillin—trihydrate	25 mg/kg	PO only	8 hourly
Cloxacillin sodium	25 mg/kg	IV only	6 hourly
Gentamicin sulfate	3.3-6.6 mg/kg	IV, IM	12-24 hourly
Kanamycin sulfate	7-10 mg/kg	IV, IM	8-12 hourly
Neomycin sulfate	5-10 mg/kg	IM	12 hourly
Amikacin sulfate	7-10 mg/kg	IV, IM	12 hourly
Erythromycin—ethylsuccinate	25 mg/kg	PO only	8 hourly
Erythromycin—gluceptate	5 mg/kg	IV only	6 hourly
Trimethoprim—sulfonamide	15-30 mg/kg of combined drugs	IV, PO (higher dose for PO)	12 hourly
Rifampin	5-10 mg/kg	PO only	12 hourly
Cephalothin	18 mg/kg	IV, IM	8 hourly
Cefazolin	12 mg/kg	IV only	8 hourly
Cefotaxime	15-25 mg/kg	IV, IM	8-12 hourly
Ceftiofur	2.2 mg/kg	IV, IM	12-24 hourly

This is advantageous if clients administer the antibiotic treatment. Details of dose rate and route of administration are given in Table 9-1.

BETA-LACTAMASE-RESISTANT PENICILLINS

These include cloxacillin, flucloxacillin, and oxacillin. The main indication for these antibiotics, which are very expensive, is treatment of beta-lactamase-producing *Staphylococcus aureus.* Details of dose rate and route of administration are given in Table 9-1.

AMINOGLYCOSIDES—GENTAMICIN, AMIKACIN, KANAMYCIN, AND NEOMYCIN

These antibiotics are effective mainly against gram-negative bacteria.

KEY POINT

Because gram-negative septicemia is relatively common in foals, the aminoglycosides are important in therapy.

Streptomycin was one of the most commonly used aminoglycosides and is still incorporated into many commercial preparations in combination with penicillin. However, streptomycin is active against few, if any, equine pathogens.

KEY POINT

The main aminoglycosides used are gentamicin and amikacin, which are often effective against Pseudomonas, Klebsiella, *and various bacteria from the Enterobacteriaceae family.*

Details of dose rate and route of administration are given in Table 9-1.

TRIMETHOPRIM-SULFONAMIDE COMBINATIONS

These are useful against a range of gram-positive and gram-negative bacteria. At higher dose rates (25-30 mg/kg of the combined ingredients), the combination is bactericidal, but care should be taken because severe toxic enteritis has been reported. Trimethoprim-sulfonamide preparations are well absorbed after oral administration and are available in both powder and paste forms. Details of dose rate and route of administration are given in Table 9-1.

ERYTHROMYCIN AND RIFAMPIN

These are used to treat *Rhodococcus equi* infections. The combination is quite expensive to use and is of limited usefulness with other infections. However, the combination of rifampin and other antibiotics may be of value in the treatment of infections of the growth plate because of good bone penetration and effectiveness against intracellular organisms. *R. equi* treatment often has to be continued for several months. The advantage of erythromycin and rifampin is that they can be administered orally. Details of dose rate and route of administration are given in Table 9-1.

Fluid Therapy

The major reason for intravenous fluid therapy in neonatal foals is diarrhea. Diarrhea can produce severe fluid and electrolyte disturbances in a short time period in foals.

KEY POINT
In the adult horse, approximately two-thirds of the total body weight is water, whereas in the first 1 to 2 weeks of life, the percentage of body weight as water approaches 80%.

This is mainly because of the relatively larger extracellular fluid volume, which can account for more than 40% of the body weight.

KEY POINT
Although the fluid requirements of the neonatal foal approximate 100 mUkg/day (5-6 L for a 50-kg foal), milk consumption is approximately double this.

This volume of milk is necessary for the foal to ingest adequate calories, which have been estimated at 150 to 200 kcal/kg/day depending on the state of the foal's health. Therefore, in the case of maladjustment, infection, or any condition that results in the foal being unable to suck the mare, maintenance fluid requirements are approximately 100 mL/kg/day. If there is diarrhea, an additional 200 to 300 mL/kg/day of electrolyte solution may be required to maintain hydration.

Although oral fluids or fluids administered by stomach tube may be useful in certain circumstances, most clinical conditions require intravenous fluid administration. The site that we find most useful for intravenous catheterization of newborn foals is the cephalic vein (see Chapter 19).

KEY POINT
Where prolonged catheterization is required for fluid administration, great care must be taken in aseptic technique for catheter insertion, and catheters should be replaced every 48 to 72 hours.

Some major theoretical considerations in fluid therapy are discussed in Chapter 19.

ASSESSMENT OF FLUID LOSSES

Clinical examination, taking into account skin turgor, mucous membranes, capillary refill time, heart rate, and pulse quality, will allow broad classification of the dehydration as mild (around 5%), moderate (around 8%), or severe (above 10%).

KEY POINT
Regular body weight measurements permit accurate assessment of acute changes in fluid balance. It is also worthwhile remembering that during the first months of life, foals will gain weight at about 1 kg/day.

Laboratory analyses that are useful in evaluating the extent of dehydration and in assessing the response to therapy include measurement of total plasma protein (TPP), hematocrit (packed cell volume [PCV]), osmolality, and serum or plasma electrolytes. Measurement of plasma bicarbonate concentration provides valuable information concerning the nonrespiratory component of any acid-base disturbance. Simple and inexpensive equipment is available to measure the total carbon dioxide (95% of which is bicarbonate) by titration, and this measurement is performed by most autoanalyzers in commercial laboratories.

KEY POINT
Because foals under metabolic stress can become hypoglycemic, it is important to measure plasma glucose concentrations.

KEY POINT
Care should be taken when interpreting changes in TPP and PCV. Changes in concentrations of the globulins may be the principal reason for a lower or higher than expected TPP, and the PCV may have a wide reference range, similar to the adult horse.

CHOICE OF FLUID

KEY POINT
In most circumstances, replacement fluid is the fluid of choice where there have been losses of fluid and electrolytes such as in diarrhea. Replacement fluids (see Chapter 19) have a similar electrolyte composition to plasma.

- *Maintenance fluids* should be used where the sodium losses do not continue, because the sodium requirements are lower and potassium requirements are higher than in acute volume depletion. This is typically the case after the acute volume deficits have been replaced. Maintenance fluids (see Chapter 19) have sodium and

chloride concentrations in the range of 40 to 50 mmol/L (mEq/L) and potassium in the range of 15 to 20 mmol/L, with 2.5% to 5% dextrose used to make up the osmotic balance.

- *Bicarbonate* often is overused in neonates and should only be given where there is loss of base, such as in severe diarrhea. In many other situations, the acidosis is due to lactate accumulation because of poor tissue perfusion.

KEY POINT

The treatment of choice in this situation is the intravenous administration of replacement fluids to increase peripheral perfusion,

- The lactate will then be metabolized in the liver. If bicarbonate is required, it can be calculated using the formula $HC0_3$ requirement in mmol (mEq) = 0.4 x (27 - measured HCO_3^-) x body weight (kg). Because bicarbonate solutions are hypertonic, it is useful to add bicarbonate to hypotonic fluids, such as 0.45% sodium chloride.
- *Hypertonic fluids* should be avoided in foals.
- *Plasma* administration may be necessary, particularly where there is substantial protein loss, such as in diarrhea. The use of normal electrolyte solutions in these circumstances will result in further hypoproteinemia with resulting movement of fluid out of the vascular compartment. The use of plasma avoids these problems and is also useful for increasing IgG levels in foals with failure of passive transfer of immunity.
- *Oral fluids* have been used with success in the treatment of diarrhea in calves. We have found that the administration of fluid and electrolyte mixtures by stomach tube is useful in foals in which the diarrhea is mild and there are no signs of hypovolemia.

KEY POINT

To enhance absorption in the small intestine, the combination of glucose, glycine, and electrolytes is useful, and commercial preparations are available (Treatment No. 57).

VOLUME AND RATE OF FLUID ADMINISTRATION

- Maintenance requirements are around 100 mL/kg/day, and this volume should be administered over a period of 24 hours.

KEY POINT

In cases of severe hypovolemia, initial flow rates can be in the range 10 to 20 mL/kg/h to restore the central circulation. After improvements in signs of hypovolemia, the flow rate can be slowed.

- It is particularly important in cold climates to warm fluids to body temperature before administration because there is a significant energy cost to the foal, which may already be in a critical energy state.
- Monitoring the flow rate is important during intravenous fluid administration. Apart from hypovolemic shock, fluid should be given over a prolonged period; otherwise, it may be rapidly excreted via the urinary tract.
- The volume of plasma used is in the range 20 to 40 mL/kg. These volumes are usually sufficient to correct IgG deficiencies due to failure of passive transfer.

KEY POINT

If frozen plasma is used, it can be administered after partial thawing because the protein fraction is available during the first part of thawing. This enables the globulins to be increased without administering a large volume of fluid.

Diseases of Foals

Abdominal Pain

KEY POINT

The most common cause of abdominal discomfort or pain in the first few days of life is retained meconium.

A variety of other problems may give rise to abdominal pain, however. It is important to examine foals with abdominal pain carefully and to note whether there is any abdominal distension.

HISTORY AND PRESENTING SIGNS

- Age of foal when first presented
- Presence/absence of abdominal distension
- Change in fecal consistency
- Change in demeanor
- Passage of urine

CLINICAL FINDINGS AND DIAGNOSIS

- A good general examination is critical for determining the cause of the pain.
- As with adult horses, determination of the vital signs (heart rate, respiratory rate, temperature,

capillary refill time) and gut sounds is essential in making an assessment of the extent of the problem.

KEY POINT

If the foal is straining, it should be determined whether it is straining to urinate (legs spread) or straining to defecate (arching of the back).

- Digital rectal examination should be performed to determine if there is retention of meconium.
- Passage of a nasogastric tube should be done to determine whether there is gastric reflux, which may indicate an upper intestinal obstruction.
- Abdominocentesis will assist in determining whether there is a problem that is likely to require surgery (see Chapter 7 for further details). This is most easily done in newborn foals with the foal in lateral recumbency, but it also may be carried out with the foal standing. A 19-gauge, 3.75-cm (1.5-inch) needle is inserted through the midline of the abdominal wall.
- Determination of the acid-base status is essential if compromise of bowel is thought to have occurred. Metabolic acidosis (decreased bicarbonate or total CO_2 concentration) may indicate decreased perfusion of intestine in conditions such as volvulus and intussusception.
- The hemogram and serum or plasma electrolytes may be useful in assessing the requirements for fluids and electrolytes (see Fluid Therapy).
- In more specialized practices, techniques such as endoscopic examination of the stomach, abdominal radiography, and ultrasound are now becoming more widely used. These techniques do require considerable experience before accurate interpretation can be made.

KEY POINT

Cases that undoubtedly require surgery are those with persistent abdominal pain where there is increasing abdominal distension. The degree of abdominal distension can be assessed by sequential recordings of abdominal circumference.

DIFFERENTIAL DIAGNOSIS

- Impaction with meconium
- Atresia ani or coli
- Ruptured urinary bladder
- Obstruction to intestine
- Peritonitis
- Gastric ulceration

TREATMENT

- Because of the wide range of possible conditions that can give rise to abdominal pain, it is essential to narrow the diagnostic possibilities.

KEY POINT

Retained meconium can be treated by the use of enemas, and most cases resolve without complicated treatment.

- Soapy water is quite satisfactory as an enema solution, but there are commercial products available. This can be supplemented by the use of mineral oil (500 mL) given by nasogastric tube.
- Exploratory laparotomy is not often necessary in newborn foals but should be considered if there is severe unremitting pain together with increasing abdominal distension.
- Analgesics should be used judiciously.

KEY POINT

The nonsteroidal anti-inflammatory drugs have considerable potential to cause gastrointestinal ulceration. They should be used at low dose rates and for short periods.

- Flunixin (Treatment No. 52) is the anti-inflammatory drug of choice, and the dose rate should be in the range of 0.2 to 0.5 mg/kg. Although some adverse effects have been reported with xylazine (particularly hypotension), we have had good results from dose rates in the range of 0.1 to 0.2 mg/kg given IV (Treatment No. 109).

Angular Limb Deformities

See Chapter 4.

Combined Immunodeficiency

Combined immunodeficiency (CID) is a hereditary disorder principally of Arabian foals.

KEY POINT

The condition results in failure of production of both T and B lymphocytes.

It is an autosomal recessive trait, and therefore the incidence of clinical disease is low, but the incidence of carriers may be quite high.

HISTORY AND PRESENTING SIGNS

- Presented usually around 1 to 3 months of age
- Nasal discharge and/or cough
- Recurring respiratory infections

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CLINICAL FINDINGS AND DIAGNOSIS

- Because foals have passive immunity from colostrum, it is not until the IgG levels decline around 2 to 3 months of age that signs of the problem appear.
- The most common presenting problems are respiratory infections that may be viral or bacterial. Adenovirus infections, which are nonpathogenic in normal foals, will cause a severe pneumonia in foals with CID. A range of other bacterial and viral infections also can be found in foals with CID.
- CID should be one of the differential diagnoses in any Arabian foal presented with signs of infection.

KEY POINT

Hematology is helpful diagnostically, and the principal finding is severe lymphopenia.

- A persistently low lymphocyte count of less than 1.0 X 10^9/L (1000/μL) is usually considered strongly supportive of a diagnosis of CID. However, in some normal foals with severe infections, the lymphocyte count also may be low. Therefore, serial hemograms are useful, and in most foals with CID, the lymphocyte counts may be less than 0.5 X 10^9/L (500/|xL).
- Determination of IgM concentrations after 1 month of age also supports a diagnosis of CID because foals with the disorder will have no or very little IgM. IgM will be absent from the serum of affected foals before drinking colostrum.
- A positive diagnosis of CID can only be made at necropsy after histopathologic examination of the thymus, lymph nodes, and spleen.

DIFFERENTIAL DIAGNOSIS

- Septicemia
- Severe bacterial or viral infections

TREATMENT

- Although bone marrow transplantation has been performed experimentally, there is no other treatment that is worthwhile.
- Because the condition is hereditary, identification of carrier animals is important. Unfortunately, under most circumstances, this is only possible from the results of matings.

Diarrhea

KEY POINT

Diarrhea is a very common problem in foals and in most cases is self-limiting.

This is important because many cases are treated with a variety of medications that are unnecessary. Because substantial fluid and electrolyte deficits can develop quickly, it is important to assess the hydration state and administer appropriate fluids. Diarrhea is a common finding in foals with septicemia and in more localized enteritis/colitis.

KEY POINT

"Foal heat" diarrhea is a transient problem usually found between 5 and 10 days of age and seems to represent changes in intestinal function and bacterial flora.

HISTORY AND PRESENTING SIGNS

- May be signs of systemic disease
- Variable volume and consistency of diarrhea

CLINICAL FINDINGS AND DIAGNOSIS

- Careful clinical examination is important to determine whether there is systemic disease or metabolic derangements associated with the diarrhea. Factors such as temperature change, prolongation of capillary refill time, poor pulse quality, and elevated heart rate may indicate not only substantial fluid loss but also septicemia.

KEY POINT

Routine hematology and fibrinogen and electrolyte measurements should be performed to assess the possibility of systemic disease and to determine electrolyte alterations.

- Because diarrhea is a common presenting sign in foals with septicemia, blood cultures may be indicated.
- Fecal culture may be indicated if there is suspicion of a bacterial enteritis. It is necessary to specify to the laboratory the range of likely pathogenic bacteria.
- If rotavirus is suspected, commercial testing kits are available (Rotazyme, Abbott Laboratories, Abbott Park, IL; Rotatest, Wampole Laboratories, Cranbury, NJ).

DIFFERENTIAL DIAGNOSIS

- "Foal heat" diarrhea—foals usually healthy with a "pastey" diarrhea
- Rotavirus diarrhea—watery diarrhea with mild systemic signs and a number of foals affected
- Bacterial diarrhea (e.g., *Escherichia coli, Clostridia, R. equi,* and *Salmonella* spp.)

- Nutritional diarrhea—overfeeding, orphaned foals
- Antibiotic-related diarrhea—usually from broad-spectrum oral antibiotics
- Parasitic diarrhea—confirm by fecal flotation

TREATMENT

KEY POINT

Maintenance of hydration is the critical factor in management of foal diarrhea.

- In cases where foals are not systemically ill, provision of adequate milk or, in older foals, drinking water may be all that is required.
- As with calves, oral rehydration fluids (see Fluid Therapy, Chapter 19) may be used, administered by nasogastric tube. Combinations of glucose-glycine and electrolyte (Treatment No. 57) are most effective in enhancing electrolyte and fluid uptake in the small intestine. Volumes up to 15 to 20 mL/kg may be administered in a single dose by nasogastric tube.
- Intravenous fluids are necessary in foals that are assessed as more than 8% dehydrated. These foals usually have lost large amounts of bicarbonate and may have a severe metabolic acidosis. Bicarbonate solutions should be considered when the blood bicarbonate concentrations decrease to less than 15 mmol/L (mEq/L). For prolonged IV fluid therapy, we find the best site for catheter placement is the cephalic vein. With the use of flexible tubing suspended in the stall, the foal can have unrestricted movement and access to the mare.

KEY POINT

Antibiotics should be avoided unless there are systemic signs or a strong suspicion that septicemia may be a problem.

- The use of antibiotics may produce diarrhea. However, because of the possibility of septicemia (gram-negative organisms in particular), it may be necessary to cover this possibility after a blood culture has been taken. The combination of Na or K penicillin (Treatment Nos. 85 and 86; 20,000 IU/kg q6h) and gentamicin (Treatment No. 56; 3.3 mg/kg q12h) or amikacin (Treatment No. 4; 7-10 mg/kg q12h) is the first choice.
- In foals with severe diarrhea, there is also hypoproteinemia, and therefore plasma transfusions may be useful. Intravenous plasma at 20 to 40 mL/kg will help to restore plasma oncotic pressure and boost the immunoglobulin concentrations.
- In most foals with diarrhea, there is no need to remove the foal from the mare. However, if the diarrhea does not respond to standard treatments, it may be useful to restrict milk intake for a 24-hour period and only allow clear fluids.

Failure of Passive Transfer of Immunoglobulins

Although the foal has some immunologic function as a fetus, immunity is essentially a passive transfer of colostral immunoglobulins during the first 6 to 12 hours after birth. Failure of passive transfer of immunoglobulins is quite common in newborn foals, and therefore, assessment of IgG concentrations in plasma is important in management.

KEY POINT

The volume of colostrum normally consumed by newborn foals in the first 8 to 12 hours has been estimated at between 1 and 2 L.

It also should be noted that alveolar macrophage function is substantially reduced in foals compared with adults.

KEY POINT

The optimal levels of IgG in foal plasma 24 hours after birth are greater than 8 g/L (800 mg/dL).

Values less than 4 g/L are considered to reflect failure of passive transfer, and those between 4 and 8 g/L are suboptimal concentrations. Foals with low IgG concentrations are more likely to be predisposed to a variety of bacterial infections.

HISTORY AND PRESENTING SIGNS

- Mare with a very distended udder, indicating foal has not sucked
- Foal with suspected septicemia
- Foal presented with "joint ill"
- Maladaption

CLINICAL FINDINGS AND DIAGNOSIS

- The availability of quick, reliable measurements of IgG concentrations in foal plasma has enabled early diagnosis of failure of passive transfer, before a foal is presented with an infection.

KEY POINT

A number of techniques are available for measuring immunoglobulin concentrations.

- These vary from the least expensive and nonspecific (zinc sulfate turbidity and glutaraldehyde

coagulation tests) to the more recent CITE test (Idexx, Portland, ME), which enables classification of IgG levels into several ranges, from 2 to 8 g/L (200-800 mg/dL). The latter measurement is the technique of choice for assessment of IgG concentrations, although the test is relatively expensive.
- Measurement of the colostral specific gravity in mares provides a good guide to the likely IgG concentrations in their foals. Colostral specific gravities greater than 1.06 generally indicate adequate antibody concentrations.

DIFFERENTIAL DIAGNOSIS

- Neonatal maladjustment syndrome

TREATMENT

- Maintenance of a colostral bank is vital on a large stud farm so that foals with failure of passive transfer or that are likely to have inadequate IgG concentrations can be given supplementation of colostrum. In most cases, around 1 L of colostrum should be given within the first 8 to 12 hours after birth.
- If colostrum is not available or if the foal is older than 24 hours, plasma transfusions are required to increase the IgG concentrations. Normally, 20 to 40 mL/kg is required to increase the IgG levels by approximately 2 to 4 g/L (200-400 mg/dL). Further details are provided in the section Fluid Therapy.

Gastroduodenal Ulcers

KEY POINT

This appears to be a relatively new disease for which stress plays an important role in the pathogenesis.

The precise etiology is unknown, with some investigators proposing a role for various bacteria and viruses such as rotavirus. Where there is primary gastric ulceration, the clinical signs are not as severe as those involving both the stomach and duodenum.

HISTORY AND PRESENTING SIGNS

- Teeth grinding
- Excessive salivation
- Pain manifested by the foal lying in dorsal recumbency
- Decreased appetite
- Pain after feeding

CLINICAL FINDINGS AND DIAGNOSIS

- Clinicians are alerted to the possibility of gastroduodenal ulcers by the classical presenting signs, detailed above.
- Examination of gastric reflux is helpful to determine the presence of blood. Many foals will show considerable discomfort when the nasogastric tube is passed through the distal esophagus.
- If an appropriate endoscope is available (8-10 mm diameter, 180 cm long), direct examination of the stomach is useful. This is done after 3 to 4 hours of feed restriction by passing the endoscope via the ventral nasal meatus into the stomach and slightly distending it with air.
- In severe cases of gastric ulceration, there may be gastric rupture. In such cases, the foal will show signs of severe colic and shock, and its condition will deteriorate rapidly.

DIFFERENTIAL DIAGNOSIS

- Septicemia
- Peritonitis
- Intestinal obstruction

TREATMENT

- If there is gastric distension, refluxing via a nasogastric tube should be done.

KEY POINT

A number of H_2 antagonists have been used and found to be clinically useful, although no double-blind trials of efficacy have been undertaken.

- The two most widely used H_2 antagonists are cimetidine (Treatment No. 26) at dose rates of 6 to 8 mg/kg orally every 6 to 8 hours or ranitidine (Treatment No. 100) given orally at dose rates of 6 to 8 mg/kg every 8 hours. Both drugs also may be administered IV, at dose rates similar to those used for oral treatment.

KEY POINT

A variety of gastric protectants have been found to be useful, and the most commonly used is sucralfate (Treatment No. 102) at a dose of 30 mg/kg given orally every 6 to 8 hours.

- In some foals, there may be partial or complete obstruction at the level of the pylorus due to stricture. In such cases, surgical treatment is

necessary to bypass the stricture. This type of procedure is best done at a specialist clinic.

Infection/Septicemia/Pneumonia

Bacterial infection is probably the most important clinical disorder in foals. Infection may be localized, but in most cases it is generalized, with subsequent localization to areas such as the lung, joints, and physes. Infections may be acquired in utero but in most cases are acquired after birth. The most important predisposing cause of infection is failure of passive transfer of immunity.

KEY POINT

It is an important part of management on stud farms to screen the colostrum IgG concentrations and/or IgG level in the foal in the first 6 to 12 hours after birth.

HISTORY AND PRESENTING SIGNS

- Previous history of abortion or death of foals
- Overcrowding/poor management
- Chronic long-standing infectious disease (e.g., *Rhodococcus equi, Salmonella)*
- Dystocia
- Premature foal
- Depressed, sick foal presented

CLINICAL FINDINGS AND DIAGNOSIS

KEY POINT

The most important signs of septicemia are behavioral.

- Affected foals will appear depressed and stop sucking. The first thing noticed by the stud-farm manager or owner may be that the mare has a distended udder. Because foals infected in utero may show signs similar to those with neonatal maladjustment syndrome, it is important to investigate sepsis as a possible cause of any foal with maladaptation.
- Body temperature provides no accurate guide to the presence or absence of infection, with normal, increased, and decreased temperatures being reported.
- There may be an increase in respiratory rate, but even in cases of pneumonia, abnormal lung sounds may not be heard, unlike the situation in adult horses.
- Diarrhea is a common finding in foals with septicemia.
- Sudden onset of lameness is an important sign, which may suggest localization of infection in a joint or physis.

KEY POINT

Hematology is a vital diagnostic aid, but total white cell count does not appear to be a good indicator of infection.

- The most consistent findings are increased band forms of neutrophils (normally not present or in very low numbers in normal foals) and toxic changes within the neutrophils.
- Fibrinogen often is increased in foals with septicemia, and values are usually greater than 5 g/L (500 mg/dL). However, there is usually a delay of 48 hours before the fibrinogen increases.
- Hypoglycemia is a common finding in septicemia, with values less than 5 mmol/L (90 mg/dL).
- Because many foals with septicemia have pneumonia, hypoxemia is common and should be anticipated even if access to a blood gas machine is not possible.

KEY POINT

Blood cultures are essential (see Diagnostic Aids and Chapter 17), and blood samples should be collected using impeccable sterile technique.

- It is vital to take blood for blood culture before beginning antibiotic therapy. It also should be noted that because some bacteria take up to 5 to 6 days to grow, a negative blood culture result should not be given too quickly.
- Transtracheal aspirates for bacteriology and cytology also may be indicated because a high percentage of cases will have pneumonia.
- Chest radiographs also are useful in assessing the degree of pulmonary compromise. Atelectasis is common in foals with pneumonia, and the lung fields will have an increase in density.

DIFFERENTIAL DIAGNOSIS

- Combined immunodeficiency
- Neonatal maladjustment syndrome

TREATMENT

KEY POINT

The combination of penicillin (sodium or potassium; Treatment Nos. 85 and 86) given TV at a dose rate of 20,000 IU/kg q6h and TV gentamicin (3.3 mg/kg ql2h; Treatment No. 56) or amikacin (7-10 mg/kg q12h; Treatment No. 4) appears to be the treatment of choice while waiting for culture results.

- The duration of antibiotic treatment should be based on clinical response and subsequent nanograms. In some cases, 5 to 7 days is adequate, whereas others may require up to 2 weeks of treatment.
- The most common sequelae of septicemia are septic arthritis or septic physitis, enteritis, and, in foals that are recumbent for some time, corneal ulceration.

"Joint Ill" (Polyarticular Septic Arthritis/ Osteomyelitis)

"Joint ill" is a common term used for polyarticular septic arthritis in foals, usually after a systemic bacterial infection/septicemia. Most commonly, infection involves multiple larger joints and/or the physes, but in some cases it may involve the metaphysis adjacent to the physis. Joint ill is a serious condition that carries a poor prognosis for future athletic function.

HISTORY AND PRESENTING SIGNS

- Prematurity
- Failure of passive transfer
- Mare with uterine infection
- Poor hygiene at foaling

CLINICAL FINDINGS AND DIAGNOSIS

- Foals with joint ill are often clinically ill and will show signs of systemic infection, with depression, inappetence, and fever.

KEY POINT

It should not be assumed that a foal does not have joint ill just because it has no signs of systemic disease.

- Foals are often reluctant to move and will show variable signs of lameness.
- Careful examination will usually reveal joint effusion, with one or more joints being distended with fluid and showing thickening of the dorsal joint capsule.
- Radiographs may not show changes in the early stage of disease but are useful to help in assessing the prognosis. One of the difficulties in radiographic interpretation of bone infection is that the bone around the physis often has a similar appearance to infected bone.

KEY POINT

Synovial fluid collection is essential for diagnosis and should be performed before any antibiotic therapy is instituted.

- Details of collection and sample handling for synovial fluid are given in Chapters 4 and 17. It is critical that the aspiration of synovial fluid be done in a sterile manner, with a cap, mask, and gloves worn by the operator and sterile drapes used around the site. We find it easy to collect samples with the foal given a gaseous anesthetic induction if the foal is less than 2 weeks old. Samples should be collected into tubes containing EDTA for a total and differential nucleated cell count. Synovial fluid nucleated cell counts greater than 10 X 107L (10,000/μL) with a predominance of neutrophils and an elevated total protein (reference range 15-25 g/L, 1.5-2.5 mg/dL) suggest infection. For bacteriology, it is best if samples are collected and submitted in a new sterile disposable syringe, after which the sample is centrifuged and the deposit placed into enrichment media.
- It may be worthwhile to take blood for blood culture early in the course of the disease.

DIFFERENTIAL DIAGNOSIS

- Trauma
- Nonseptic joint effusion

TREATMENT

- It is important to obtain samples for bacteriology before beginning any therapy.
- If a Gram stain of the synovial fluid deposit reveals bacteria, antibiotic therapy can be based on the result of the Gram stain.
- If there are no Gram stain results, the use of a bactericidal combination of drugs such as IM penicillin (15,000 IU/kg, or 15 mg/kg; Treatment No. 84) and IV gentamicin (3.3 mg/kg; Treatment No. 56) given twice daily is appropriate.
- Irrigation of infected joints by joint lavage (see Septic Arthritis, Chapter 4) is worthwhile using polyionic fluid. This should be done with the foal under general anesthesia.

Neonatal Isoerythrolysis

Neonatal isoerythrolysis is a condition found most commonly in Thoroughbreds when during pregnancy the mare manufactures antibodies against the foal's red cells. After birth, ingestion of colostrum by the foal results in absorption of the anti-red cell antibodies, which destroy the foal's red cells.

KEY POINT

The condition is found mainly in multiparous mares and is associated with production of antibodies against specific erythrocyte antigens, usually Aa and Qa.

HISTORY AND PRESENTING SIGNS

- Multiparous mare
- Foal shows weakness within first few days of birth

CLINICAL FINDINGS AND DIAGNOSIS

- Foals are normal at birth and for the first 12 to 72 hours after birth.
- The onset of clinical signs is variable and depends on the amount of antibody ingested and absorbed.
- Foals usually show signs of listlessness and decreased appetite.

KEY POINT

Clinical examination reveals severe jaundice of the mucous membranes and elevated heart and respiratory rates. The temperature is normal.

- Hematologic study shows very low red cell values, with the PCV usually in the range 0.10 to 0.20 L/L (10-20%), and icteric plasma. An increase in the white cell count is also common and may be a stress-related response. In most foals that show clinical signs, the PCV is usually below 0.10 L/L (10%).
- Urine samples will demonstrate hemoglobinuria.

DIFFERENTIAL DIAGNOSIS

- Equine herpesvirus
- Neonatal septicemia
- Tyzzer's disease
- Rupture of the bladder

TREATMENT

- Prevention is the most important aspect of management, and mares that have lost foals within a few days of birth should be suspected. Blood samples can be taken for determining the presence of Qa and Aa antibodies. If the mare does not have these red cell antigens (Qa or Aa negative), the risk of producing antibodies likely to cause neonatal isoerythrolysis is increased.
- If there is a likelihood of neonatal isoerythrolysis, colostrum from the mare should be discarded and colostrum from a colostrum bank administered. The foal can be muzzled for at least 48 hours to prevent anti-red cell antibodies.

KEY POINT

Treatment of an affected foal involves a blood transfusion with administration of the mare's blood that has had the red cells washed three or four times.

- We have had good cooperation from local blood banks. Alternatively, cross-matching may be carried out and a suitable donor found. If facilities are not available to do this, a transfusion from a gelding is unlikely to cause a transfusion reaction. Usually 20 to 40 mL/kg (or 1-2 L) of whole blood is adequate to increase the PCV approximately 10 percentage points, and this is satisfactory for the foal to survive until further red cells are manufactured. If the mare's red cells have been washed, packed red cells are returned after centrifugation. If a gelding's blood is to be used, most of the plasma can be removed before the transfusion.
- It is important to perform an IgG evaluation to determine whether there has been failure of passive transfer of immunity. Many foals with neonatal isoerythrolysis survive only because they absorb inadequate IgG. Therefore, plasma transfusion may be necessary, and the foal should be covered for 3 to 5 days with a bactericidal broad-spectrum antibiotic combination such as procaine penicillin (Treatment No. 84; 15,000 IU/kg, or 15 mg/kg q12h) IM and gentamicin (Treatment No. 56; 3.3 mg/kg q12h) IV.

Neonatal Maladjustment Syndrome

KEY POINT

Neonatal maladjustment syndrome is really a descriptive term that may encompass a number of conditions resulting in maladaptation in the newborn foal.

The condition appears to be related to central nervous system dysfunction and may be induced by birth asphyxia or intracranial hemorrhage. It is important to distinguish maladjusted foals from septicemic foals.

HISTORY AND PRESENTING SIGNS

- Dystocia in some cases
- Most foals affected are not premature.
- Signs usually noticed within 24 hours of birth

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CLINICAL FINDINGS AND DIAGNOSIS

- Foals will stop sucking and show a progression of disease from depression and weakness through to wandering behavior, loss of recognition of the mother, and finally recumbency.
- Foals may have a grunting respiration and make an abnormal sound. For this reason, they have been termed "barker foals" by some clinicians.
- Neurologic signs can include rigidity of fore- and hindlegs, apparent blindness, and convulsions.
- Most foals will demonstrate some degree of dyspnea, and it is important to eliminate a primary respiratory complaint.
- Blood cultures may be necessary to eliminate the possibility of septicemia.

DIFFERENTIAL DIAGNOSIS

- Septicemia
- Blood loss (via umbilical rupture during birth)
- Metabolic disorders

TREATMENT

- Nursing care is the key to management, ensuring that metabolic and hydration status is normal.
- Some foals will be hypoxemic and may require intranasal oxygen administration.
- No specific treatment is useful, although it is essential to ensure that foals are not septicemic.
- Foals that have reversible disease usually show improvement within the first 3 to 5 days.
- Benefit may also be gained from administration of IV dimethyl sulfoxide (Treatment No. 34) at a dose rate of 0.5-1.0 g/kg as a 10% solution IV.

Prematurity

Although premature foals are usually considered to be those born at less than 325 days of gestation, Dr. Peter Rossdale, in Newmarket, U.K., has highlighted the importance of "readiness for birth." That is, a foal may be full term in gestational length but still unready to be born and show some of the signs of prematurity. Premature foals may have dysfunction in a number of body systems and require careful management. For a successful outcome, many of these foals require full-time intensive care for several weeks, and economic factors are of major importance in decisions about treatment.

HISTORY AND PRESENTING SIGNS

- Mare with vulval discharge
- Mare showing signs of dripping milk from the udder
- Weak foal, unable to stand and suck within 1 to 2 hours of birth

CLINICAL FINDINGS AND DIAGNOSIS

KEY POINT

Premature foals are usually small and are generally weak, being unable to get to their feet and suck within a few hours of birth.

- The hair coat is often short and may have a silky appearance, particularly over the rump.

KEY POINT

Most premature foals will have an exaggerated range of fetlock movement, there being overextension of the joints. This results in the fetlocks sinking toward the ground.

- Many premature foals are delivered because of placental insufficiency. These foals have a better prognosis for survival than those delivered prematurely because of infection.
- A number of body systems may not be functioning normally:
  - **Adrenal Dysfunction**—Can be recognized on routine hematology because the neutrophil-to-lymphocyte ratio is less than 1.5:1. A low white cell count ($<5.0 \times 10^9$/L, i.e., 5000 cells/pi) is an unfavorable sign.
  - **Pulmonary Problems**—Resulting in some cases from surfactant deficiency and in others because of in utero infection will cause hypoxemia and hypercapnia.
  - **Musculoskeletal Immaturity**—Is often the major reason why these foals are not viable in the long term. Tarsal and carpal collapse and angular deviations are common, particularly if foals are not restricted and their limbs supported. Radiographs should be taken of the carpi and tarsi to evaluate skeletal maturity.
  - **Infection**—Is a common problem in premature foals, and blood cultures and transtracheal aspirates should be considered. Plasma fibrinogen concentrations should be measured, and values over 5g/L (500 mg/dL) indicate an inflammatory focus.

DIFFERENTIAL DIAGNOSIS

- Neonatal maladjustment syndrome
- Septicemia

TREATMENT

- Complete evaluation of the main body systems is necessary to identify the problem areas. Infec-

tion should not be ruled out until there is supporting hematologic evidence and negative blood cultures.
- Antibiotic prophylaxis may be worthwhile, and a combination of penicillin and an aminoglycoside is most appropriate while waiting for culture results. For dose rates, see Antibiotic Therapy.

KEY POINT
Hypoxemia and hypercapnia are common in premature foals, indicating ventilation-perfusion mismatch and alveolar hypoventilation.

- It is important for premature foals to be positioned in sternal recumbency, and intranasal oxygen administration is often worthwhile if the hypoxemia is severe (Pao_2 < 60 mm Hg).
- Exercise and weight bearing should be restricted because of the excessive loads on the immature skeleton. Taping wooden tongue depressors to the heels as extensions to the feet may be useful in preventing overextension of the fetlocks.

Rhodococcus Equi Infection ("Rattles")

R. equi (formerly *Corynebacterium equi)* is an important cause of infection in foals aged 1 to 3 months.

KEY POINT
Infection may be acquired via the alimentary or respiratory routes, and there may be localization of infection in several sites, including joints.

The exact factors that lead to infection in foals (but not adults) are incompletely understood. Reduced activity of the alveolar macrophages has been reported, and there may be factors in local gut immunity that give rise to infection. *R. equi* infections can cause extensive morbidity and mortality on some stud farms, although the problem tends to be found only in dry climates and where there are sandy soils. The infection is common in California, the southern part of the United States, and Australia.

HISTORY AND PRESENTING SIGNS

- Foal aged 1 to 3 months
- Signs of respiratory or alimentary disease

CLINICAL FINDINGS AND DIAGNOSIS

- Foals usually appear depressed and have a decreased appetite. The mare will often have a distended udder.
- Involvement of the respiratory tract is the most common form of the disease, and foals will often have a productive cough.
- Dyspnea is usually evident, and auscultation of the chest will reveal gurgling sounds over the hilar region.
- Variable degrees of fever are found, and there are typically exacerbations and remissions.
- Diarrhea is often a feature of the gastrointestinal form of the disease.
- Most stud-farm owners are adept at recognizing *R. equi* infections, but positive confirmation is only possible by demonstration of the typical appearance of the organism on a Gram stain from a transtracheal aspirate. However, in very ill foals, a transtracheal aspirate may result in further debility or death. Blood cultures may be useful if there are systemic signs, although it often requires up to 5 days of incubation for the bacteria to grow.
- Chest radiographs can be valuable in assessing the extent of abscesses. *R. equi* typically causes multiple abscesses throughout the lungs and various abdominal lymph nodes. More recently, ultrasound examination using a rectal probe has proven a useful and reliable method for early diagnosis of pulmonary abscesses in foals with *R. equi* infection. This is a simple and rapid method for assessing the parietal surface of the lungs, and in many cases a diagnosis is made with treatment commenced before foals become debilitated.

DIFFERENTIAL DIAGNOSIS

- Bacterial pneumonia
- "Foal heat" diarrhea
- Bacterial diarrhea
- Septicemia

TREATMENT

KEY POINT
Prolonged antibiotic treatment for 2 to 3 months is essential, and even in cases with severe pulmonary abscesses, complete recovery is possible.

- The antibiotics of choice are erythromycin (Treatment Nos. 38 and 39) and rifampin (Treatment No. 101). These are available in forms that can be administered orally. Erythromycin ethylsuccinate is given orally at 25 mg/kg every 8 hours, and rifampin is administered at a dose rate of 5 to 10 mg/kg every 12 hours. This treatment should be continued for 2 to 3 months to enable complete resolution of the infection.

Obviously, this treatment is very costly and only worthwhile in valuable foals.
- In foals in which cost precludes the use of erythromycin and rifampin, we have had some good response to the use of neomycin at 5 mg/kg every 12 hours given IV or IM. Neomycin is slightly more nephrotoxic than the other aminoglycosides but does not cause major renal dysfunction in most foals if administered over 10 to 14 days.
- Some experimental studies have shown the protective effects of hyperimmune plasma administered prophylactically to foals. The hyperimmune plasma is prepared by administering an *R. equi* vaccine to horses that will be used as plasma donors to obtain an increase in specific antibodies. Plasma is then administered prophylactically to foals after birth. On stud farms where *R. equi* is endemic, such a program may be worthwhile.

Seizures

See Chapter 14.

Urachal Problems

The urachus is the normal communication pathway between the bladder and the allantois during fetal life. After the umbilical cord ruptures, the urachus may remain open or reopen several days after birth. It also may be the site of infection, and some cases develop abscessation.

HISTORY AND PRESENTING SIGNS

- Foal usually less than 1 week of age
- Swelling around the umbilicus
- Urine dribbling from umbilicus

CLINICAL FINDINGS AND DIAGNOSIS

- Infection around the urachus will usually be manifested as swelling around the umbilicus.
- Patent urachus is simple to diagnose because of obvious urine leakage from the umbilical stump.

DIFFERENTIAL DIAGNOSIS

- Rupture of the urinary bladder or intraabdominal urachus

TREATMENT

- Many cases of patent urachus will resolve without any treatment.

KEY POINT

If the urachal opening does not close within 2 to 3 days, we have found that a caustic agent such as phenol or strong tincture of iodine, applied carefully around the opening with a cotton bud, will result in closure of the urachus.

- If there is infection, surgical treatment is necessary to remove the infected umbilical stump.

Urinary Tract Disruption

Urinary tract disruptions occur relatively frequently in newborn foals. Rupture of the urinary bladder and the abdominal urachus can occur, resulting in uroperitoneum. Mostly this occurs during delivery, and the signs are manifested within a few days of birth. A small percentage of cases can occur from an infected umbilical stump.

HISTORY AND PRESENTING SIGNS

- More common in male foals
- Small amounts of urine passed
- Abdominal distension
- Foal 2 to 3 days of age

CLINICAL FINDINGS AND DIAGNOSIS

- Foals usually become depressed and stop sucking.
- By the time signs of depression are seen, there is usually significant abdominal distension.

KEY POINT

Straining to urinate is often a sign, together with passage of small volumes of urine and progressive distension of the abdomen.

- Serum biochemistry profiles often show lower Na^+ and Cl^- values together with increased K^+ and urea. A mild metabolic acidosis is commonly found on acid-base assessment.

KEY POINT

Abdominocentesis is the critical technique for the diagnosis of uroperitoneum.

- With the foal standing or in lateral recumbency, a 19-gauge, 25-mm (1-inch) needle is inserted through the abdominal midline just caudal to the umbilicus.

KEY POINT

The sample may smell like urine, and the creatinine concentration will be elevated to

more than twice the concentration in the serum or plasma.

- It is worthwhile measuring the blood leukocyte count and the total protein in case there is urinary tract infection.
- If a sterile dye is available (methylene blue is the dye of choice), a small amount (5-10 mL) can be injected into the bladder via a urinary catheter and abdominal fluid later taken to determine if the dye is present. This technique rarely is used, and in most cases abdominocentesis will yield diagnostic results.

DIFFERENTIAL DIAGNOSIS

- Rupture of the ureter
- Trauma to the urethra (from catheterization)
- Septicemia
- Gastrointestinal problems

TREATMENT

KEY POINT

Surgery is necessary, but the foal should be stabilized before surgery.

- This usually involves drainage of fluid from the abdomen while administering potassium-free polyionic fluid intravenously. A urinary catheter is placed to facilitate removal of urine. If the foal has a plasma or serum potassium value greater than 6 mmol/L (mEq/L), bicarbonate also should be given intravenously at a dose rate of 5 mmol/kg (5 mEq/kg).
- A midline laparotomy is necessary to repair the defect, and the usual approach is to make an elliptical incision around the umbilicus. After the abdomen is entered, it is important to determine if there are infected foci along the course of the urachus. The area of leakage in the urinary bladder is then identified and repaired.

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CHAPTER 10

Urinary System

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EXAMINATION OF THE URINARY TRACT

As with diseases in other body systems, the key to diagnosis when approaching a horse with suspected urinary tract disease is a thorough investigation following a logical sequence. Factors to be considered are the signalment, history, duration of signs, and physical examination findings so that a judicious selection can be made of appropriate tests.

History

Some pertinent questions when approaching a horse with suspected urinary tract dysfunction include

- Is the urine normal or discolored? Are there blood clots? Is the urine abnormal only at certain times during micturition (i.e., beginning, middle, or end)?
- Does the horse strain or adopt unusual postures when attempting to urinate?
- Does the horse make frequent attempts to urinate?
- Does urine flow in a normal stream or is the horse incontinent?
- What volume of urine is passed?
- Does the horse drink large volumes of water?
- Is there a history of colic?
- Has the horse lost weight?
- Has the horse been exposed to toxic plants (e.g., fast-growing sorghum or Sudan grass) or other toxins (e.g., blister beetles in alfalfa, heavy metals, red maple leaves, bracken fern, wild onion)?
- Has the horse been administered potentially toxic products (e.g., aminoglycosides, vitamin K_3, high doses of nonsteroidal anti-inflammatory drugs)?
- Has the horse had a Coggins test recently? If so, what was the result?
- Are other horses on the farm affected?
- Is there a history of respiratory disease or abortion on the farm?

Physical Examination

After an appropriate history, a physical examination is performed. Particular attention should be focused on the following:

- Examination of the penis and prepuce in geldings and stallions should be undertaken, looking for swellings, wounds, discharges, and obstructions due to smegma, tumors, or habronemiasis. Sedation with xylazine (Treatment No. 109) or acepromazine (Treatment No. 1) may be required to encourage the horse to extend its penis out of the prepuce.
- Examination of the vulva and perineum of mares also should be performed. Visual and manual inspection of the vulva and vagina should be considered with the aim of determining whether perineal urine scalding, vaginal urine pooling, or pneumovagina is present.

KEY POINT

A rectal examination is an important part of the physical examination.

- In performing a rectal examination, care should be taken to examine the bladder (the bladder may require emptying by normal voiding or catheterization to facilitate thorough examina-

tion) for degree of filling; thickness of the bladder wall, and presence of abnormal masses, such as calculi. A check should be made to identify the ureters, which are palpable only if dilated or thickened. The pole of the left kidney should be located, taking note of size and if pain can be elicited. The pelvis should be palpated to detect any evidence of trauma (e.g., fractures).

- A neurologic examination may be indicated. Abnormalities can include paralytic bladder and hypotonia and hypalgesia of the tail, perineum, and anus. Retention of feces and dilatation of the anus also may be noted and are reflective of neurologic dysfunction. Care also should be taken to identify the presence of cranial nerve deficits, because these may occur in association with a lesion causing urinary tract dysfunction.

DIAGNOSTIC AIDS

Urinalysis

KEY POINT

Urinalysis is used to assess the functional capacity of the kidneys and to reveal the presence of abnormal cells, pigments, or bacteria in the urine.

Urine is collected by free catch into a sterile container or catheterization of the urinary bladder using a sterile lubricated urinary catheter or foal stomach tube (see Fig. 10-1). Geldings and stallions usually require sedation with xylazine (Treatment No. 109) or detomidine (Treatment No. 28) to ensure patient compliance and to induce eversion of the penis out of the prepuce. After urine collection, the following observations and analyses are performed.

- *Visual inspection.* Equine urine is normally pale yellow to brown in color, slightly turbid and viscous. The viscosity is due to the presence of mucus, and the turbidity is associated with calcium carbonate crystals. Abnormalities that may alter these findings include pigmenturia, pyuria, bacteruria, spermuria, or excessive crystalluria. Nonturbid clear urine may be a feature of polyuria, because of dilute urine. Pigmenturia throughout micturition may be indicative of a bladder or renal lesion, whereas pigmenturia at the beginning or end of micturition may indicate a urethral or accessory gland lesion.
- *Specific gravity (SG),* using a refractometer. Urine SG is an estimate of the number of particles and hence the concentration of urine. Normal values are 1.008 to 1.040 for adult horses (1.001-1.025 in foals). Increases in SG normally occur in response to decreased water intake. A failure to concentrate urine (> 1.020) in the face of water deprivation may indicate renal tubular dysfunction. Horses with chronic renal dysfunction will typically produce isosthenuric urine (1.008-1.017).
- *Reagent strip or "Dipstick" methods* can be used to measure urine pH and protein, glucose, and bilirubin concentrations. Normal values for pH are 7.5 to 8.5 in adults (5.5-8.0 in foals). Urine pH decreases with metabolic acidosis and after prolonged starvation. Normal urine should not contain protein, glucose, or bilirubin.

KEY POINT

Because horse urine is normally alkaline, false-positive results for protein are common with the dipstick procedure.

- Highly concentrated urine also has the potential to produce false-positive results for protein. To overcome this, urine protein should be measured with the *sulfosalicylic acid procedure.* Significant proteinuria may occur in association with glomerular disease, infection, inflammation, and after exercise. Glucose commonly spills into the urine after the renal threshold is reached. This value is thought to be greater than 10 mmol/L (180 mg/dL) for blood glucose. If blood glucose is not elevated and urine glucose is positive, the most likely cause is renal tubular dysfunction. Bilirubinuria occurs in response to intravascular hemolysis, hepatic necrosis, and cholestatic diseases, where the concentration of direct bilirubin in plasma increases to high values.
- *Pigments*—Dipsticks give positive results for blood, hemoglobin, and myoglobin in urine. When positive results occur for pigments, proteinuria also will be present. Hemoglobinuria results from excretion of heme pigments in the blood secondary to hemolysis. Urine also may be hemoglobin positive when there is hemorrhage within the urinary tract. Myoglobin is excreted in urine subsequent to myolysis.
- *Cytology* should be performed on urine sediment after centrifugation, looking for abnormal cells or bacteria. When examined by light microscopy, up to five red cells or leukocytes per high-power field are considered normal. Increases in the number of erythrocytes reflect hemorrhage. Elevations in urinary leukocytes indicate inflammation in the urinary tract and when combined with a bacteruria are reflective of infection. Increased numbers of transitional cells in the urine may be indicative of neoplasia.
- *Casts* are the accumulation of protein and cellu-

lar material that form in the renal tubules. Their presence in urine is an indication of renal dysfunction, particularly tubular disease. Cellular casts can consist of red blood cells, leucocytes, or renal tubular epithelial cells. Casts are unstable in alkaline urine, and as such urine examination should be performed within 30 to 60 minutes of collection.

- *Crystals* are common in alkaline horse urine. Calcium carbonate crystals are the most common, particularly in horses eating alfalfa hay, and are considered a normal finding.

Bacteriologic Examination

Bacteria in small numbers are common in free-catch urine samples and are probably surface contaminants. In contrast, infections in the urinary tract are usually associated with a significant pyuria. When any doubt exists as to the presence of pyuria, a sample of urine collected aseptically by catheter is indicated. Urine sediment (after centrifugation at 5000 X *g* for 5 minutes) is examined by Gram stain and subjected to bacterial culture and antibiotic sensitivity testing (see Chapter 17). Quantitative bacterial colony counts provide some indication of infection severity.

Serum Biochemistry Values

Serum urea nitrogen (SUN) and serum creatinine (SCr) concentrations provide a crude indication of renal function in the horse. These variables always should be measured when investigating a horse with suspected urinary tract disease. They are, however, not particularly sensitive indicators of renal function.

KEY POINT

SUN and SCr concentrations can be elevated in response to prerenal, renal, and postrenal disorders.

Dehydration is a common cause of prerenal azotemia. Renal tubular damage (with acute or chronic renal failure) produces renal azotemia, whereas obstructive urinary tract disease (urolithiasis) and ruptured bladder are postrenal inducers of azotemia. It is difficult to ascribe specific ranges of SCr or SUN to each of the categories of azotemia because of large overlap.

KEY POINT

Plasma total protein and albumin concentrations may be altered in horses with renal disease.

Elevations in plasma total protein are commonly found in horses with plasma volume contraction and prerenal azotemia, whereas decreases can occur as a result of protein loss in response to glomerulotubular disease. In these latter cases, the urine commonly has elevated protein concentrations.

Electrolyte Determinations and Fractional Excretion Values

Serum electrolyte concentrations (Na^+, K^+, Cl^-, Ca^{2+}, $P0_4^-$) also may be reflective of renal dysfunction.

KEY POINT

Hypochloremia is the most common electrolyte abnormality in acute and chronic renal disease.

Other electrolyte derangments have been reported in adult horses with renal failure, including hyponatremia, hyperkalemia, hypercalcemia, and hypophosphatemia. However, these derangements are variable and depend on hydration status, diet, and age of the horse. Younger animals (<1 year old) respond similarly to other species and develop hyperphosphatemia and hypocalcemia with renal failure.

The excretion of electrolytes in urine often is altered in response to changes in the diet, renal function, water intake, and the activity of hormones that influence fluid and electrolyte balance. To overcome the effects of these influences and determine whether the kidney is controlling electrolyte homeostasis in an effective manner, the clearance of electrolytes in urine relative to creatinine can be determined. These are referred to as *fractional excretion (FE) values* and are calculated using the formula

$$FE_x\% = [urine]_x/[serum]_x \times [serum]_{Cr}/[urine]_{Cr} \times 100$$

where FE_x is the fractional excretion of electrolyte$_x$; $[urine]_x$ is the urinary concentration of electrolyte$_x$; [serum], is the serum concentration of electrolyte$_x$; $[urine]_{Cr}$ is the urinary creatinine concentration; and $[serum]_{Cr}$ is the SCr concentration. Normal FE values for adult horses and foals are presented in Table 10-1. Ideally, serum and urine should be collected at the same time, because serum electrolyte and SCr concentrations are variably stable in animals with renal disease or azotemia.

TABLE 10-1. Fractional Excretion Values for Various Electrolytes

	Fractional Excretion Value (%)	
Electrolyte	***Foal (Neonate)***	***Adult***
Sodium	0.15-0.45	0.02-1.0
Potassium	9-18	15-65
Chloride	0.10-0.75	0.04-1.6
Phosphate	0-7	0-9

KEY POINT

Decreases in the FE of various electrolytes (e.g., sodium and chloride) may reflect dietary salt deficiency. In contrast, elevated $FENa^+$ (>1.5%) in a horse with azotemia often reflects renal tubular damage with sodium wasting, whereas a low value for $FENa^+$ (<1.0%) is supportive of prerenal azotemia.

Elevations in $FEP0_4^-$ (>1.0%) may be evident in horses with primary renal failure. Measurement of $FEP0_4^-$ and Ca^{2+} has been advocated as a method of assessing adequate dietary supplementation. Similarly, decreases in $FENa^+$ and K^+ have been observed in horses suffering from chronic intermittent rhabdomyolysis. Because other factors influence electrolyte excretion in urine, FE values must be interpreted with caution.

Catheterization of the Urinary Bladder

KEY POINT

Bladder catheterization is a useful and easy technique for collection of urine samples, particularly when bacterial culture is to be attempted or to check the patency of the lower urinary tract.

Before insertion of the catheter, the horse should be placed in stocks. In males, sedation with xylazine (Treatment No. 109) or detomidine (Treatment No. 28) facilitates introduction of the catheter and relaxation of the penis. Sedation is optional in mares. After sedation in the male, the penis is grasped with one hand, and the glans penis is washed with an appropriate dilute disinfectant (e.g., povidone-iodine; Treatment No. 92). A sterile flexible urinary catheter or foal stomach tube is then picked up with the other surgically gloved hand, prepared with sterile surgical lubricant (K-Y Jelly, Johnson & Johnson), and inserted into the urethra (Fig. 10-1). The catheter is passed approximately 60 cm into the urethra until it reaches the bladder. Urine does not always flow freely initially, and suction on the catheter or injection of air with a sterile 60-mL catheter-tip syringe often promotes flow of urine. Samples are then placed in sterile containers and submitted for urinalysis and/or bacteriologic examination. Catheterization in mares generally is easier. The mare is placed in stocks, and the perineum is prepared with dilute disinfectant. A lubricated gloved hand is inserted into the vagina, and the urethral opening is identified on the floor of the vagina about 10 cm from the vulval opening. The lubricated sterile catheter (flexible or a rigid Chambers catheter) is then guided into the urethra using the index finger. After insertion, the procedure is similar to that described for stallions and geldings.

Endoscopy of the Lower Urinary Tract

Insertion of a flexible endoscope into the urethra and bladder is possible in adult horses of both sexes.

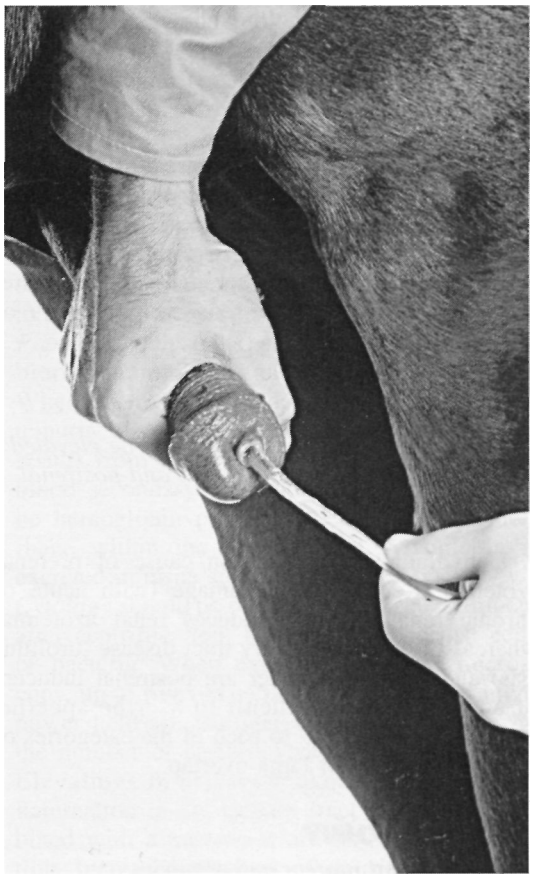


Figure 10-1. Catheterization of the urinary bladder in a gelding.

KEY POINT

Indications for the procedure include identification of calculi; inflammation, hemorrhage, or tumors of the bladder and/or urethra; and the presence of abnormal urine discharging from a ureter.

In addition, the endoscope can be used to guide a catheter into a ureter to collect urine samples from that kidney or to collect biopsies from sites within the lower urinary tract.

A flexible endoscope, 60 cm long and 7 mm or less in diameter, is suitable for males, whereas a shorter or larger diameter instrument can be used in the mare. The endoscope should be sterilized in an appropriate disinfectant (e.g., Cidex; Treatment No. 31) and rinsed with sterile water. Preparation of the horse is similar to that described for urinary catheterization, but general anesthesia may be required. The bladder is initially catheterized and emptied of urine. Infusion of 50 mL sterile lidocaine (Treatment No. 67) into the bladder and urethra (done as the catheter is withdrawn) reduces the potential for discomfort during endoscopy. The tip of the endoscope is prepared with sterile surgical lubricant and then inserted into the urethra. Once in the bladder, any remaining urine can be evacuated with the suction apparatus of the endoscope.

Inspection of the bladder is then begun. If the clinician wishes to examine the trigonal region, it is necessary to turn the tip of the endoscope through 180 degrees. Biopsy of masses within the bladder can be performed through the biopsy port or, alternatively, a small-diameter catheter can be passed through the port and inserted into a ureter. Samples of urine can then be aspirated. In the mare, the procedure for endoscopy is similar to that described for catheterization. After insertion of a sterile gloved hand into the urethral orifice, the lubricated tip of the endoscope is passed into the urethra. Subsequent to this, the procedure is the same as that described for stallions or geldings.

Diagnostic Ultrasound

Ultrasound may be used to assess the shape, size, and structure of the kidneys and bladder.

KEY POINT

Using this technique, it is possible to identify calculi in the pelvis of the kidney, renal neoplasia, and the presence of hydronephrosis.

In addition, ultrasound-guided biopsy of the kidney is possible.

Because penetration of the ultrasound beam is usually about 20 cm or less, interpretation of renal structures using transcutaneous techniques is possible. Transrectal examination of the caudal part of the left kidney and bladder also may be useful. Under most circumstances, sector scanners, which give superior resolution to that provided by linear-array machines, are the scan heads of choice for these examinations. Of course, if a sector scanner is not available, a linear-array scanner with a 3-MHz head can provide useful images.

The right kidney is horseshoe shaped and best imaged through the last few intercostal spaces high on the right side. The left kidney is bean shaped and lies medial to the spleen and can be imaged through the paralumbar fossa and the last two intercostal spaces. In acute renal failure the kidneys may be larger than normal, and in chronic renal diseases renal atrophy may be evident. Ultrasound-guided biopsy of the kidneys can be undertaken with greater potential for collection of diagnostic samples and decreased risk to the patient.

Renal Biopsy

Renal biopsy may be indicated in cases in which renal disease is suspected (from findings such as profound proteinuria) or has been diagnosed.

KEY POINT

Biopsy is performed to gain an indication of the site and extent of disease and its prognosis. Two techniques are described: a percutaneous "blind" technique and an ultrasound-guided technique.

Before any renal biopsy, the horse's hemostatic status should be evaluated, and the procedure should be forgone in patients with evidence of a coagulopathy. For the "blind" procedure, the horse is restrained in stocks and sedated with xylazine (Treatment No. 109) or detomidine (Treatment No. 28). The left paralumbar fossa is clipped, shaved, and aseptically prepared. An assistant then performs a rectal examination and identifies the left kidney and the site for infusion of local anesthetic. Then, 10 mL of local anesthetic is infused at the site on the flank. A stab incision is made with a no. 11 or 15 scalpel blade. With the assistant stabilizing the left kidney, a Tru-Cut (Travenol Laboratories, Inc., Deerfield, IL) biopsy needle is advanced slowly through the body wall until the tip is either felt by the hand of the assistant in the rectum or it is introduced into the renal parenchyma with the assistant feeling the kidney move (Fig. 10-2). It is best if the

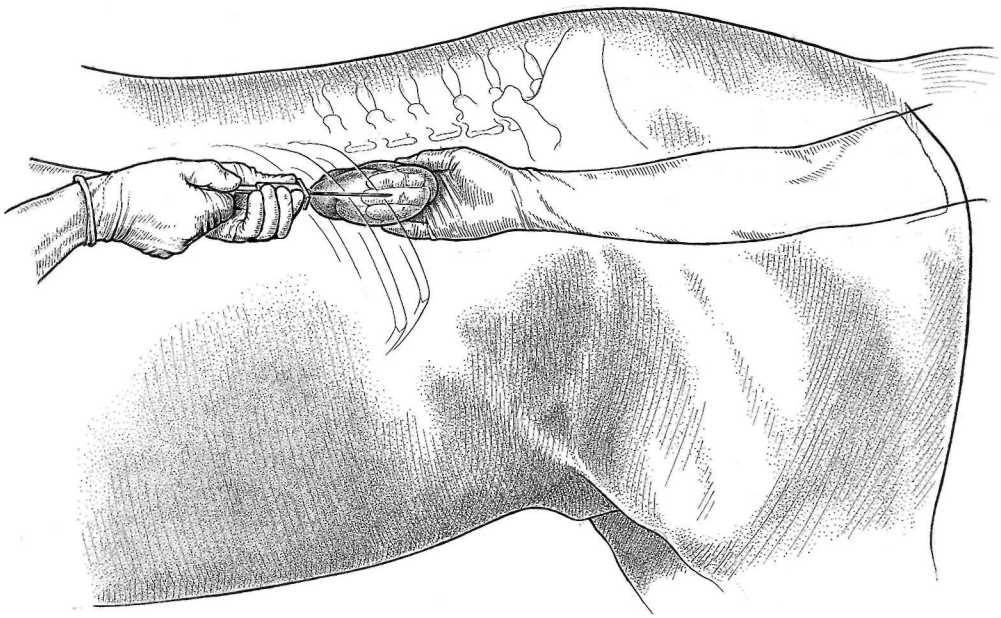


Figure 10-2. Renal biopsy. The drawing demonstrates the "blind" technique for renal biopsy. The left kidney is stabilized against the abdominal wall by an assistant. After skin preparation, local anesthetic infiltration, and a stab incision in the skin, a Tru-Cut biopsy needle is inserted into the renal parenchyma.

kidney is guided to the point of the needle rather than trying to do the reverse. It is important to attempt to collect a biopsy from the caudal pole of the kidney to ensure that a diagnostically useful sample is collected and that major renal vessels are avoided. After the biopsy needle is inserted into the renal parenchyma, the biopsy is collected. *Note:* It is important that the kidney is immobilized during the procedure to decrease the potential for hemorrhage. After collection, the sample may be submitted for histopathologic, immunofluorescence, or bacteriologic examination. The horse should be kept quiet for 24 to 48 hours after the procedure and observed for clinical signs of blood loss (e.g., pale mucous membranes, tachycardia, anxiety or evidence of pain, decreasing packed cell volume [PCV] and total plasma protein [TPP]).

KEY POINT

Ultrasound-guided biopsy of the kidney is now the most common method for collection of samples.

Biopsies can be obtained from the left or right kidney. The kidney is imaged, and precise identification is made of the site from which the biopsy is to be collected. Sedation of the horse, skin preparation, and infusion of local anesthetic are performed. With the scan head encased in a sterile surgical glove filled with coupling gel and sterile gel on the skin surface, the kidney is identified and the biopsy needle is passed into the renal parenchyma. Postoperative observation is the same as that described for the "blind" technique.

Radiography of the Urinary Tract

Radiographic examination is rarely used in the adult horse. Plain and/or contrast radiography may be of benefit in assessing patency of the lower urinary tract in foals suspected of bladder or urachal rupture.

Renal Scintigraphy

Renal scintigraphy using technetium (^{99m}Tc)-DTPA coupled to glucoheptanate can be used to provide information of proximal tubular function on an individual kidney basis. Its use, however, is largely restricted to tertiary referral centers.

Water Deprivation Test

KEY POINT

This test is useful to determine if normal renal mechanisms exist to concentrate urine.

The test is used to differentiate between central diabetes insipidus, nephrogenic diabetes insipidus, and psychogenic polydipsia.

KEY POINT

This test is contraindicated in horses that are clinically dehydrated or azotemic.

The test is initiated by collection of urine and blood samples and measurement of body weight. Urine may have to be collected by catheter. Urine specific gravity, PCV, and TPP are measured. Water is withheld, and urine and blood samples are collected at 12-hour intervals for 48 to 72 hours. This assumes no dramatic increase in urine SG or change in metabolic status of the animal (e.g., azotemia, dehydration, or dramatic reduction in body weight). If any of these events occur, the test is terminated. The maximal response is indicated by an increase in urine SG to approximately 1.050, a 12 to 15% reduction in body weight, and a 12 g/L. (1.2 g/dL) increase in TPP after approximately 48 hours. Practically, the test can be terminated well before this point is reached if the urine SG is elevated to greater than 1.030, a level indicating appropriate renal concentrating capacity. Horses with central or nephrogenic diabetes insipidus cannot concentrate urine in response to a water deprivation test. Differentiation of these two conditions can be done through administration of exogenous antidiuretic hormone (Vasopressin).

Diseases of the Urinary Tract

Cystitis

KEY POINT

Primary cystitis is uncommon in horses, whereas cystitis secondary to conditions inducing urine stasis or bladder injury is diagnosed more frequently.

Most cases of cystitis are thought to be the result of ascending rather than descending urinary tract infections.

HISTORY AND PRESENTING SIGNS

- More common in mares
- Frequent attempts to urinate
- Problems leading to urine stasis or bladder injury (e.g., urolithiasis and urinary obstruction)
- Ataxia/paralytic bladder
- Late-gestation or postpartum mare
- Repeated urinary catheterization

CLINICAL FINDINGS AND DIAGNOSIS

- Pollakiuria (frequent passage of urine), dribbling urine, and urine scalding may be evident. Pain (grunting) may be obvious during urination, and the position adopted for micturition may be held for extended periods.
- Penile relaxation may be obvious in males.
- Increases in cardinal signs (heart rate, rectal temperature) may occur in more severe cases.
- In diseases where cystitis occurs unrelated to neurogenic causes, rectal examination often reveals a small bladder with a thickened wall. In contrast, neurogenic disorders resulting in secondary cystitis may have a distended tense atonic bladder. Animals in the latter group also will often demonstrate other neurologic deficits (e.g., ataxia, weakness).
- Urinalysis shows proteinuria, pyuria, hematuria, desquamated epithelial cells, and debris. Bacteria will often be present in free-catch samples, but these are often contaminated with environmental bacteria and are not suitable for bacterial culture and sensitivity testing.

KEY POINT

Samples for bacterial culture and sensitivity should be collected via catheterization using aseptic technique before the institution of antimicrobial therapy (see Chapter 17).

- Quantitative bacterial colony counts provide the most effective way of assessing the severity of the infection. Greater than 10,000 cfu/mL of urine is considered highly suggestive of urinary tract infection.
- Bacteria commonly implicated in the induction of cystitis in horses include *Escherichia coli, Proteus mirabilis, Enterobacter* spp., *Klebsiella* spp., *Pseudomonas* spp., *Streptococcus* spp., *Staphylococcus* spp., and *Corynebacterium* spp.
- Cystoscopy reveals congested bladder mucosa, which may appear roughened, hemorrhagic, and ulcerated.
- Hematology may provide a nonspecific indication of inflammation (e.g., leukocytosis, hyperfibrinogenemia, hypergammaglobulinemia). However, these alterations are not present in all cases.

DIFFERENTIAL DIAGNOSIS

- Urolithiasis
- Urinary incontinence

- Pyelonephritis
- Neoplasia
- Prolapse of the bladder
- Ectopic ureter
- Renal failure
- Urethritis

TREATMENT

- Treatment of the primary underlying disease is imperative. Consideration of hydration and metabolic status also is important, with administration of fluids if indicated. Maintenance of urine flow is important to ensure optimal removal of debris and infectious organisms from the bladder. Indwelling bladder catheters (Foley catheter) may be useful in cases of postpartum urethral trauma or temporary neurogenic bladder dysfunction.
- Specific therapy directed at elimination of bacterial infection from the bladder also should be instituted. In mild cases of cystitis, resolution of the primary predisposing problem is often sufficient to allow local defense mechanisms to eradicate the infection.
- Initial therapy involves irrigation of the bladder with sterile isotonic fluids to remove sediment and debris from the bladder lumen.
- Antimicrobial therapy should be based on results of culture and sensitivity testing. Antimicrobial agents that are useful for treatment of bacterial cystitis include trimethoprim-sulfadiazine (Treatment No. 108), penicillin (Treatment Nos. 84-86), gentamicin (Treatment No. 56), amikacin (Treatment No. 4), ampicillin (Treatment No. 6), cephalosporins (Treatment Nos. 16 and 18-21), and oxytetracyclines (Treatment No. 81). In chronic cases, antimicrobial therapy may need to be continued for 2 to 3 weeks or more, because bacteria localize in the bladder wall. Trimethoprim-sulfonamide combinations and penicillin are exctreted via the kidneys and are concentrated in urine. As such they are often the first choice while awaiting sensitivity results. If in vitro antimicrobial resistance is found, sometimes these drugs still may be effective given their selective concentration in urine.

KEY POINT

Urinalysis and urine culture should be repeated 3 to 4 days after completion of antibiotic therapy to ensure success of treatment.

- On occasion, primary cystitis will be resistant to therapy, and additional intervention and antibiotic therapy is necessary.
- The use of urinary acidifying agents such as ammonium chloride does not appear to induce significant reductions in urine pH at palatable dose rates.

Polyuria/Polydipsia

Polyuria is an increase in urine output, whereas *polydipsia* describes an increase in water consumption. In general, a 500-kg adult horse will produce 12 to 17 L of urine per day, with an average urine SG of 1.020 to 1.030. These values may vary slightly depending on diet and ambient conditions. However, under most conditions, daily urine production of more than 20 to 25 L is regarded as being indicative of polyuria.

Renal, hormonal, and psychogenic causes of polyuria have been reported. There are two common disease states that produce polyuria. Chronic renal failure, the most common cause, produces polyuria because there are insufficient numbers of functional nephrons to concentrate urine. Tumors of the pars intermedia (pituitary) associated with secondary hyperadrenocorticism is a less common cause of polyuria found mainly in older horses.

HISTORY AND PRESENTING SIGNS

- Increased water consumption (polydipsia)
- Abnormal behavior (e.g., restlessness, frequent searching for water, drinking of urine)
- Concurrent fluid therapy or administration of osmotic diuretics (e.g., glucose, dimethyl sulfoxide, mannitol)
- Weight loss

CLINICAL FINDINGS AND DIAGNOSIS

- Polyuria can result from an increased solute concentration of the urine (e.g., glucose), referred to as *solute diuresis,* or can be due to an increase in water content of urine, known as *water diuresis.* With solute diuresis, there is polyuria with urine having a SG in the 1.008 to 1.012 range. In contrast, water diuresis results in urine with a specific gravity of less than 1.008.
- With solute diuresis, other frequently detected abnormalities may include glucosuria, proteinuria, or dramatically increased $FENa^+$.
- More common causes, lesions involved, and diagnostic procedures indicated in horses with polyuria are outlined in Table 10-2.

TREATMENT

- If there is *chronic renal failure,* treatment strategies for this disorder dicused later in this chapter may be used.

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TABLE 10-2. Causes of Polyuria and Methods for Diagnosis

Disorder	Pathogenesis	Clinical Manifestations	Diagnosis/Diagnostic Tests
Chronic renal failure* (loss of nephrons)	Loss of >75% of functional nephrons	Weight loss, isosthenuria (SG 1.008-1.017), proteinuria, urinary casts (SUN and SCr)	Serum biochemistry, urinalysis, renal biopsy
Hyperadrenocorticism* (Cushing's disease, tumors of the pars intermedia)	Increased production of adrenocorticosteroids (usually due to pituitary tumors in older horses)	Weight loss, hirsutism, f sweating, urine SG 1.008-1.017, persistent hyperglycemia and glucosuria, laminitis and susceptibility to infection.	Urinalysis; determination of plasma Cortisol, plasma glucose; TRH stimulation (50-100% in serum Cortisol). No response to dexamethazone suppression test
Exogenous isotonic fluid administration*	Parenteral fluids leading to volume diuresis	Increased plasma volume leading to GFR and diuresis, urine SG < 1.008	History of exogenous fluid administration; urine production and urine SG when fluid therapy is reduced or terminated
Central primary diabetes insipidus	Decreased production of AVP by the hypothalamus	Very low urine SG (1.001-1.007)	Urinalysis; water deprivation test shows inability to urine SG; exogenous ADH (40-80 IU in oil IM) urine SG > 1.025 in 24-36 hours
Nephrogenic (secondary) diabetes insipidus	Decreased response of kidney to AVP	Very low urine SG (1.001-1.007)	Urinalysis; water deprivation test shows inability to urine SG; no response to exogenous ADH (40-80 IU in oil IM)
Psychogenic water consumption	Psychological disorder producing compulsive water consumption	Maximally dilute urine (SG ~1.001)	Urinalysis; water deprivation test shows urine SG
Diabetes mellitus (type 1 insulin dependent)	Decreased insulin production	Weight loss; appetite; persistent hyperglycemia (300-500 mg/dL); urine SG 1.008-1.017; glucosuria; ketonuria.	Urinalysis, serum biochemistry, endogenous [insulin], response to insulin tolerance test
Psychogenic salt consumption	Behavioral abnormality t increased salt consumption, water consumption and diuresis	Urine SG 1.008-1.017	History of desire to consume salt; urinalysis shows $FENa^+$

*Most common causes.

AVP, arginine vasopressin; ADH, antidiuretic hormone; GFR, glomerular filtration rate; IM, intramuscular; SCr, serum creatinine; SG, specific gravity; SUN, serum urea nitrogen; TRH, thyrotropin-releasing hormone.

- Tumors of the pars intermedia producing *Cushing's disease,* although often benign, are usually slowly progressive; therefore, treatment may be considered. Use of cyproheptadine (Periactin, Merck & Co., Inc., West Point, PA) has been suggested. The recommended dosage schedule is 0.25 mg/kg PO q24h in the morning for 2 to 3 weeks, with the dosage doubled (0.5 mg/kg/day) and given as a divided dose if there is no response. Response, if it occurs, should be present in 6 to 8 weeks. After 3 months of therapy, reduction to alternate-day treatment can be considered. Unfortunately, in our experience, responses have been quite disappointing.
- More recently, the use of dopaminergic agonists bromocriptine mesylate (Parlodel, Sandoz, East Hanover, NJ) and pergolide (Permax, Athena Neurosciences, South San Francisco, CA) have been reported. Bromocripitine has been used at a dose rate of 0.04 mg/kg PO in the morning and 0.02 mg/kg in the evening. Clinically it would appear that pergolide is the more effective of the two at dose rates commencing at 0.001 mg/kg PO q24h.
- *Psychogenic water or salt consumption* is rare, but when diagnosed, it is managed by reducing the animal's water or salt intake, respectively, closer to daily needs. In some chronic cases of psychogenic polydipsia, a routine water deprivation test may result in partial urinary concentration, presumably due to medullary washout. In these cases, gradual restriction of water intake over a period of a few days should result in a more normal urine SG. The etiology of psychogenic polydipsia is unknown but may be related to alterations in housing, exercise, diet, and medications.

KEY POINT

It is important to ensure that water restriction does not produce continued polyuria, isosthenuria, and progressive dehydration. If this occurs, then it is unlikely that the diagnosis is psychogenic water consumption or salt consumption.

- *Primary diabetes insipidus* may respond to daily administration of exogenous arginine vasopressin (40-80 units in oil IM q24h).
- Only a few cases of *type 1 diabetes mellitus* have been reported in the horse. Often, tumors of the pars intermedia are confused with diabetes mellitus. Treatment of type 1 diabetes mellitus has been attempted using protamine zinc insulin. Dosages providing appropriate control of blood glucose concentrations are difficult to adjust, often making treatment unrewarding.

Prolapse of the Urinary Bladder (Displaced Bladder)

KEY POINT

Bladder prolapse is unusual and occurs almost exclusively in mares after foaling.

Relaxation of the supporting pelvic muscles, regional edema, dilatation of sphincters in the urinary tract, and the straining associated with parturition all predispose to prolapse of the bladder.

HISTORY AND PRESENTING SIGNS

- Mares
- Postpartum

CLINICAL FINDINGS AND DIAGNOSIS

- The bladder is observed everting from the vulva in a postpartum mare. Edema of the bladder mucosa is common owing to the restriction of venous drainage secondary to eversion.
- On rare occasions, the bladder may rupture, and intestines may prolapse out through the tear in the bladder wall.
- Affected mares commonly strain and show signs of abdominal pain.

DIFFERENTIAL DIAGNOSIS

- Eversion of the rectum
- Uterine prolapse
- Urolithiasis
- Cystitis
- Second-stage parturition

TREATMENT

- Affected mares are sedated with xylazine (Treatment No. 109) or detomidine (Treatment No. 28), and epidural anesthesia is induced with lignocaine or xylazine or both to control straining and reduce discomfort.
- The everted mucosal surface of the bladder is cleansed with saline or dilute povidone-iodine solution (Treatment No. 92).
- The bladder is replaced in its normal location via the urethra. In some cases, a urethral sphincterotomy is required to allow replacement of the bladder. Once in its normal position, the sphincterotomy is sutured.
- Postoperatively, antibiotics should be administered (e.g., trimethoprim-sulfadiazine, 15-20 mg/kg of the combination PO q12h; Treatment No. 108) for 5 to 7 days.
- In cases where bladder rupture has occurred

and bowel has eventrated, induction of general anesthesia and a laparotomy are required. The affected bowel is cleansed with generous volumes of warm sterile saline. The integrity of the bowel is then assessed, and if it is in good condition, it is retracted into the abdomen. If the bowel is compromised, resection and anastomosis of the offending bowel are undertaken. After replacement of bowel into the abdomen, the rent in the bladder wall is sutured. Replacement of the bladder in the normal anatomic location then proceeds as described above.

- Complications of bladder prolapse include postoperative straining and reprolapse of the bladder. An indwelling bladder catheter and or indwelling epidural catheter may prevent straining and subsequent reprolapse of the bladder.

Pyelonephritis

Pyelonephritis refers to bacterial infection of the renal parenchyma, calyces, and pelvis. It has been reported to occur most commonly in the adult horse as a result of ascending infection secondary to lower urinary tract infection, urine stasis, and urolithiasis. Normally, the distal ureter and renal papillae prevent urinary reflux into the kidney and act as a barrier to ascending infection. However, conditions such as those described above may compromise these defense mechanisms, resulting in pyelonephritis. Embolic (hematogenous) pyelonephritis is rare in the adult but may occur in septicemic neonates. The most commonly isolated bacteria include those identifed in cystitis, although *Actinobacillus* spp. and *Salmonella* spp. are reportedly more common in septicemic foals.

HISTORY AND PRESENTING SIGNS

- Inappetence, weight loss
- Abnormal urination
- Behavioral changes

CLINICAL FINDINGS AND DIAGNOSIS

- Many clinical signs described for cystitis may be present, because ascending infection from the lower urinary tract is the usual mechanism for induction of pyelonephritis.
- Systemic manifestations (e.g., fever and signs of depression) may be present.
- Rectal examination may reveal evidence of cystitis and distended and thickened ureters or abnormalities of the kidneys (e.g., altered size, shape, or the presence of pain on palpation).
- Ultrasound examination of the kidneys is also useful in providing information relating to alterations in size, shape, or consistency of the kidneys.
- Urinalysis reveals pyuria, hematuria, epithelial cells, and possibly casts. Collection of urine using aseptic technique is necessary to ensure that appropriate samples are submitted for bacterial culture and sensitivity.
- Hematologic changes commonly occurring in chronic pyelonephritis include anemia, leukocytosis, hyperfibrinogenemia, and hypergammaglobulinemia. If there is sufficient renal involvement, elevations in SUN and SCr also may occur.

DIFFERENTIAL DIAGNOSIS

- Urolithiasis
- Urinary incontinence
- Neoplasia
- Ectopic ureter
- Renal failure
- Urethritis

TREATMENT

- Many therapeutic strategies outlined for cystitis are suitable for pyelonephritis. In severe unilateral cases, nephrectomy of the affected kidney may be a satisfactory means for providing resolution of the problem. In chronic bilateral cases, prolonged therapy is necessary but is often unrewarding. Horses showing evidence of renal failure (i.e., increased SUN and SCr) have the least favorable prognosis for successful treatment.

Renal Failure, Acute

Acute renal failure is characterized by a rapid fall in the glomerular filtration rate and clinical signs of uremia. Concurrent alterations in fluid, electrolyte, and acid-base homeostasis ensue.

CAUSES OF ACUTE RENAL FAILURE

KEY POINT

There are three major causes of acute renal failure: (1) nephrotoxic, (2) hemodynamic, and (3) obstructive.

Toxic. Acute renal failure most commonly is induced by aminoglycoside antibiotics and nonsteroidal anti-inflammatory drugs (NSAIDs) such as phenylbutazone. Aminoglycoside-induced acute renal failure is the most common cause, resulting from a therapeutic drug, and is more likely if the horse is concurrently suffering from a condition

that limits renal blood flow (e.g., diarrhea, inadequate intake of fluids). Aminoglycosides bind selectively to the proximal tubular cells, resulting in impaired cell function and tubular necrosis. Administration of high doses of NSAIDs, also in the face of decreased renal blood flow, is another common cause of acute renal failure. Administration of NSAIDs to patients suffering from hypovolemia results in inhibition of renal production of prostaglandins. These local prostaglandins are thought to have vasodilatory effects and offset the reductions in renal blood flow secondary to hypovolemia. The net effect may be reduced glomerular filtration and cellular ischemia resulting in acute renal failure. Other causes include heavy metals, acorn buds, blister beetle, and myoglobin/hemoglobin. A single severe bout or repeated episodes of rhabdomyolysis ("tying up") with liberation of muscle pigments into the circulation also is known to induce acute renal failure. Myoglobin is thought to interfere with renal blood flow and cause tubular obstruction, perhaps because of cast formation. This manifestation is more likely in association with significant reductions in the extracellular fluid volume. Acute renal failure should always be suspected in horses that remain depressed for 3 to 7 days after an episode of tying up or that have suffered from the "exhausted horse syndrome" at an endurance ride.

Hemodynamic. Acute renal failure also can result from hemodynamic causes. Diseases resulting in shock with secondary renal failure include acute diarrhea, endotoxemia, severe hemorrhage, and bacterial septicemia (e.g., pleuropneumonia). Hemodynamic acute renal failure usually results from a combination of marked hypotension and release of vasoactive agents (e.g., endotoxin). During hypovolemic episodes, activation of the renin-angiotensin system results in further reductions in renal blood flow and glomerular filtration rate (GFR).

Obstructive. Obstruction of the urinary tract is likely to result in temporary renal failure. As described previously, obstruction to urine flow can be the result of urolithiasis, urethritis, paralysis of the bladder, neoplasia, trauma, or infection. Obstructive or postrenal causes of acute renal failure are uncommon in the adult horse. Obstruction of urine outflow results in increases in ureteral pressure that ultimately lead to reductions in renal blood flow and GFR through complex feedback mechanisms.

Other causes of acute renal failure include acute interstitial nephritis and acute glomerulonephritis. The etiology of acute interstitial nephritis is thought to be immune mediated, whereas acute glomerulonephritis represents the early stages of immune complex deposition within the glomerulus.

HISTORY AND PRESENTING SIGNS

- Diseases causing cardiovascular compromise (e.g., acute diarrhea, colic, endotoxemia)
- Signs of depression, inappetence
- History of exposure to known toxic substances (e.g., phenylbutazone, blister beetle, acorn buds)
- Signs of urinary tract obstruction (e.g., straining to urinate, discolored urine, frequent attempts to urinate)
- Reluctance to move (i.e., signs that may accompany laminitis, pleuritis, or a myopathy)

CLINICAL FINDINGS AND DIAGNOSIS

- There often will be signs consistent with the primary cause of the disorder. For instance, horses with acute renal failure show signs of depression, are lethargic and inappetent, and may or may not have a fever. Other signs of hypovolemia such as increased heart rate, injected mucous membranes, and decreased skin turgor also may be present. Edema, laminitis, evidence of a severe myopathy or spontaneous hemorrhage, and colic also may be detected.
- Oliguria is relatively common, particularly when hemodynamic alterations predispose the horse to acute renal failure. Nonoliguric renal failure is a less frequent presentation in the horse but may be associated with nephrotoxic causes.
- Dysuria and discolored urine are apparent in some horses with acute renal failure.
- In addition to the history and clinical signs, diagnosis often is confirmed by ancillary tests, particularly laboratory analyses.

KEY POINT

Horses with acute renal failure are azotemic and often hyponatremia and hypochloremic.

- Alterations in serum potassium and calcium concentrations are variable. Metabolic acidosis is common owing to bicarbonate loss in urine.
- Urinalysis reveals isosthenuria (urine SG of 1.008-1.017), with casts, protein, and microscopic hematuria commonly identified. Values for the FE of sodium and phosphate often are elevated. Enzymuria (elevations in GGT and AP in the urine) may be found.
- Diagnosis in suspected cases of obstructive renal failure is assisted by rectal palpation and ultrasound examination of the kidneys and lower urinary tract. Depending on the site of obstruc-

tion, oliguria or anuria may be important differentiating signs.
- Aseptic collection of urine is indicated for the identification of pyuria, hematuria, epithelial cells, casts, and debris, the result of damage to the urinary tract. Repeated urinalysis, looking for renal tubular cell casts, is a useful method of detecting renal damage.

DIFFERENTIAL DIAGNOSIS

- Colic
- Endotoxemia
- Hemorrhage
- Ingestion or administration of nephrotoxic substances (e.g., aminoglycosides, NSAIDs, acorn buds, blister beetles, heavy metals, mycotoxins)
- Laminitis
- Pleuropneumonia
- Rhabdomyolysis
- Obstructive urolithiasis
- Pyelonephritis

TREATMENT

- Removal of the inciting cause (if known) is the primary aim of treatment. The principles of therapy include correction of hypovolemia and maintenance of intravascular fluid volume, GFR, and urine production.

KEY POINT

This usually involves intravenous fluid therapy with attention to volume deficits and correction of electrolyte and acid-base abnormalities.

- Normal saline or other polyionic isotonic fluid solutions are good initial choices (see Fluid Therapy, Chap. 19). If possible, regular monitoring of urine output; serum or plasma sodium, chloride, and potassium concentrations; and plasma bicarbonate concentration is of value. In cases where hypernatremia exists, the use of 0.45% NaCl + 2.5% dextrose may be indicated. Fluids administered via nasogastric tube also should be considered.
- After introduction of initial fluid therapy, it should be determined whether the renal failure has resulted in oliguria/anuria or polyuria. If oliguric renal failure is present despite the infusion of fluids, particular caution must be exercised to ensure that volume overload is avoided. Intravenous fluids at initial rates of 50 to 100 mL/kg/day have been recommended until SCr begins to approach reference values. The rate of intravenous fluid administration may be determined through assessment of clinical signs, presence of peripheral edema, and regular monitoring of body weight, PCV, and TPP Significant proteinuria can occur in acute cases of glomerulonephritis, and supplementation with intravenous plasma may be beneficial.
- When oliguric renal failure is present, administration of diuretics should be considered. Traditionally, the loop diuretic furosemide is recommended (1.0 mg/kg IV q2h for two to three treatments; Treatment Nos. 54 and 55). Furosemide acts at the loop of Henle through inhibition of $Na^+ + K^+$ resorption, resulting in diuresis. Its use may be most beneficial in early acute renal failure and in cases of tubular obstruction. An alternative to furosemide is the osmotic diuretic mannitol (0.25-1 g/kg IV as a 20% solution once; Treatment No. 68). Mannitol results in increased plasma osmolality that in turn induces fluid shifts, resulting in a reduction in plasma oncotic pressure and an increase in renal blood flow and GFR. The use of mannitol is contraindicated in hypovolemic patients. Dopamine may be useful if either of these treatments fail (3-5 μg/kg/min IV in 5% dextrose as an infusion). Dopamine exerts adrenergic and dopaminergic effects, resulting in improved renal perfusion and GFR. At higher dose rates, dopamine may induce vasoconstriction, resulting in reduced renal blood flow. Horses should be monitored closely during administration for side effects, including tachycardia and hypertension.

KEY POINT

It is common for horses that initially have oliguric renal failure to subsequently develop polyuria as the disease progresses. Therefore, diuretic therapy must be monitored carefully to avoid exacerbation of polyuria and isosthenuria.

- Once urine flow is reestablished, continued infusion with isotonic polyionic fluids may result in marked reduction in the degree of azotemia.
- If endotoxemia or septicemia exists, treatment may include low doses of NSAIDs (flunixin meglumine 0.25 mg/kg IV q8h; Treatment No. 52) to assist in the maintenance of renal blood flow and systemic blood pressure, although consideration must be given to their potential for nephrotoxicity and inhibition of locally produced protective prostaglandins.
- Treatment of acute renal failure, particularly fluids, should be continued IV or by nasogastric tube at rates of 20 to 50 mL/kg/day until the SCr returns to normal and the horse begins to eat and drink voluntarily.

- The prognosis for horses with acute renal failure is variable and depends on the inciting cause.

KEY POINT

In general, the following rules can be applied: toxic nephropathies tend to have the best prognosis for return of appropriate renal function; the shorter the time between onset of signs and induction of therapy, the better is the prognosis; and a more rapid response to therapy (dramatic increase in urine output and fall in SCr) is usually associated with a better prognosis.

Renal Failure, Chronic

Chronic renal failure is a problem that occurs most frequently in older horses and is the result of glomerular or tubulointerstitial disease. The most common cause of chronic renal failure is proliferative glomerulonephritis, which is thought to be the result of deposition of antigen-antibody complexes within the glomeruli. Other causes include renal glomerular hypoplasia, chronic interstitial nephritis, pyelonephritis, and a variety of other miscellaneous causes. Chronic renal failure can occur in younger horses as well and may be the result of congenital abnormalities.

HISTORY AND PRESENTING SIGNS

- Older horses

KEY POINT

Weight loss is the most common sign associated with chronic renal failure.

- Inappetence and signs of depression

CLINICAL FINDINGS AND DIAGNOSIS

- Affected horses are often thin and may be depressed.

KEY POINT

Polyuria and/or polydipsia are common features of chronic renal failure.

- Substantial plaques of ventral edema (anasarca) may be obvious in more advanced cases suffering from hypoalbuminemia (albumin < 20.0 g/L or 2 g/dL).
- Some horses have fetid breath and oral ulcerations. Dental calculus can be present.

KEY POINT

Common clinicopathologic findings include anemia, azotemia, hypochloremia, and hyponatremia and possibly hypercalcemia, hypophosphatemia, and hyperkalemia.

- Although hypercalcemia and hypophosphatemia have been suggested to be pathognomonic indicators of chronic renal failure, these changes appear to occur only in horses with chronic renal failure that are consuming diets rich in calcium (e.g., alfalfa hay). Variable degrees of hypoproteinemia may occur. In septic conditions, an inflammatory response may occur, as reflected by leukocytosis and hyperfibrinogenemia.

KEY POINT

Urinalysis reveals isosthenuria (SG 1.008-1.017), and proteinuria may be present.

- Pyuria, hematuria, and bacteriuria are possible, particularly if the chronic renal failure is secondary to pyelonephritis.
- Rectal examination should be performed to determine the size, shape, and consistency of the left kidney and ureters.
- Ultrasound examination of the kidneys also may be of value when attempting to assess the anatomy of structures within the urinary tract.
- Diagnosis is based on the history, presenting signs, physical examination findings, clinicopathology results, and possibly a renal biopsy. A renal biopsy is of value when attempting to determine the prognosis and classify disease according to glomerular, tubular, or interstitial lesions. Immunohistopathologic examination of biopsy specimens may confirm immune complex disease.

DIFFERENTIAL DIAGNOSIS

- Acute renal failure
- Obstructive urolithiasis
- Pyelonephritis
- Internal parasitism
- Neoplasia
- Abdominal abscessation/peritonitis
- Malabsorption syndromes (e.g., granulomatous enteritis)

TREATMENT

- Classically, chronic renal disease tends to be progressive in nature despite etiology and, in many cases, treatment. Nonetheless, therapeutic principles are similar to those for acute renal

failure, with the aim to treat the primary inciting cause, if known, and to provide sufficient fluids, electrolytes, and nutrients for the horse. Low protein diets have been recommended in chronic renal failure. However, maintaining the animal in a neutral protein balance should suffice. Replacement of alfalfa hay with grass or meadow hay and addition of low protein concentrates may be beneficial. Supplementation with vitamin B complex and anabolic steroids also may be useful.

- Inhibition of the inflammatory and/or immunologic response via adminsitration of NSAIDs or corticosteroids may be of benefit in selected cases. However, the potential detrimental effects of anti-inflammatory drugs on renal blood flow and renal papillae may preclude their use in long-term therapy of chronic renal failure.

KEY POINT

When considering prognosis, horses that are unable to increase their urine SG to greater than 1.015 and have persistent elevations of SCr greater than 880 μmol/L (10 mg/dL), despite therapy, have substantial impairment of renal function, which is associated with a poor prognosis for prolonged life.

- Elevations of SCr greater than 1300 μmol/L (15 mg/dL) indicate a grave prognosis. In general, unless an easily treatable cause for the chronic renal failure is identified and removed (e.g., urolithiasis), the prognosis for long-term survival in most cases is poor.

Renal Tubular Acidosis

Renal tubular acidosis is a disorder of renal tubular function characterized clinically by metabolic acidosis and hyperchloremia. Renal tubular acidosis tends to occur for one of two reasons: failure of the distal tubule to excrete hydrogen ions (type I) or inability of the proximal tubule to resorb bicarbonate (type II). To date, type II is reported to occur in horses. Two additional forms of renal tubular acidosis have been described in humans: type IV and type III, a variant of type I. Primary and secondary forms of renal tubular acidosis exist. Secondary renal tubular acidosis has been reported as a sequel to other systemic disorders such as endotoxemia and diarrhea.

HISTORY AND PRESENTING SIGNS

- Signs of depression, altered behavior
- Weakness, ataxia
- Weight loss
- Intermittent abdominal pain

CLINICAL FINDINGS AND DIAGNOSIS

- Evidence of the presenting signs outlined above is usually sufficient to prompt further investigation of a metabolic derangement.

KEY POINT

Clinicopathologic findings in horses with renal tubular acidosis are hyperchloremia (with values greater than 110 mmol/L being customary) and metabolic acidosis.

- In the latter case, plasma bicarbonate values may be less than 10 mmol/L (mEq/L) (normal, —27-33 mmol/L). This metabolic acidosis occurs in the absence of signs commonly associated with other causes of acidemia (e.g., colic, endotoxemia, diarrhea). Alkaline urine (pH > 8.5), in the presence of the systemic acidosis, is a recurrent finding. Hypokalemia may occur in some horses with renal tubular acidosis. Azotemia is not a feature.
- Diagnosis is normally based on the clinical signs and clinicopathologic findings described and by ruling out other possible causes of these signs. In some cases, administration of ammonium chloride (100 mg/kg PO in 4-6 L of water) has been shown to result in urine acidification (pH < 6.5) by 4 hours after administration. *Note:* The bladder should be catheterized and emptied before administration of the ammonium chloride.

DIFFERENTIAL DIAGNOSIS

- Other forms of renal disease
- Neurologic diseases resulting in ataxia and weakness (e.g., spinal cord disease)
- Colic, endotoxemia
- Cardiac failure
- Rhabdomyolysis
- Pleuropneumonia
- Laminitis

TREATMENT

- Correction of low plasma bicarbonate is the major aim of therapy for renal tubular acidosis. The bicarbonate deficit can be calculated using the formula body weight (kg) X 0.3 X base deficit (mmol). Base deficit is calculated by subtracting the measured plasma bicarbonate value from the normal value of 27 mmol/L.

KEY POINT

Initially, one-half of the bicarbonate deficit should be administered intravenously using

isotonic sodium bicarbonate solution over 4 to 6 hours. The remainder of the calculated dose should be administered over the next 24 to 36 hours. Sodium bicarbonate can be administered via nasogastric tube also.

- Correction of the acid-base balance usually is associated with a worsening of the hypokalemia, particularly if the horse remains inappetent. Administration of potassium chloride (40-60 g [1.5-2 oz] in 4 L of water via nasogastric tube q8-12h) is effective. When the horse begins consuming feed voluntarily, the need for potassium supplementation diminishes.
- When the plasma bicarbonate values have been corrected, oral bicarbonate therapy is instituted (150 g/450 kg in feed ql2h). This dose is usually sufficient for maintenance but should be modified in individual cases according to plasma bicarbonate values.
- Some cases will resolve spontaneously over 1 to 2 weeks, whereas others require protracted therapy to prevent recurrence of the renal tubular acidosis.

Rupture of the Urinary Bladder

KEY POINT

Although relatively common in foals (see Chapter 9), rupture of the urinary bladder is an uncommon problem in adult horses. When it does occur, it is usually a sequel to foaling or urinary tract obstruction.

HISTORY AND PRESENTING SIGNS

- Most common in mares, postpartum
- Signs of depression and inappetence a few days after foaling
- Secondary to urolithiasis and obstruction in adult males

CLINICAL FINDINGS AND DIAGNOSIS

- Signs of depression and possibly a mild increase in heart rate and respiratory rate are found.
- Small amounts of urine may still be voided.
- Clinicopathologic findings include azotemia, hyperkalemia, hyponatremia, and hypochloremia.
- Abdominocentesis reveals a large volume of yellow fluid. The presence of calcium carbonate crystals in the peritoneal fluid is strongly supportive of a diagnosis of uroperitoneum.

KEY POINT

Diagnosis is confirmed by comparing SCr concentration with that of the peritoneal fluid.

- In the presence of uroperitoneum, peritoneal fluid creatinine concentration is 1.5- to 2-fold greater than that in the serum.
- If obstructive urolithiasis has predisposed the bladder to rupture, many signs and diagnostic criteria outlined for urethral calculi are pertinent.
- Cystoscopy may assist in defining the presence of a urethral calculus or rent in the bladder wall. This procedure helps identify the extent and location of the tear.

DIFFERENTIAL DIAGNOSIS

- Other causes of colic
- Peritonitis
- Urolithiasis
- Renal failure
- Cystitis

TREATMENT

- Small tears with limited leakage of urine may be left to heal by second intention. Such tears are indicated by the ability of the horse to pass moderate amounts of urine and the presence of limited metabolic derangements. Such horses may benefit from placement of an abdominal drain (sump drain) if significant uroperitoneum is present.
- Horses with dehydration and other metabolic disorders benefit from the infusion of isotonic fluids that *do not* contain potassium because of the presence of hyperkalemia. Good fluid choices include 0.9% sodium chloride or dextrose 5% in water (see Chapter 19).
- Horses with more severe disruption to the bladder wall require surgery. In these cases, referral to a university or well-equipped surgical clinic should be considered. Stabilization of the horse before referral or surgery is important and can be achieved through placement of an abdominal sump drain and commencement of appropriate intravenous fluid therapy.

Urethral Diverticular Concretion ("Bean")

Urethral diverticular concretion is a disorder in which smegma accumulates in the urethral diverticulum.

KEY POINT

In general, this process does not create any problems for the horse; however, on occasion, the size of the concretion and associated inflammatory response can result in adverse signs.

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HISTORY AND PRESENTING SIGNS

- Adult males
- Frequent attempts to urinate, penis extended
- Urine spraying may occur

CLINICAL FINDINGS AND DIAGNOSIS

- Dysuria and urine spraying during micturition may be observed.
- Urine staining or scalding on the legs is possible.
- The region of the penis near the urethral opening may be swollen.
- Diagnosis is based on examination of the penis, after sedation with xylazine (Treatment No. 109) or detomidine (Treatment No. 28) to encourage relaxation, revealing accumulation of a large amount of foul-smelling smegma in the urethral diverticulum.

DIFFERENTIAL DIAGNOSIS

- Urethral calculi
- Cystic calculi
- Penile/urethral neoplasia (e.g., squamous cell carcinoma)
- Habronemiasis of the penis
- Penile injury (e.g., hematoma, lacerations)
- Urethritis (stallion)

TREATMENT

- Manual removal of the "bean" after sedation results in resolution of the problem.

Urethritis

Urethritis occurs most commonly in stallions and may be associated with hemospermia. The cause of the disorder is not known.

HISTORY AND PRESENTING SIGNS

- Stallions
- Discolored discharge from the penis after mating
- Difficulty urinating

CLINICAL FINDINGS AND DIAGNOSIS

- The penis may be extended from the prepuce, and there may be obvious swelling of the urethra.
- Blood or serosanguinous discharge may be noted from the urethra after coitus, and hemospermia is common.

In severe cases, dysuria, pollakiuria (frequent passage of urine), stranguria (straining to urinate), and urinary retention can occur because of a significant reduction in urethral lumen diameter.

KEY POINT

Diagnosis is based on endoscopic examination of the urethra.

- Bacterial culture and sensitivity of samples from the urethra can be useful when attempting to undertake treatment.

DIFFERENTIAL DIAGNOSIS

- Hemospermia
- Cystitis
- Urolithiasis
- Pyelonephritis
- Trauma to the penis

TREATMENT

- Sexual rest is advised.

KEY POINT

Establishment of urethral patency is important, and in cases where urethral occlusion has occurred, a perineal urethrostomy is indicated.

- Urethrostomy wounds are normally left to heal by second intention.
- Systemic antibiotic therapy is useful when using agents active against the bacteria involved and excreted in the urine. Local antibiotic therapy is possible, with drugs administered by a sterile catheter inserted through the urethral orifice or urethrostomy wound. Alternatively, suppositories can be inserted into the distal urethra.
- Complications include poor response to therapy, urethral cicatrix formation with subsequent obstruction, and urethral fistula at the urethrostomy site.

Urinary Incontinence

Urinary incontinence results from a variety of disorders, including neurologic, bladder, and urethral causes. Neurologic causes of incontinence can result from those affecting the spinal cord above the sacral nerves (upper motor neuron) and those affecting the sacral nerves, detrusor muscle, and urethral sphincter (lower motor neuron).

KEY POINT

The most common causes of urinary incontinence in the horse are herpesvirus myelitis, neuritis of the cauda equina, and the sorghum ataxia-cystitis syndrome.

Other causes include equine protozoal myeloencephalitis, trauma, urolithiasis, neoplasia, and ectopic ureter.

HISTORY AND PRESENTING SIGNS

- Apparent difficulty urinating
- Small amounts of urine voided
- Recent history of consuming rapidly growing sorghum or Sudan grass
- Ataxia
- Respiratory disease
- More than one horse affected
- Possibly a young horse (ectopic ureter)

CLINICAL FINDINGS AND DIAGNOSIS

- Intermittent passage of small amounts of urine.
- Exercise may induce an increase in the volume of urine voided.
- Horses with upper motor neuron signs may be ataxic and demonstrate no voluntary ability to urinate. Variable degrees of urine dribbling occur. Rectal examination reveals normal anal tone and a bladder of variable size. Resistance to emptying of the bladder by application of pressure during the rectal examination is often a significant finding. After a few weeks, a bladder voiding reflex is invoked, and spontaneous partial vesicular emptying occurs.
- With lower motor neuron disease, rectal examination reveals a large, tense, yet atonic bladder. Manual evacuation of the bladder is usually easy to achieve, unless the lesion involves the bladder wall or pelvic nerve. In the latter case, difficulty is encountered when attempting to manually empty the bladder. Anal hypotonia and hypalgesia are commonly associated with lower motor neuron dysfunction.
- With an ectopic ureter, the bladder will be normal on rectal examination; however, urine will constantly dribble from the urethra because of the discharge of urine into the proximal urethra.
- If an ectopic ureter is suspected in a young horse, referral for an excretory urogram is recommended.

DIFFERENTIAL DIAGNOSIS

- Equine herpesvirus myelitis
- Neuritis of the cauda equina
- Sorghum ataxia-cystitis syndrome
- Equine protozoal myeloencephalitis
- Osteomyelitis of the sacral vertebral column
- Trauma (vertebral fractures)
- Neoplasia
- Urolithiasis
- Ectopic ureter
- Cystitis

TREATMENT

- There is no specific therapy for neurogenic incontinence.
- If primary disease predisposes the horse to incontinence (e.g. equine herpesvirus type 1 [EHV-1] myelitis, equine protozoal myeloencephalitis, or urolithiasis), appropriate therapy should be undertaken. Of the primary diseases predisposing to urinary incontinence, EHV-1 myelitis has one of the best prognoses for return of appropriate bladder function.
- Symptomatic therapy involves manual expression of the bladder per rectum three to four times per day. Soft Foley catheters with inflatable cuffs can be inserted into the bladder, although the presence of the catheter is likely to be irritating and increases the chance of local infection.
- Empirical pharmacologic therapy involves phenoxybenzamine (0.6-1.0 mg/kg PO q6-8h; Dibenzyline, SmithKline Beecham, Philadelphia, PA) and bethanechol (0.02-0.08 mg/kg SC q8h, titrated for the patient; Urecholine, Merck & Co., Inc., West Point, PA). These treatments appear to have most benefit in horses with EHV-1 myelitis and neuritis of the cauda equina. Possible, yet rare, side effects of treatment include penile relaxation, sedation, sweating, and abdominal pain.

KEY POINT

Pharmacologic therapy must he instituted early in the syndrome to prevent the onset of severe bladder-wall contractile dysfunction.

Urolithiasis (Urinary Calculi)

Calculi can form in many sites throughout the urinary tract, including the kidneys, ureters, bladder, and urethra. Although urolithiasis is not common, and when clinical signs occur, they are usually associated with urinary tract obstruction.

FACTORS AFFECTING THE INCIDENCE OF UROLITHIASIS

KEY POINT

Calculi in the bladder and urethra are more common causes of urinary tract obstruction in

males, owing to the narrow urethra, than in females.

- The short large-diameter urethra of the mare generally allows natural evacuation of all but the largest cystic calculi.
- Stallions and geldings have a long urethra that narrows at the ischial arch.
- Many factors affect the formation of urinary calculi, including

Urinary Constituents. Alkaline pH, high incidence of crystalluria, high concentration of mucoprotein, high mineral content (e.g., calcium carbonate), combination of desquamated epithelial cells and mucous clots may serve as a nidus for urolith formation.

Diet. Consumption of feed and water high in mineral content may predispose to cystic calculi.

Other Factors. Urinary retention or stasis, decreased water intake, bacterial infections.

TYPES OF CALCULI

The most common types of calculi are composed of calcium carbonate in various hydrated forms. At times, calculi also will be composed of magnesium, ammonium, and phosphate. Calculi have two common appearances: a rough spiculated type that is moderately friable, with a yellow-brown color, usually oval or irregularly shaped, and a smooth type, round to oval, and gray white. Calculi range in size from 0.5 to more than 20 cm and in weight from a few grams to more than 5 kg.

Nephrolithiasis and Ureterolithiasis

The incidence of calculi in the kidney or ureter was approximately 16% in a series of 68 cases of urolithiasis. When calculi do occur, there are often nonspecific clinical signs.

HISTORY AND PRESENTING SIGNS

- Usually in adults (>3 years old)
- Weight loss may be reported
- Poor performance
- Nonspecific hindlimb lameness
- Back pain
- Inappetence

CLINICAL FINDINGS AND DIAGNOSIS

- The horse may be in relatively poor condition.
- Serum biochemistry often reveals azotemia (increased SUN and SCr), a reflection of renal failure. If chronic renal failure has occurred, hypochloremia, hyponatremia, alterations in calcium and phosphorus concentrations, and anemia may occur. Oral ulcerations and dental tartar also may be present.
- Urinalysis demonstrates hematuria, proteinuria, and at times pyuria and cellular or protein casts.
- Rectal examination may reveal a dilated ureter or the presence of a calculus in the ureter.
- Ultrasound examination (transabdominal and per rectum) is useful to reveal the size and consistency of the kidney and also to indicate the presence of nephroliths in the renal pelvis. Nephroliths may be indicated within the renal pelvis by the presence of an acoustic shadow.

KEY POINT

If a nephrolith is identified, the contralateral kidney should always be examined.

- Cystoscopy may be used to indicate the patency of the upper urinary tract. The ureteral openings are visualized, and the normal pulsatile discharge of urine (one to two times per minute) is looked for.
- Diagnosis is based on clinical signs and laboratory, ultrasound, and cystoscopic examination results.

DIFFERENTIAL DIAGNOSIS

- Cystic calculi
- Urethral calculi
- Other causes of urinary tract obstruction
- Other causes of colic
- Acute renal failure
- Chronic renal failure
- Bladder atony
- Other causes of weight loss (e.g., malabsorption syndromes, internal parasites)
- Pleural effusion
- Laminitis

TREATMENT

Treatment is difficult and often unrewarding. In cases where nephroliths are found as an incidental finding on ultrasound examination and the horse has no obvious evidence of renal dysfunction, continued observation is likely to be the best course of action. Techniques for the surgical removal of nephroliths are described. However, if chronic renal failure is present, removal of the urolith is unlikely to improve the long-term prognosis for the horse. In valuable animals or those with only limited evidence of impaired renal dysfunction, referral for surgery is an option. Recently, lithotriptic fragmenation has been de-

scribed as a technique to enable removal of ureteral calculi.

Cystic Calculi (Bladder Calculi)

KEY POINT

Cystic calculus is the most common form of urolithiasis.

Clinical signs are more common in stallions and geldings than in mares for the reasons outlined above. Identification of cystic calculi can be an incidental finding during rectal examination. When clinical signs occur, they can be dramatic and consistent with urinary obstruction. These clinical signs are described below.

HISTORY AND PRESENTING SIGNS

- More common in stallions and geldings
- Adult horses (>3 years old)
- Restlessness and evidence of colic
- Frequent attempts to urinate
- Small amounts or no urine passed
- Relaxation of the penis and adoption of an unusual stance when attempting to urinate
- Discolored urine
- Blood in the urine, especially at the end of urination
- Lameness/stilted hindlimb gait

CLINICAL FINDINGS AND DIAGNOSIS

- The signs are often nonspecific and similar to those demonstrated by horses with other causes of abdominal pain.

KEY POINT

Dysuria, straining to urinate (stranguria), and increased frequency of urination (pollakiuria) are common.

- Urine scalding on the hindlimbs in the male and perineum in the mare may occur.
- Blood at the urethral orifice may be noticeable. At times, gross hematuria is evident, particularly after exercise and toward the end of urination. Microscopic hematuria is always present.
- Maintenance of the stance adopted for urination for protracted periods may be observed. The horse may strain, grunt, and appear anxious during these periods.

KEY POINT

Diagnosis is based on rectal examination, which usually reveals a large firm mass within the lumen of the bladder.

- If the bladder is distended with urine, it is necessary to pass a urinary catheter to evacuate the urine. After catheterization, the bladder is again palpated per rectum.
- Ultrasound examination (per rectum) and cystoscopy will provide additional diagnostic information. Endoscopy helps define the size, shape, and number of calculi and the integrity of the bladder mucosa.
- Complications occurring with urethral calculi include persistent cystitis, pyelonephritis, rupture of the urinary bladder, and bladder atony.

DIFFERENTIAL DIAGNOSIS

- Nephrolithiasis, ureterolithiasis
- Urethral calculi
- Other causes of urinary tract obstruction (e.g., smegma accumulation)
- Other causes of colic
- Acute renal failure
- Chronic renal failure
- Bladder atony
- Other causes of weight loss (e.g., malabsorption syndromes, internal parasites)
- Pleural effusion
- Laminitis
- Myopathies

TREATMENT

- In the first instance, emptying of the bladder is important to reduce the possibility of rupture. This is done by the passage of a urinary catheter. This process may be difficult if the urolith is lodged in the outflow tract or urethra.
- In stallions or geldings, surgical correction by cystotomy under general anesthesia or pelvic urethrostomy or pararectal cystotomy subsequent to epidural anesthesia may be used. Referral to a university or well-equipped surgical referral clinic should always be considered an appropriate option. In mares, smaller calculi can, at times, be removed manually via the urethra. The mare is restrained in stocks, sedated with xylazine (Treatment No. 109) or detomidine (Treatment No. 28), and epidural anesthesia is induced with lidocaine and xylazine (see Chapter 8). The vagina is rinsed with appropriate disinfectant, and the surgeon's lubricated gloved hand is inserted into the urethra. The urethra is dilated sufficiently to allow the insertion of a lithotrite or grasping forceps. With large calculi, the lithotrite is used to break the calculus into small pieces to aid removal. The forceps can be better manipulated if the operator has his or her other hand in the rectum and on the bladder.

If greater access is required, a dorsal urethral sphincterotomy can be performed. After removal of the calculus, the bladder is rinsed using large volumes of saline to remove any residual fragments or debris, and the sphincterotomy is sutured (if performed).

- Postoperatively, the horse should be administered antibiotics (e.g., trimethoprim-sulfadiazine 15-20 mg/kg of the combination PO q12h; Treatment No. 108) for 5 to 7 days. Water intake, and therefore urine output, should be increased by feeding a diet rich in salt (30-60 g added to the feed morning and night) for approximately 2 weeks.

Urethral Calculi

Urethral calculi are encountered almost exclusively in stallions and geldings because of the long urethra that narrows near the ischial arch. The most common sites for obstruction are the pelvic or distal portions of the urethra.

HISTORY AND PRESENTING SIGNS

- Males
- Adults (>3 years old)
- Similar signs to those described for cystic calculi

CLINICAL FINDINGS AND DIAGNOSIS

- The signs are often nonspecific and similar to those seen in horses with cystic calculi.

KEY POINT

Dysuria, pollakiuria (frequent passage of urine), and stranguria (straining to urinate) are common.

- If the obstruction is complete, anuria will occur; but if it is partial, small amounts of urine may be voided. This urine often is discolored (red-brown) with obvious or microscopic hematuria.
- Blood draining from the external urethral orifice may be seen.
- The horse may stand as though attempting to urinate for protracted periods. The horse may strain, grunt, and appear anxious during these periods.
- Rectal examination reveals a large tense bladder (if it is not ruptured).

KEY POINT

Diagnosis is based on the history, clinical signs, palpation of the urethra per rectum and percutaneously, by the passage of a urinary catheter (an obstruction to continued passage is met), and possibly by endoscopic examination.

- Complete obstruction eventually leads to rupture of the bladder.

DIFFERENTIAL DIAGNOSIS

- Nephrolithiasis, ureterolithiasis
- Other causes of urinary tract obstruction (e.g., smegma accumulation)
- Other causes of colic
- Acute renal failure
- Chronic renal failure
- Bladder atony
- Other causes of weight loss (e.g., malabsorption syndromes, internal parasites)
- Pleural effusion
- Laminitis
- Myopathies

TREATMENT

- Given the risk of bladder rupture, correction of urethrolithiasis should be considered an emergency.
- Calculi in the distal urethra often can be expressed manually or by an incision through the median raphe over the calculus. This procedure usually is performed under general anesthesia and in dorsal recumbency to ensure patient compliance, to improve urethral relaxation, and to decrease the risk to the attending clinician. After removal of the calculus, the wound is sutured or allowed to heal by second intention.
- Calculi lodged near the ischium can be removed via an ischial urethrostomy. This is performed in the standing horse after sedation with xylazine (Treatment No. 109) or detomidine (Treatment No. 28) and induction of epidural anesthesia with lidocaine and xylazine (see Chapter 8). A catheter is passed into the urethra to aid in location of the calculus and identification of the urethra. The skin over the urethra is prepared for surgery, and an incision into the urethral lumen is made. Once entered, the lumen is dilated to allow passage of a grasping forceps or lithotrite. The calculus is then removed while flushing saline into the urethra to assist with lubrication or is crushed with the lithotrite and the pieces removed. Care must be exercised during this procedure to avoid rupturing the urethra. After removal of the calculus, the bladder is emptied and flushed with water to remove fragments and debris. Endoscopy can then be

performed to gain an estimate of any damage to the urethra and bladder.
- After removal, the urethra is sutured or allowed to heal by second intention.
- Postoperative management is similar to that described for cystic calculi.
- Acidification of the urine is indicated to prevent further formation of urinary calculi, but this is difficult to achieve.
- Complications include ruptured bladder or urethra, postobstruction urethral scar formation leading to subsequent obstruction, urethral diverticulum or fistula, persistent cystitis, and pyelonephritis.

Idiopathic Renal Hematuria

A condition of moderate to severe unilateral or bilateral renal hematuria has been reported in the horse. Gross hematuria often accompanied by blood clots is the primary feature. The condition may occur in otherwise clinically normal horses or in conjunction with other systemic disease states (e.g. pleuropneumonia, endotoxemia). In severe cases, hemorrhage can be life threatening, and supportive therapy in the form of whole blood transfusions is required. The pathogenesis is unknown; however, an underlying immune-mediated cause is suspected. Diagnosis is based on visualization of hemorrhage from the ureter on the affected side. Additional investigations should include hematologic and biochemical analysis, renal ultrasonography, urinalysis, and evaluation of clotting profiles to rule out other causes. Treatment may not be necessary in some cases; however, in others, unilateral nephrectomy may be required.

Urethral Defects

Hematuria associated with urethral defects has been reported in stallions and geldings. Commonly, hematuria is noted at the end of micturition, and in stallions the presenting complaint may be hemospermia. The pathogenesis of the condition is not completely understood; however, it is thought to be related to a rupture in the corpus spongiosum. In some cases, underlying transitional cell carcinoma has been diagnosed. The most common site is at the level of the ischial arch, and diagnosis can be achieved through endoscopic examination of the urethra. The presence of urethral ulceration should alert the clinician to the possibility of concurrent neoplasia or urethritis. In many cases the hematuria resolves over a variable period. However, in those cases with long-standing hematuria, temporary subischial urethrostomy may provide resolution of the problem.

Urinary Tract Neoplasia

Neoplasia of the equine urinary tract is relatively rare. Reported neoplasms include squamous cell carcinoma, lymphosarcoma, renal cell carcinoma or adenocarcinoma, and transitional cell tumors. Squamous cell carcinoma occurs most commonly in the bladder and on the external genitalia, especially as penile or preputial tumors in older male horses. Occasionally they may involve the urethra and local lymph nodes, with metastatic rates of up to 12% reported. Renal cell carcinomas are often well advanced at the time of diagnosis, with metastatic rates of approximately 60 to 70% reported.

HISTORY AND PRESENTING SIGNS

- Clinical signs may be nonspecific and include weight loss, anorexia, anemia, fever, and intermittent colic depending on the location of the neoplasm. Hematuria, pollakiuria, and stranguria may be a feature of renal, bladder, and urethral neoplasms.

CLINICAL FINDINGS AND DIAGNOSIS

- A diagnosis of urinary tract neoplasia may be reached through history and clinical examination and after ruling out other differential diagnoses.
- Urinalysis may be useful to determine the nature of the pigmenturia, and occasionally neoplastic cells may be demonstrable on cytologic examination.
- Rectal examination may reveal bladder and/or renal masses.
- Transrectal and/or percutaneous ultrasound examination will allow assessment of renal and bladder architecture. Ultrasound-guided biopsy can be performed if abnormalities are detected.
- Cystoscopic examination may allow direct visualization of a neoplastic mass.

DIFFERENTIAL DIAGNOSIS

- Nephrolithiasis, ureterolithiasis
- Urethral calculi
- Other causes of urinary tract obstruction (e.g., smegma accumulation)
- Acute renal failure
- Chronic renal failure
- Bladder atony

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TREATMENT

- In the case of squamous cell carcinomas involving the distal urinary tract, the treatment of choice is surgical resection. Unilateral nephrectomy has been reported as a treatment for renal hematuria. However, by the time of diagnosis, many renal neoplasms are well advanced and are not amenable to surgical treatment. Partial bladder wall resection has been attempted, with poor results, for cystic neoplasia.

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CHAPTER11

Ophthalmology

Cameron J. G. Whittaker and David A. Wilkie

Eye diseases in the horse represent a significant component of equine practice. Although fundamental concepts in ophthalmology may be applicable across all the species, the horse has some unique characteristics. This offers a challenge to the general practitioner not only diagnostically but also therapeutically. Equine ophthalmic disease, if inadequately treated, can lead to serious sequelae due to poorly understood inflammatory and reparative mechanisms in this species. Unfortunately, even the slightest scars in the horse eye at best look poor cosmetically and at worst may preclude the animal from participating in specialized events, such as racing. This emphasizes the role the general practitioner may play in the final outcome of equine ophthalmic disease. Minor scars elsewhere in the body may be inconsequential but in the eye can be disastrous. This section is designed for use by the general practitioner and covers the important equine ocular diseases. Acquisition of a good history, detailed ocular and physical examination, use of appropriate diagnostic aids, and sound knowledge and thoroughness are the most important aspects of equine ophthalmology.

KEY POINT

More mistakes are made by not looking than not knowing.

GENERAL EQUIPMENT NEEDED

1. Fishing tackle box—Ideal to store equine ophthalmology equipment and fits easily into a car.
2. Focal light—Finoff transilluminator with rechargeable handpiece and direct ophthalmoscope head.
3. Magnifying spectacles—Head mounted preferably with attached light source. This allows use of both hands. Obtain 4 and 10 x magnification lenses.
4. Fluorescein paper—Use in any eyes that appear red, blue, or painful.
5. Schirmer tear test strips.
6. Indirect ophthalmoscopy:
   Light source—Finoff transilluminator or head mounted source (Heine all pupil headset, Herrsching, Germany).
   Condensing lens—We prefer a 14 and a 20 diopter lens.
7. Mydriatic—1% tropicamide (Mydriacyl, Alcon, Fort Worth, TX).
8. Topical anesthetic—0.5% proparacaine HC1 (Ophthaine HC1, Squibb, Princeton, NJ).
9. Eye stream/artificial tears—To clean ocular debris from the eye.
10. von Graefe or other nontraumatic fixation forceps.
11. Open-ended nos. 3.5 and 5 French tomcat urinary catheter for nasolacrimal cannulation, and silastic tubing with a 0.065-inch external diameter and 0.03-inch internal diameter for subpalpebral lavage (Silicone medical tubing, VWR Scientific, Bridgeport, NJ).
12. Microbiological culturettes—noncotton, moistened (Culturette, Becton Dickinson Microbiology Systems, Becton Dickinson and Company, MD). For any corneal ulcers.
13. Glass slides and a Kimura platinum spatula (Sparta Ophthalmic Instrument Corporation, Fairfield, NJ) or scalpel blades—For cytologic specimens before initiating therapy.

14. Local anesthetics—For nerve blocks, such as 2% lidocaine, prilocaine, or mepivacaine.

15. Roll of 2-cm-wide tape, buttons for stents, tongue depressors, sterile injection caps, hubless 12-gauge or 14-gauge needles all for placement of a subpalpebral lavage system.

16. Suture—2/0 nylon on a cutting needle for subpalpebral lavage systems, and 3/0 or 4/0 silk and 5/0 absorbable suture for subcutaneous sutures for eyelid lacerations.

17. Sedatives—Xylazine HC1, 100 mg/mL, and butorphanol tartrate, 10 mg/mL.

18. Syringes—3-mL syringes.

More Specialized Equipment

19. Applanation tonometer—For diagnosing glaucoma and uveitis. Although expensive (about $3000), we believe this piece of equipment will quickly pay for itself if used as part of the routine examination. We currently use a Tonopen XL (Mentor O and O, Norwell, MA).

20. Slit lamp biomicroscope—We prefer the Kowa SL-14 (Kowa Company Limited, Tokyo, Japan) due to its portability. The slit lamp provides excellent magnification and the ability to localize lesions in the eye; however, it may be hard to justify the cost.

EQUINE VISION

Little information is available on equine vision, evaluation of visual deficits, and correction of vision loss in the horse. Horses with reduced or absent vision may show little behavioral evidence of blindness, whereas others may show profoundly abnormal behavior.

KEY POINT

Blind horses, like people, if kept in a familiar environment may not overtly show evidence of blindness. Only with a change in environment will owners notice the "sudden blindness."

The equine eye is designed to detect motion as an early warning sign to avoid predators. The eyes therefore provide a large panoramic view (greater than 350 degrees), with the only blind spots immediately anterior to the nose and several meters posterior to the rump. Horses may be using their limited binocular vision when their ears are erect and facing forward. Horses are thought to see yellow, green, and blue colors but appear to have poor perception of red.

VISION TESTING

- Signs of blindness—Bumping into walls, high stepping gait, reluctance to pass through gates, difficulty in leading.
- Menace testing—This test provides a threatening gesture to the horse's eye, causing the horse to blink. We usually move a hand from ventral to dorsal rapidly rather than in and out, which may generate air currents. This reflex may not develop until 2 to 4 weeks of age.

KEY POINT

Do not create air currents in menace testing a horse, otherwise the horse will blink not because of visual abilities but because it can feel the air move.

- Obstacle tests—Use plastic buckets of different sizes and shapes. Make sure to use a "patch" to test each eye individually. Tests are performed in lighted and dark conditions. Animals can be led, on a loose reign, over small jumps.

DIAGNOSIS OF EYE PROBLEMS

History

A thorough history is often a shortcut to a correct diagnosis, saving time and effort by directing the clinician to the main ophthalmic abnormality, thereby avoiding unnecessary tests. Useful questions include

- Does the horse have problems in seeing things? If so, for how long?
- Is the problem worse in one eye?
- Has there been a history of the problem before?
- Is vision worse at night or day?
- How long has the problem been present?
- Has the animal been given any previous medication?
- Is the problem changing in response to treatment?
- Has there been ocular discharge? If so, what is it like?
- Is there any identifiable cause?
- Are other animals affected? If so, do these affected animals have contact with each other? Are family members affected?
- Has the eye appeared a different size?
- Has the eye been a different color than usual?
- Is there any evidence of pain?

Ophthalmic Examination

KEY POINT

"A picture is worth a thousand words"—wherever possible, try to draw with colored pencils what you see or photograph and record the abnormality. This can be used for comparison in follow-up examinations. Sequential photographs can also be used as part of the record.

Essential for the completeness of an ophthalmic examination is an eye examination sheet (Fig. 11-1). As an example, we follow the following steps when performing an equine ophthalmic examination:

1. Stand in front of the horse and look at the symmetry of the eyelashes, the globe size and position, the symmetry of the pupils (as demonstrated by the tapetal reflection), and the symmetry of the orbits. Simultaneously palpate the orbital areas.
2. Assess the menace, pupillary light reflexes and perform a neuroophthalmology examination if indicated.

KEY POINT

Use of sedatives and an auriculopalpebral nerve block may alter the results of the neuroophthalmic examination.

3. Sedation and an auriculopalpebral nerve block then may be performed.
4. Microbiologic samples for culture and sensitivity are taken before topical anesthesia is used. After the samples are taken, topical anesthesia may be used to facilitate obtaining cytologic specimens.
5. Fluorescein staining is performed.
6. Intraocular pressure (IOP) measurement and nasolacrimal irrigation may then be performed.
7. Evert and examine the eyelids, and look behind the third eyelid for foreign bodies.
8. If the IOP is normal, then the pupil may be dilated.
9. Once the pupil is dilated, the anterior structures, then the lens, vitreous, and fundus can be evaluated.

CHEMICAL RESTRAINT FOR EXAMINATION OF THE EYES

Intravenous sedation accompanied by regional anesthesia are essential components of the examination. Many horses are refractory to ocular manipulation because of their size and strong ocular muscles. We have found the following useful:

Xylazine—0.3 to 0.6 mg/kg IV is suitable for most nonpainful or minimally painful situations where minor restraint is required.

Xylazine and Butorphanol—0.2 to 0.5 mg/kg xylazine is given 2 to 3 minutes before administration of 0.03 to 0.08 mg/kg butorphanol. This combination provides excellent analgesia and may be useful for examination of the eye when there is significant ocular pain.

AURICULOPALPEBRAL NERVE BLOCK

KEY POINT

Blepharospasm can be eliminated by the use of an auriculopalpebral nerve block.

This nerve block does not provide analgesia but is probably the most useful ocular nerve block in the horse. The block is performed using 2 mL of one of the local anesthetics (2% lidocaine, mepivacaine, or prilocaine) (Fig. 11-2). The nerve runs over the highest point of the zygomatic arch and can be palpated easily after moistening the area with alcohol. A 23- or 25-gauge, 15-mm (5/8-inch) needle (without the syringe attached) is placed subcutaneously. This initial "prick" may cause the horse to flinch its head; hence, not having the syringe attached is indicated so that the needle is more likely to stay in place. The horse may also object initially when the local anesthetic is injected. Akinesia of the upper eyelids usually occurs within 5 to 10 minutes.

FRONTAL NERVE BLOCK

The frontal nerve provides sensory innervation to the middle two-thirds of the upper eyelid. Usually this nerve is blocked to place a subpalpebral lavage system or repair superior eyelid lacerations. If the medial and lateral areas need to be blocked, then a line block can be performed inserting the needle into the blocked middle part of the eyelid and threading it along the eyelid, injecting as you go (Fig. 11-3). The frontal nerve is blocked by infiltrating 3 to 4 mL of local anesthetic into the supraorbital foramen of the frontal bone or along the dorsal orbital rim. The foramen itself may have associated blood vessels, so be sure to check that you have not penetrated a blood vessel before injecting the local anesthetic.

TOPICAL ANESTHESIA

Topical anesthesia is useful when performing some examinations, collecting samples, subpalpe-

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Ophthalmic Examination Sheet

Date

Client	**Diagnostic Tests**			
		OD	OS	OD	OS
	Culture			PLR	
Presenting Visual Complaint:	Cytology			Direct	
	Fluorescein			Consensual	
History	Photos			Vision	
	Nasolacrimal irrigation			STT	
	Other			IOP	

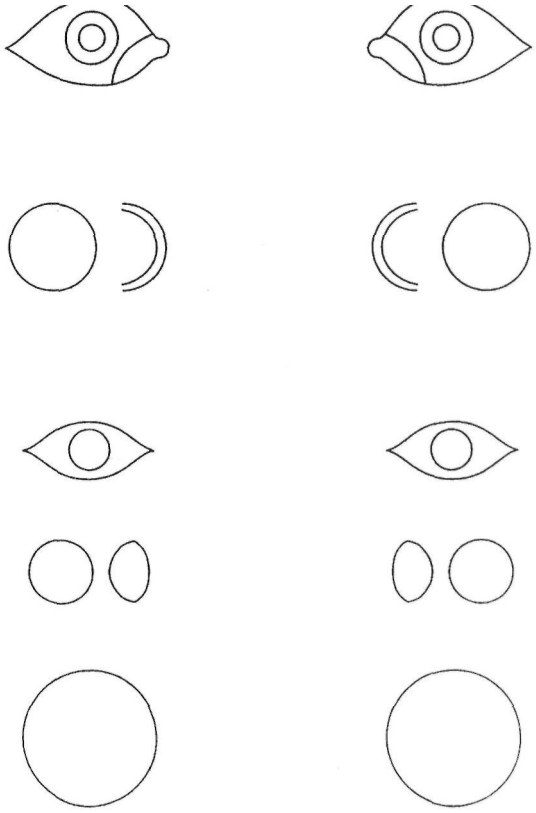
1 Assessment

N = Normal NE = not examined
X = Abnormal NV = not visible

	OD	**OS**
Globe / Orbit		
Eyelids		
Position		
Function		
Nictitans		
Cornea		
Color		
Transparency		
Anterior Chamber		
Contents		
Depth		
Iris/ Pupil		
Shape		
Color		
Lens		
Vitreous		
Fundus		
Optic nerve		
Vessels		
Tapetum		
Non Tapetum		

1 Comments and Illustrations

OD **OS**



Diagnosis:

Treatment:

Revisit:

Figure 11-1. Form used for ophthalmic examination.

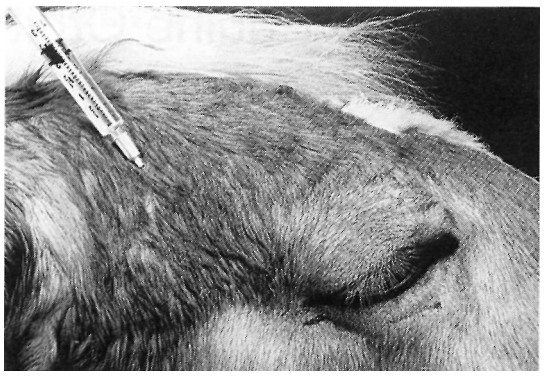


Figure 11-2. Needle position for an auriculopalpebral nerve block. The block is performed using 2 mL of local anesthetic. The nerve runs over the highest point of the zygomatic arch and can be palpated easily after moistening the area with alcohol. Akinesia of the upper eyelids usually occurs within 5 to 10 minutes.

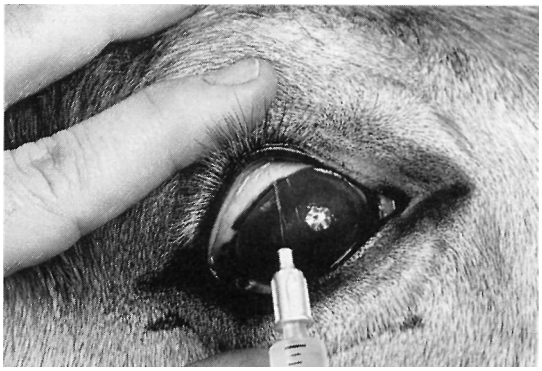


Figure 11-4. Administration of topical liquid medication to the equine eye. Possibly the easiest way to apply all drops to the eye is to gently spray them into the eye. This is achieved by placing 0.2 to 0.3 mL in a 3-mL syringe. Then a 22-gauge needle with the tip broken off is placed on the syringe and the contents irrigated from 2 to 3 cm and at an angle onto the eye.

bral lavage, and for subconjunctival injections. We find the easiest way to apply all drops to the eye is to gently spray them into the eye (Fig. 11-4). This is achieved by placing 0.2 to 0.3 mL in a 3-mL syringe. Then a 22-gauge needle with the tip broken off is placed on the syringe and the contents irrigated from 2 to 3 cm and at an angle onto the eye. Analgesia occurs after about 1 to 2 minutes and lasts about 20 minutes.

Anatomy of Clinical Significance

ORBIT

Some important ocular anatomy is shown in Figure 11-5. The horse has a complete bony orbit; therefore, proptosis is unlikely. Palpation of both orbits concurrently may help diagnose orbital fractures, as shown by crepitus. If radiographs are to be taken, dorsal oblique views are the best to identify fractures or osseous invasion by tumors.



Figure 11-3. Needle position for a frontal nerve block. Three to 4 mL of local anesthetic is infused into the supraorbital foramen of the frontal bone or along the dorsal orbital rim. The foramen itself may have associated blood vessels, so be sure to check that you have not penetrated a blood vessel before injecting the local anesthetic.

GLOBE

The adult equine globe is approximately 38 to 44 mm from anterior to posterior. This distance will change in disease conditions. Comparison of the angle of eyelashes and presence of eversion of the third eyelid are diagnostic aids. Cases of enophthalmos and smaller eyes will have the eyelashes directed more ventrally than the contralateral eye. Conversely, swollen globes or exophthalmos may cause dorsal deviation of the eyelashes. Globe size can also be assessed by ultrasound using A- or IB-mode (7.5-MHz transducer probe) using obstetric lubricant as the coupling agent after topical anesthesia of the cornea.

KEY POINT

Differentiation of exophthalmos (normal sized globe pushed anteriorly due to retrobulbar disease such as tumors) from buphthalmos (enlarged globe due to glaucoma) is essential if an appropriate diagnostic plan is to be formulated.

A small globe present at birth is referred to as microphthalmos and can be unilateral or bilateral. Thoroughbreds are predisposed to this problem.

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Anatomic Areas of Interest of the Equine Orbit

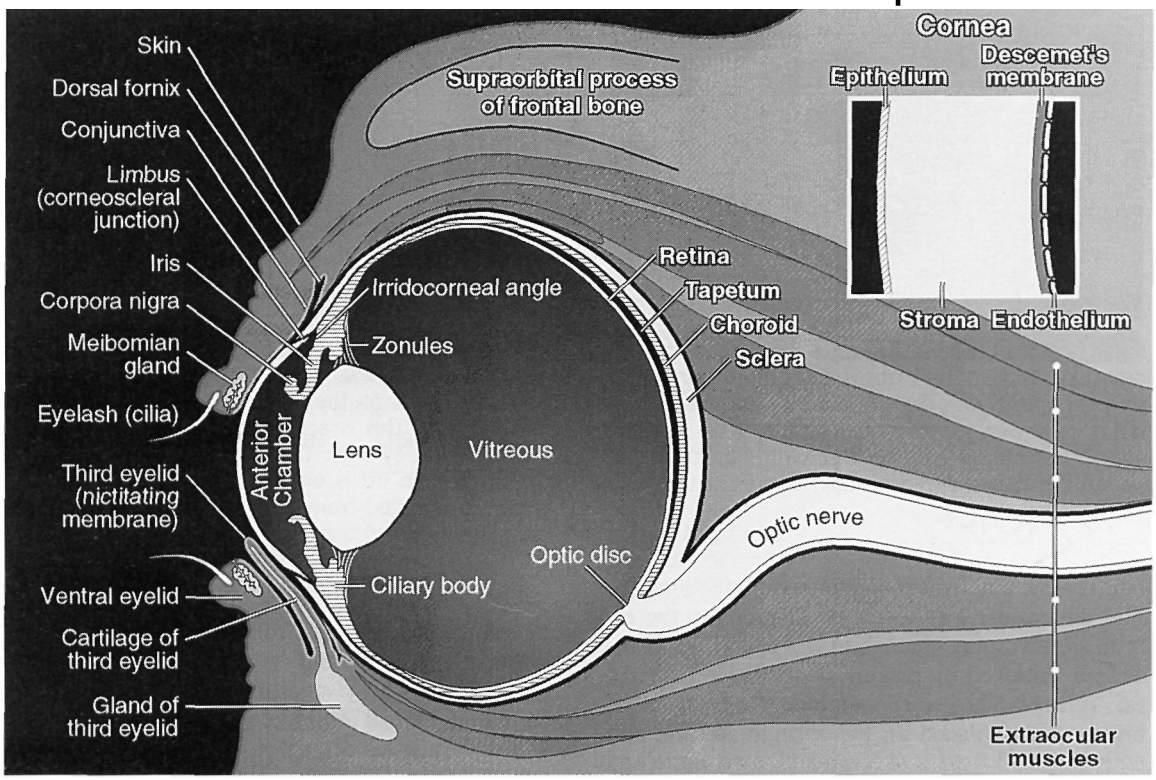


Figure 11-5. Anatomy of clinical significance in the equine eye.

Acquired globe shrinkage, phthisis bulbi, is most often the result of severe inflammation or trauma.

KEY POINT

A small globe needs to be differentiated from enophthalmos (a recession of the globe in the orbit), which is usually a response to ocular pain.

The pressure in the eye is a further diagnostic aid. An estimate of IOP can be made digitally through the eyelids with both eyes being palpated at the same time for comparison. For a more accurate evaluation, a tonometer should be used that can help diagnose glaucoma or uveitis. Normal IOP in the horse is 15 to 30 mm Hg.

EYELIDS

The horse relies more on the upper eyelid to blink than the lower eyelid. The eyelids also have a large vascular supply.

KEY POINT

Surgical treatment of eyelid damage, especially dorsally, is based on minimal tissue resection and excellent anatomic alignment of the eyelid margin.

The symmetry of the eyelids can be assessed by standing directly in front of the horse and comparing eyelids and eyelashes from both eyes at the same time. The eyelids should be palpated with a lubricated gloved hand, after performing an auriculopalpebral nerve block, with one finger inside the eyelid and thumb on the outside. This is especially true for suspected eyelid neoplasms to assess for possible bony involvement.

A blink reflex in response to lightly touching the cornea with a wisp of cotton or by lightly touching the medial/lateral canthus implies cranial nerves V (trigeminal—afferent supply) and VII (facial nerve—efferent supply) are intact.

Eyelids must be carefully inspected for extra hairs such as distichia, ectopic cilia, and for foreign bodies. Vertical corneal ulcers imply eyelid abnormality as the cause.

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KEY POINT

It is essential to look for foreign bodies and abnormal hairs. Excellent lighting and magnification are required.

THIRD EYELID

The third eyelid can be assessed visually by retropulsion of the globe via the dorsal eyelids to cause eversion of the third eyelid. Then using either nontraumatic forceps or a moistened cotton tip applicator, the everted third eyelid can be grasped and the ventral fornix visualized.

KEY POINT

The third eyelid must always be checked if a horse has a corneal ulcer.

EXCISION OF THE THIRD EYELID

Partial or complete excisional biopsies are used mainly for diagnostic and therapeutic reasons, especially in suspected cases of neoplasia. The major complications encountered after surgical excision include retrobulbar fat prolapse and chronic accumulation of mucopurulent debris in the medial canthus due to the large residual dead space. In other species, such as the dog, postoperative dry eye problems have been reported. The steps involved in excisional biopsy of the third eyelid are:

1. In a heavily sedated horse, 2 mL of local anesthetic is injected at three to five sites in the medial canthus with a 22-gauge needle directed into the base of the third eyelid and medial canthus. The needles are not directed toward the globe but are directed toward the medioventral orbital wall.
2. The medial canthus is flooded with topical anesthetic (usually 0.5-1 mL).
3. The third eyelid is then grasped by toothed forceps, and a large curved hemostat is used to clasp deep into the medial canthus at the base of the third eyelid, including the T-shaped cartilage. Sometimes it may be necessary to use two hemostats, one directed from dorsally and one ventrally to encompass all of the third eyelid (Fig. 11-6).
4. Either curved Mayo scissors or a scalpel blade are then used to excise the third eyelid, using the hemostats to cut up against (i.e., once the third eyelid has been excised, the hemostats should still be attached to the remaining stump of third eyelid tissue). Some advocate then using a continuous inverting suture pattern with 5-0 absorbable suture to minimize the risk of retrobulbar fat prolapse; however, we have not found this to be a problem and do not suture the remaining tissue.

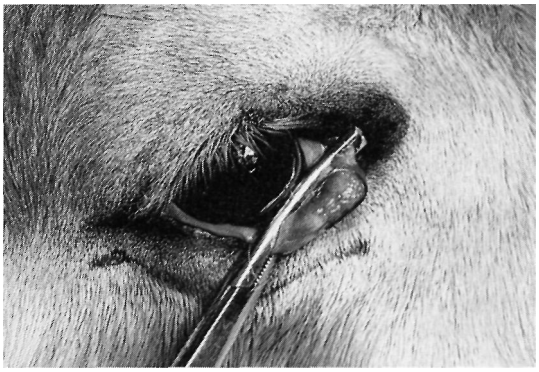


Figure 11-6. Placement of a curved hemostat at the base of the third eyelid before excision after induction of appropriate local anesthesia. The third eyelid is grasped using toothed forceps, and a large curved hemostat is used to clasp deep into the medial canthus at the base of the third eyelid including the T-shaped cartilage. Sometimes it may be necessary to use two hemostats, one directed from the dorsal aspect and the other from the ventral aspect to encompass all of the third eyelid.

LACRIMAL SYSTEM

The nasolacrimal duct has dorsal and ventral puncta located 8 to 9 mm from the medial canthus, both of which converge to the lacrimal sac and pass to the nose, exiting in the external nasal meatus. The nasolacrimal duct lies dorsal to a line drawn from the infraorbital foramen to the nasal canthus. Trephination below this point should not disrupt the nasolacrimal system. The nasolacrimal system can be assessed by

- Fluorescein passage along the nasolacrimal duct. Usually fluorescein appears at the nasal meatus in 3 to 5 minutes. This can be highlighted by using ultraviolet light or a Wood's lamp. (Placing a fluorescein strip in a 3-mL syringe with 2 mL sterile saline or eyewash and attaching a 22-gauge needle with the tip broken off and then irrigating the contents onto the eye is a good method to administer fluorescein to the eye.)
- Mechanical flushing is achieved by inserting a small-gauge intravenous catheter into the nasal openings for retrograde flushing (16- to 18-gauge catheter or nos. 3.5 or 5 Fr urinary catheter) or into the puncta for normograde flushing (20- to 22-gauge catheter or tomcat urinary catheter). Topical anesthesia with lidocaine spray is useful to allow passage of the catheter and allow

retrograde flushing and establish patency of the duct.
- Injection of radiopaque contrast materials may also be used to evaluate the nasolacrimal system radiographically when attempting to localize an obstruction.

Horses rarely develop dry eye problems, but Schirmer tear test values of 15 to 20 mm in 30 seconds in unanesthetized normal eyes have been reported. Occasionally, trauma (facial fractures), drugs, and plant toxins may cause dryness.

CORNEA/CONJUNCTIVA

The normal cornea is avascular and transparent. This is due to perfect alignment of collagen lamellae in the corneal stroma. Anything that disrupts this alignment will cause corneal opacity.

Blue Eye—This is usually due to corneal edema. The fluid enters either due to corneal ulceration or intraocular disease. Therefore, if fluorescein is not retained and the eye is blue, think of intraocular disease as the most likely cause. Focal corneal edema is usually seen with corneal ulceration, whereas diffuse corneal edema implies intraocular disease, as the endothelial cells are affected.

Red Eye—Due to corneal vascularization. If the vessels look like trees with an arborizing pattern, they are usually superficial. If the blood vessels are shorter, brush-like, and near the limbus, they are usually deep and imply intraocular disease.

Other—Neoplasia (especially squamous cell carcinoma and lymphosarcoma), corneal scars (usually white), corneal pigmentation, and cellular infiltrates (such as pus or crystals) may also occur.

KEY POINT

All blue eyes and red eyes should be stained with fluorescein and if possible intraocular pressures measured.

Fluorescein is a water-soluble dye. It is not taken up by the epithelium or Descemet's membrane (deep in the cornea) but is by the corneal stroma.

KEY POINT

A corneal ulcer that does not stain with fluorescein in the center is probably a deep ulcer with Descemet's membrane exposed. Surgical intervention is usually warranted for such deep ulcers or descemetocoeles.

In cases of red eye, blue eye, or suspected ulceration, a sample for microbiologic culture and sensitivity should be taken before any ophthalmic preparation is placed in the eye. Aerobic and fungal cultures should be performed. This is done by rolling a culturette swab across the affected region. Topical anesthetic may then be used to allow cytology to be performed, with either a moistened cotton tip applicator, a Kimura platinum spatula, or with the back of a scalpel blade. A Diff-Quik or Gram stain may then be done to look for bacteria or fungal elements and to characterize the inflammatory response. Often with severe equine ocular disease, the normal gram-positive ocular flora are replaced by gram-negative organisms.

KEY POINT

Antimicrobial therapy should be based on cytology, which is quick and easy to do. Most simple corneal ulcers are complicated by infection with gram-positive bacteria and therefore use of gentamicin, which has mainly a gram-negative spectrum, is not indicated as the first line of therapy.

ANTERIOR CHAMBER

The anterior chamber usually is filled with clear fluid. Look for abnormal contents ventrally in the chamber. If the contents are

Yellow/White—Usually this implies inflammation or fibrin. Do not attempt to drain this.

Red—Hemorrhage, which may be settled (no active bleeding) or diffuse. It also may be associated with fibrin.

Brown or Black—This may be a sign of neoplasia, adhesions of the iris, or iris cysts.

Also look at the depth of the anterior chamber from the side of the eye. Increased depth usually indicates glaucoma or lens abnormalities. A small anterior chamber may indicate the lens has moved forward, adhesions, tumors, foreign bodies, or phthisis bulbi.

IRIS AND PUPIL

The iris surface is not flat but has small folds and furrows. Iris color may vary between horses and between eyes of the same horse. Usually, most irides are brown but can be gold, blue, and white. These variations are often seen in color-dilute breeds such as Appaloosas, Paints, and Pintos. The iris of the horse has a cluster of pigmented spheres dorsally on the pupil margin and some-

times ventrally (corpora nigra or granulae iridicae).

The iris should be assessed with diffuse light for color and topography. Viewing from the lateral aspect of the eye may be beneficial. The pupils are horizontal ellipses and should be compared by standing directly in front of the horse and comparing the bright tapetal/fundic reflections.

PUPIL

Pupillary light reflexes (PLRs) in the horse are slower than most species and often incomplete. A more brisk and greater PLR can be elicited by shining a light onto the temporal/lateral retina from the nasal/medial side of the eye. The PLR requires functioning retina, optic nerve (cranial nerve II) optic tract, midbrain, parasympathetic branch of the oculomotor nerve (cranial nerve III), and iris. The eye only requires a very small amount of functioning retina, however, to elicit a PLR.

KEY POINT

PLRs should always be performed in a dark room using a bright light source.

LENS

The lens is best examined with a fully dilated pupil and shining a light source from laterally (to detect faint anterior opacities) and from in front. Use of the tapetal reflection may assist in seeing subtle lens opacities. Lens fibers grow in such a way that when they meet, they form "suture lines." Usually these are seen as a Y shape in the anterior lens and inverted Y posteriorly but can vary in configuration.

In old horses, a hardening of the nucleus of both lenses causes a "pearl"-like appearance (nuclear sclerosis). The fundus can still be viewed through this normal old age change. These are not cataracts.

VITREOUS

In the normal animal, the vitreous is a clear gel. Examination of the vitreous is facilitated by pupil dilation. Abnormalities include the presence of blood, a gray or white veil (detached retina), or white hyperreflective speckles (asteroid hyalosis).

RETINA AND OPTIC NERVE (FUNDUS)

KEY POINT

To facilitate examination of the fundus, good pupil dilation and a dark room are required.

Diagnostic dilation is achieved within 15 to 20 minutes in most horses with 0.2 mL of 1% tropicamide. (Reluctance to dilate in one eye compared with the other may suggest inflammation or synechiae in the more miotic eye.)

It is important to realize that tremendous variation of what is regarded as being "normal" exists in the fundus of most species. Therefore, if you are unsure if what you are seeing is normal or abnormal, compare the findings with the opposite eye, and if still unsure compare with another animal of similar coat color, breed, and age.

What To Look for

Optic nerve

- Size, shape—usually circular or slightly oval
- Color—usually salmon pink with 40 to 60 blood vessels extending one to two disc diameters from the optic nerve
- Location—at the junction of tapetum/nontapetum

Retina

- Tapetum—located dorsally (note the red/brown spots evenly spread, these are end on capillaries called the Stars of Winslow)
- Reflectivity—hyper- or hyporeflective
- Color—may be yellow, green, blue, or red

KEY POINT

Some color-dilute horses may lack a tapetum, in which case the redness seen ophthalmoscopically may be the underlying choroidal blood vessels.

- Nontapetum—located ventrally and in most breeds is a homogenous brown/black color (some color-dilute animals, which also often have blue irides, have little brown pigment and this reveals the underlying choroidal vessels).

What To Use for Fundic Examination

We scan the whole retina with indirect ophthalmoscopy, and if a lesion needs to be more carefully evaluated, then use direct ophthalmoscopy.

Indirect Ophthalmoscopy (Fig. 11-7) requires a focal light source or headset and a 14 or 20 diopter lens. Use of a 14 diopter lens provides more magnification. Indirect ophthalmoscopy provides an inverted reversed image (i.e., the tapetum appears ventral and a medial lesion in the retina will appear lateral). A 20 diopter lens provides about 0.8 times magnification (i.e., the image is smaller than actual size), whereas a 14 diopter lens provides somewhere between 1.2 and 1.8 times magnification. The benefit of indirect oph-



Figure 11-7. Indirect ophthalmoscopy using a focal light source or headset and a 14- or 20-diopter lens. This provides an inverted reversed image (i.e., the tapetum appears ventral), and a medial lesion in the retina will appear in the lateral field of view.

thalmoscopy is that it provides a wide field of vision, which allows a quick examination. Furthermore, the observer's head is not near the horse.

Direct Ophthalmoscopy (Fig. 11-8) may be the most popular way to view the fundus due to its simplicity but is not necessarily the best. The fundus is observed with a setting of 0 to —3 (usually negative numbers are in red, positive in black). These numbers represent the diopter setting or focal length. One diopter equals 1.3 mm in the horse (i.e., a mass on the retina that is in focus at 0 diopters, whereas the rest of the fundus is in focus at — 3 diopters is elevated 4.9 mm off the retina). The advantages of direct ophthalmoscopy are its portability, provision of an upright magnified image (8 x), and relative ease of use. The disadvantages are that it is slow (only a small area is examined at a time) and the observer has to be about 2 to 5 cm from the animal's eye, which predisposes the observer to injury if the horse is fractious.



Figure 11-8. Direct ophthalmoscopy for examination of the fundus. The fundus is normally observed with the ophthalmoscope setting at 0 to -3 diopters.

Principles of Therapeutics

The main objective of any drug treatment is to maintain a therapeutic drug tissue level to achieve the desired pharmacologic effect. In the horse this may be difficult because of the often uncooperative nature of the horse, possibly resulting in damage to its eye and injury to personnel administering the treatment.

The type of formulation and route of administration of medications depend on the location of the lesion to be treated, frequency of treatment required, and compliance of the animal. To decide on the route of administration, consider using the following (listed in order of preference):

Eyelids—systemic, topical therapy.
Conjunctiva—topical, nasolacrimal/subpalpebral lavage systems, systemic to supplement topical, subconjunctival injections.
Cornea—topical, nasolacrimal/subpalpebral lavage systems, subconjunctival injections, if vascularized then systemic.
Iris and Ciliary Body—topical, systemic
Retina, Choroid, and Optic Nerve—systemic

KEY POINT

The efficacy of topical medications can be improved by either increasing the frequency of drug application or increasing the concentration of the drugs.

TECHNIQUES FOR OCULAR THERAPY

We generally place subpalpebral lavage systems rather than lavage via the nasolacrimal duct. Physiologically this is the best technique, because the nasolacrimal duct is the outflow path of ocular debris. Furthermore, the nasolacrimal lavage requires greater volumes of medications to be used. Only in cases of eyelid abscesses, the result of leakage of ocular medications subconjunctivally, will we use nasolacrimal lavage systems.

Subpalpebral Lavage

We prefer to use silastic tubing for the subpalpebral lavage because it causes less irritation than

other materials. A subpalpebral lavage apparatus can be placed after induction of heavy sedation with local and topical anesthesia or under general anesthesia. We have found that when placing these under general anesthesia, caution must be exercised, because it is very easy to place the tubing too low in the eyelid, which is only apparent once the horse recovers from anesthesia. We palpate up into the fornix with a gloved lubricated finger and then guide the trocar or needle into the area (Fig. 11-9 A).

KEY POINT

A subpalpebral lavage system must be placed as high up into the dorsal fornix (the blind pouch between the eyelids and globe) as possible; otherwise, corneal ulceration will result. Try to minimize the amount of tunneling in the subconjunctival tissue before the exit of the tubing through the eyelid skin.

The 14- or 16-gauge needle is driven up through the eyelids in two places, one medial and one lateral, and the tubing is threaded through the needle (Fig. 11-9B). We prefer to keep the short end on the lateral side so that it does not interfere with subsequent auriculopalpebral nerve blocks. The short end is kept in place by stents or by tying knots in the tubing. The long end, which is exiting medially, is sutured into place on the forehead with 2-0 nylon and using 2-cm-wide tape to secure it. It is then directed over the poll and is

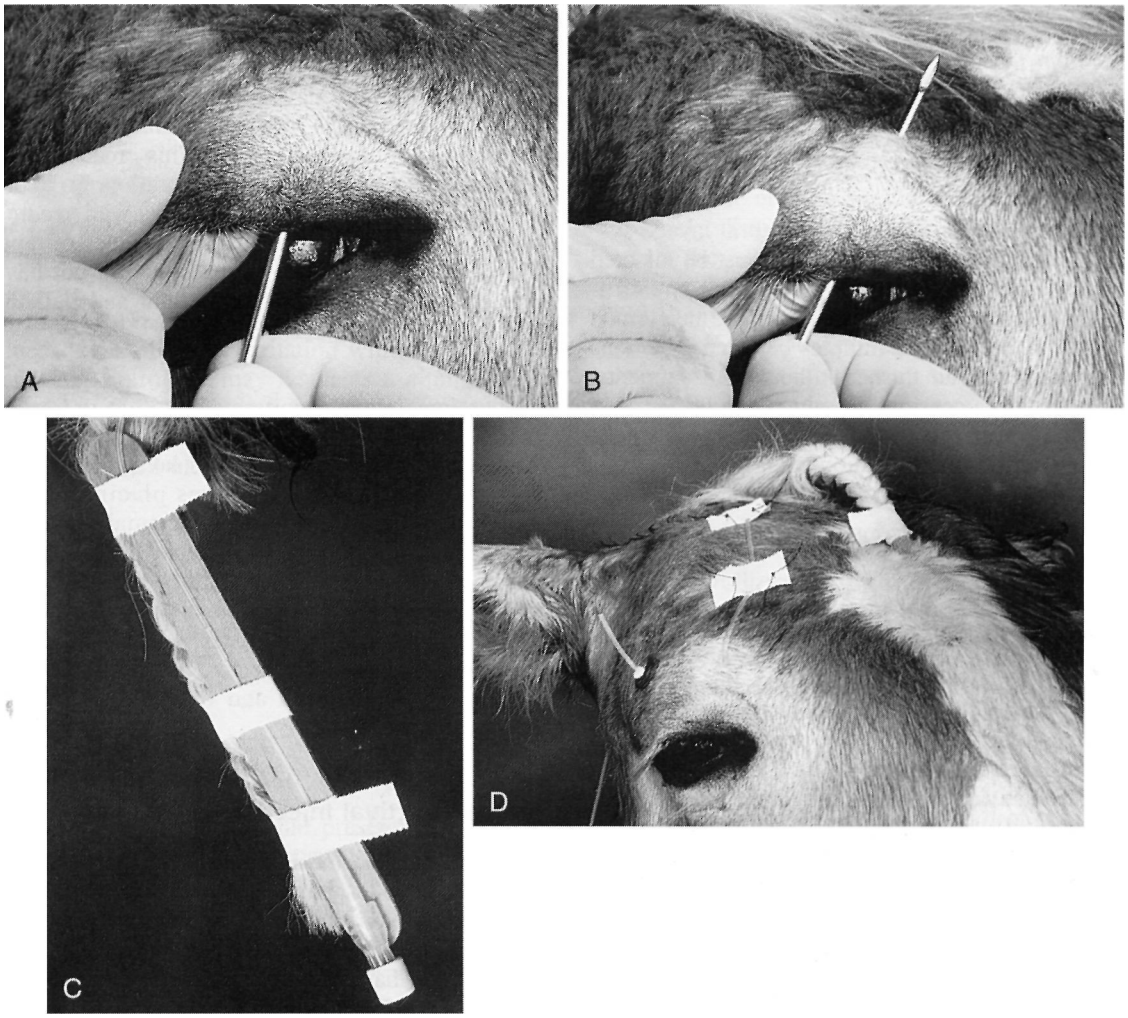


Figure 11-9. Subpalpebral lavage system. *A,* Guiding the trocar into the dorsal fornix for a subpalpebral lavage system. *B,* Driving the trocar through the dorsal fornix. C, The injection port apparatus located on the wither and attached to the mane. *D,* The appearance of the subpalpebral lavage system, showing the correct tension of the tubing, the use of a button stent, and attachment to the forehead.

incorporated into mane braids as it courses down the neck to the wither. The end is cannulated with a tomcat catheter, and an injection port is then placed. This is all secured in place by taping it to a wooden tongue depressor on the mane (Fig. 11-9C). Before finally securing it into place, some small holes must be made in the part of the tubing in the dorsal fornix by simply advancing a piece of the tubing out the short end (laterally) and either cutting some holes or using a 22-gauge needle to punch holes.

KEY POINT

Make sure the subpalpebral lavage holes are discharging into the fornix by flushing the system with fluorescein. Ordinarily, the fluorescein should exit the medial canthus. Also, check the exit points for the eyelids for signs of leakage. Some drugs, especially natamycin, are extremely irritating if they gain access to subconjunctival tissues.

COMPLICATIONS OF THE SUBPALPEBRAL LAVAGE

Daily examination of the subpalpebral lavage system is required. The medications must be injected very slowly, because horses quickly become difficult to work with if the injections are performed quickly. A period of 5 minutes between injections is advised, with air flushed in between treatments. We have had occasions in which the lavage systems have been left in for periods of 4 to 6 weeks with no serious complications.

DAILY MAINTENANCE OF A SUBPALPEBRAL LAVAGE SYSTEM

The important points in maintaining a subpalpebral lavage include the following:

1. Check the injection port at the wither to ensure there is no leakage from the connections, the tongue depressor has not broken, and there is no resistance to injection of fluid into the system. If resistance to fluid injection is present, the obstruction is usually due to debris blocking the holes in the tubing in the fornix. If this is the case, then slide some of the tubing out of the eyelid through the short end, so the irrigation holes created in the tubing can be inspected and either forcefully cleared by injecting sterile saline into the injection port using a 6-mL syringe or by creating new holes. If this is attempted while the lavage is in its normal position, the horse will violently object, making subsequent medications more difficult to administer. This is also avoided by flushing air in between medications so that precipitates do not form.

2. Ensure that the medications injected into the injection port are discharging into the fornix and not into the subconjunctival tissue or at the entry and exit points of the tubing into the eyelid. If this is occurring, then either readjust the tubing or slide the piece of tubing through the short end and retie it and create new holes. We have found to avoid this problem, it is best to place the original holes in the eyelids quite a distance apart, so the amount of tubing in the fornix is at a maximum. Also, it is best not to place the holes in the tubing itself too far apart. If injected material gains access to the subconjunctival tissue, eyelid abscesses may develop. These are best managed with hot packing every 6 hours, nonsteroidal antiinflammatory drugs, systemic antibiotics, and removal of the subpalpebral lavage tubing. There can be spectacular swelling due to the excellent blood supply to the eyelids.

3. A potentially devastating complication may occur if the lavage system becomes loose and dangles down onto the cornea. This creates irritation and can result in corneal ulceration. To avoid this, ensure that the lavage system is well secured onto the forehead with at least two pieces of tape. The lavage system once in place should not have any slack nor should it be too tight and pull the eyelid upward (Fig. 11-9D).

Nasolacrimal Lavage

An alternative to a subpalpebral lavage is a nasolacrimal lavage. This involves placing a fine-gauge catheter (Equine Nasolacrimal Cannula, Jorgensen Laboratories, Loveland, CO) via the nasolacrimal punctum. To secure it in place involves taking the tube through the false nostril before insertion into the distal puncta. An extension tube is then connected to travel over the middle of the forehead and over the poll to be attached at the wither.

Subconjunctival Injection

After topical anesthesia and an auriculopalpebral nerve block, a 23-gauge or 25-gauge, 15-mm (5/8-inch) needle is introduced under the dorsal bulbar conjunctiva and treatment given. When injecting, it is best to have the needle at an oblique angle rather than perpendicular to the eye. As a general rule, no more than 1 mL should be injected in one site. A few subconjunctival drug doses are given.

Antibiotics—amikacin, 25 mg; cephaloridine

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or cefazolin, 100 mg; gentamicin sulfate, 20 mg; penicillin G, 500,000 IU.

Antifungals—miconazole, 20 mg; nystatin, 5000 mg

Autonomic drugs—atropine sulphate, 15 mg

Corticosteroids—betamethasone, 15 mg every 3 weeks; dexamethasone, 2 mg every 2 days; triamcinalone, 40 mg every 3 to 7 days; methylprednisolone acetate, 40 mg every 1 to 3 weeks. Subconjunctival granulomas have been associated with some depository steroids.

KEY POINT

Long-acting steroids are potentially extremely dangerous in the eye. Rule out corneal injury and infection first.

Diseases of the Eyelids

Entropion

KEY POINT

Entropion in the foal is usually a secondary phenomenon to some other systemic problem.

It may be bilateral or unilateral and usually produces signs of ocular irritation within a few days of birth. Entropion may result as a complication of malnutrition, dehydration, or septicemia due to the resulting enophthalmos. If untreated, it may lead to corneal ulceration and subsequent perforation.

HISTORY AND PRESENTING SIGNS

- Excess lacrimation
- Blepharospasm
- Photophobia

CLINICAL FINDINGS AND DIAGNOSIS

- Any young foal with the presenting signs detailed above should be suspected of having entropion.
- Close examination of the eye will reveal inversion of the lower eyelid(s) margin.
- In some cases there may be corneal ulceration from abrasion due to the eyelashes.
- Topical anesthetic in cases of secondary entropion may correct the problem but will not improve cases of congenital entropion.
- Fluorescein staining is essential.

KEY POINT

Repeated applications of topical anesthetic is toxic to the cornea and is contraindicated in treatment.

DIFFERENTIAL DIAGNOSIS

- Eyelash disease
- Keratitis
- Conjunctivitis
- Trauma
- Septicemia with uveitis in both eyes (look for miosis, and hypopyon in the anterior chamber)

TREATMENT

KEY POINT

Most cases resolve with conservative therapy, which may be as simple as eversion of the lower eyelid margin and use of antibacterial eye ointment if there is corneal ulceration.

- In more severe cases, mechanical eversion of the eyelid margin is required. This can be performed using saline or local anesthetic injected subcutaneously. Supergluing folds of eyelid skin to evert the affected area may also help. Avoid getting the glue into the eye.
- Surgical correction of entropion may be required. A temporary vertical mattress suture with 3-0 silk is effective.

KEY POUT

Skin resection to provide permanent eversion rarely is needed.

Eyelid Swelling (Blepharoedema)

The flighty nature of some horses predisposes them to orbital trauma and subsequent blepharoedema. Systemic illness may also cause bilateral blepharoedema.

KEY POINT

The extremely vascular nature of the eyelids means that blepharoedema develops very rapidly and conversely decreases quickly too.

HISTORY AND PRESENTING SIGNS

- Acute onset of eyelid swelling in cases of blunt trauma, more insidious development in cases of systemic disease
- Blepharospasm
- Excessive lacrimation

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CLINICAL SIGNS AND DIAGNOSIS

- Warm edematous eyelids
- Orbital fractures (orbital palpation needed)
- Fundic examination for retinal detachment
- Fluorescein stain
- Complete blood count and biochemical analysis if systemic disease suspected
- Inability to blink correctly (i.e., ensure the central cornea is covered by a complete blink)

DIFFERENTIAL DIAGNOSIS

- Blunt facial trauma
- Acute allergic reactions
- Insect/snake bites
- Systemic diseases that may cause cellulitis or protein loss

TREATMENT

- Systemic nonsteroidal antiinflammatory drugs (NSAIDs) (e.g., phenylbutazone 2.2 mg/kg q12h or flunixin meglumine 1 mg/kg q12h) for 3 days, then the dose rate tapered.
- Systemic antimicrobials if infectious agent suspected.
- Hot packing four to six times daily (we find placing a bran mash inside a rectal palpation glove then evenly microwaving the pack is excellent—check the temperature in the middle of the glove before application).
- Topical ointments to protect the cornea.

Eyelid Lacerations

Eyelid lacerations are very common because horses tend to rub their eyes on fences, feed bins, and other objects that result in trauma.

KEY POINT

Although many lacerations appear quite severe, because of the excellent blood supply, most will heal with appropriate treatment. It is important that all vital tissue be preserved and that wound debridement be kept to a minimum.

The major objective is anatomic realignment of the tissues so that distortion of the eyelids does not occur and result in corneal ulceration.

HISTORY AND PRESENTING SIGN

- Obvious trauma

CLINICAL FINDINGS AND DIAGNOSIS

- Diagnosis is not a problem with eyelid lacerations, but assessment of the extent of the laceration and decisions regarding treatment are critical to a cosmetic and functional result.
- Profound sedation is required for assessment and treatment, and we often use a combination of xylazine HCl and butorphanol given IV. If there is difficulty in restraint, xylazine and ketamine anesthesia or "triple-drip anesthesia" may be required.

DIFFERENTIAL DIAGNOSIS

- Assessment of any corneal trauma

TREATMENT

Medication

- Systemic NSAID therapy is useful. The object is to promote wound healing, so corticosteroids should be avoided. Nonsteroidal preparations are given orally (e.g., phenylbutazone 2.2 mg/kg ql2h or flunixin meglumine 1 mg/kg q12h for 3 days, then q24h for 5-7 days) to prevent extensive swelling. (If in doubt about systemic effects of NSAIDS, check oral mucosa for ulceration, perform hematocrit and plasma total protein analysis, blood urea nitrogen and creatinine concentrations, and possibly urinalysis).
- Systemic antibiotics are indicated.
- Topical antibiotics are indicated if corneal ulceration has occurred.
- Tetanus prophylaxis is essential, as for any wound.

Surgical Repair

KEY POINT

Do not remove flaps of skin unless the tissue is necrotic.

- Skin preparation is necessary because the skin frequently is infected. This is best achieved with diluted povidone-iodine (1 part povidone-iodine to 3 parts saline for the skin [avoid contact with the ocular tissues otherwise severe edema will result]; 1 part povidone-iodine to 30 parts saline for the conjunctiva and cornea); irrigate and use a gentle rubbing action to clean the tissues for 2 to 3 minutes, then lavage with sterile saline, especially the cornea and conjunctiva.
- Repair should involve minimal debridement; scarify necrotic margins and smooth jagged edges in preparation for wound closure.
- Do not take a full thickness bite with the subcutaneous suture. A figure of 8 suture in the eyelid margin is ideal, with subsequent simple interrupted sutures incorporating the ears of the figure of 8 suture to direct them away from the cornea.

KEY POINT

Alignment of the eyelid margins must be exact and should be done first, because poor alignment of the eyelid margins often leads to corneal ulcers. A two layer closure is also a necessity.

- The conjunctiva usually does not require suturing, and 3-0 to 5-0 absorbable suture material should be used in the fibrous tarsal plate found deep in the subcutaneous tissue and the skin.
- Even if the wound is more than 24 hours old, attempt a primary closure if possible after minimal debridement.

Habronemiasis

Habronema infestation commonly involves the eyelids and conjunctiva. Lesions are found most commonly near the medial canthus of the eye. Flies serve as vectors.

HISTORY AND PRESENTING SIGNS

- Yellow caseous gritty foci containing mineralized necrotic tissue near the eyelids
- Usually in summer

CLINICAL FINDINGS AND DIAGNOSIS

KEY POINT

Typical habronemiasis results in granulomatous reactions, with the lesions having a caseous appearance.

- It is quite common to find lesions in a number of sites, particularly the prepucial area in geldings and stallions.
- The lesions are often pruritic and painful.
- Habronemiasis has a typical appearance, and biopsy samples are seldom required, unless there is concern about the possibility of neoplasia.
- Cytology of affected areas reveals numerous eosinophils, mast cells, neutrophils, and plasma cells. Larvae are rarely seen.

DIFFERENTIAL DIAGNOSIS

- Sarcoid
- Neoplasia (e.g., squamous cell carcinoma, mastocytoma)
- Fungal granuloma

TREATMENT

KEY POINT

Ivermectin is effective in the treatment of most lesions when given orally at a dose rate of 0.2 mg/kg.

- The host response to dying larvae is sometimes quite dramatic, with severe irritation being present. In these cases, topical ophthalmic corticosteroid drops can be used.

Sarcoid

KEY POINT

Any mass around the eye should be suspected of being a sarcoid.

HISTORY AND PRESENTING SIGNS

- Slowly progressive enlargement of a mass or masses close to the eye.
- Quarterhorses have a higher incidence than Thoroughbreds or Standardbreds.

CLINICAL FINDINGS AND DIAGNOSIS

- Raised mass of tissue that is either covered with epithelium or appears similar to granulation tissue. The overlying skin may lack hair.
- Although clinical appearance may suggest a sarcoid, definitive diagnosis is made histologically.

DIFFERENTIAL DIAGNOSIS

- Habronemiasis
- Squamous cell carcinoma
- Viral papillomatosis
- Trauma
- Parasitic granuloma

TREATMENT

- Immunotherapy is most successful in our experience, and the injection of a mycobacterial cell-wall extract (Equimune, Vetrepharm, Athens, GA) into the base of the lesion has given excellent results.
- The dose that we have used is 1 mL/cm^3 of tissue. The lesion is debulked before injection, and the volume of the tissue remaining is estimated so that an appropriate volume of solution is injected.
- Repeat injections, usually three to four are needed, once the inflammatory response has decreased (usually 2-3 weeks).
- Subsequent injections tend to promote a pro-

found local inflammatory response 24 to 48 hours after injection. In these cases, systemic administration of flunixin meglumine at a dose rate of 1 mg/kg is indicated.

KEY POINT
Warn the owners about the possibility of anaphylaxis when injecting foreign proteins.

- Up to 100% success rates and good cosmetic results have been reported with bacille Calmette-Gueren (BCG) therapy.
- Cryosurgery has a success rate of 60 to 70%, but great care is required because of the possibility of damaging the cornea by freezing. Lesions should be frozen to —25°C (use thermocouples deep into the mass if possible), and two freeze-thaw cycles should be used.

KEY POINT
After cryosurgery, hair may depigment at the treated site, and if overzealous treatment occurs, extensive cicatricial tissue may result and eyelid necrosis can occur.

- Surgical excision alone is associated with up to 50% recurrence.
- Radiation therapy, often in combination with surgical debulking, offers up to 90% success rates but is limited by the expense, hazards of radioactive isotopes, need for postoperative isolation, and licensing requirements.

Squamous Cell Carcinoma

KEY POINT
Squamous cell carcinoma (SCC) is one of the two most common forms of neoplasia involving the eyelids, nictitating membrane, and third eyelid.

Horses most at risk are those with little or no pigment around the eyelids. The most common sites affected are at the medial canthus, especially the conjunctiva and third eyelid. The cornea and eyelids may also be affected. There is a predilection for Appaloosas and draft breeds. SCC is characterized by a low metastatic potential to regional lymph nodes (about 10%) but is aggressive locally. Furthermore, 10% of horses have SCC on another part of the body if the eye is involved. SCC associated with the bony orbit, and to a lesser extent the eyelids, has a poor prognosis, whereas SCC of the cornea and conjunctiva has a better prognosis.

KEY POINT
Do not forget to palpate the regional lymph nodes (especially retropharyngeal and submandibular) and to auscultate the chest for evidence of metastasis. Careful palpation of the bony orbit and dorsal oblique radiographs of the orbit are also necessary.

HISTORY AND PRESENTING SIGNS

- Ulcerative lesion around the eyelid margins.
- Nonpigmented skin at the eyelid margins.
- About 15% have bilateral involvement.
- The average age of affected animals is between 8 and 10 years.
- Geldings are at an increased risk of developing SCC compared with mares and stallions.

CLINICAL FINDINGS AND DIAGNOSIS

- Ulcerative skin lesions close to the eyelid margins are suggestive of SCC.
- In some cases there is involvement of the third eyelid, and this area should be examined closely by applying pressure on the eyeball through the upper lid to evert the third eyelid (retropulsion).
- A biopsy is essential for diagnosis.

KEY POINT
Check the opposite eye and perineal and prepucial areas for signs of tumors.

DIFFERENTIAL DIAGNOSIS

- Habronemiasis
- Sarcoid
- Trauma
- Other neoplasms (e.g., melanoma, lymphosarcoma, fibrosarcoma, mastocytoma, adenoma and adenocarcinoma, hemangioma and hemangiosarcoma) have all been reported. Specific therapy and prognosis depends on tumor type, tumor site, and extent.

Treatment

- Surgical excision alone results in 55% recurrence rates.
- A combination of surgical excision and radiation therapy or cryotherapy provides excellent results.
- Immunotherapy with cell-wall extracts (BCG) is reported to be of value in some cases (see section on treatment of sarcoid).
- Intralesional injections of cisplatin in a water-oil emulsion at 1 mg cisplatin/cm^3 of tumor has

been used (four treatments, 2 weeks apart). The emulsion is obtained by mixing 10 mg of cisplatin in 1 mL of water and 2 mL of purified sesame oil for 1 minute. Tetanus prophylaxis is advised before treatment.
- Topical 5% 5-fluorouracil has been used for SCC elsewhere on the body in combination with surgical debulking. The benefit of this treatment is that it is applied topically every 24 hours for 14 days, then once every 2 weeks. 5-Fluorouracil is an antimetabolite, and owners must wear gloves when applying it. The authors have not had significant experience with this form of treatment to advise on the efficacy of its use.
- Removal of the third eyelid is indicated with lesions affecting this site (see section on ophthalmic examination).
- More extensive tumors may require removal of the entire eye.
- Orbital involvement has a poor prognosis.

Diseases of the Cornea and Conjunctiva

Conjunctivitis

Conjunctival disease may be associated with a wide range of ocular and nonocular processes in the horse.

KEY POINT

As in most species, bacterial conjunctivitis is usually a secondary process. Look for an inciting cause.

HISTORY AND PRESENTING SIGNS

- Most common in summer with dusty conditions and flies
- Allergic or irritant conjunctivitis is routinely encountered and is prevalent in stabled horses with straw, dust, and dirt in their environment
- Viral respiratory infection
- Enzootic outbreaks may occur
- Pruritus

CLINICAL FINDINGS AND DIAGNOSIS

- Mucopurulent ocular discharge with evidence of inflamed and injected conjunctiva.
- May be unilateral or bilateral.
- Bacterial culture and sensitivity testing and viral isolation can be performed on samples collected from the conjunctiva.

KEY POINT

Fluorescein staining should be undertaken to determine whether there is corneal ulceration and is also helpful in determining whether there is obstruction of the nasolacrimal duct.

- Conjunctival depigmentation is seen with *Onchocerca.*

DIFFERENTIAL DIAGNOSIS

- Keratitis
- Uveitis
- Habronemiasis
- Onchocerciasis

TREATMENT

KEY POINT

Treatment of conjunctivitis is based on identifying the underlying cause.

- Local therapy is indicated with moistened gauze sponges to remove purulent material.
- If the conjunctivitis is secondary to viral respiratory disease, the discharge is usually serous, and no treatment is required.

Corneal Ulceration

Corneal ulcers are among the most common of all ocular diseases in the horse. Almost all are initiated by trauma. The microbial agents of most importance are usually gram-negative bacteria or fungi, but this varies in different geographic regions. The importance of gram-positive bacteria in this condition should not be underestimated because these organisms constitute much of the normal flora and are often the first to infect the damaged cornea. It is important to note that anterior uveitis usually accompanies corneal ulceration, so that treatment of even simple corneal ulcers with atropine and an antimicrobial agent is indicated.

KEY POINT

Cytology should be performed on all corneal ulcers. Aerobic and fungal cultures should be considered essential for all nonhealing corneal ulcers.

HISTORY AND PRESENTING SIGNS

- Photophobia
- Blepharospasm
- Excess lacrimation

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- Purulent ocular discharge

CLINICAL FINDINGS AND DIAGNOSIS

- The presenting signs described are found in a range of ocular diseases and therefore are of no help in making a specific diagnosis.
- Important signs include
  - o Corneal edema (blue eye), which initially is regional but as anterior uveitis develops may become more diffuse
  - o Corneal vascularisation (red eye)
  - o Miosis (constricted pupil)
  - o Inflammatory debris in the anterior chamber
  - o Ulceration as determined by fluorescein stain

KEY POINT

An auriculopalpebral nerve block is required to permit examination of the cornea and subsequent bacterial and fungal culture. Topical anesthetic may then be applied to desensitize the cornea for the collection of corneal scrapings for cytology.

- Although a number of organisms are involved in corneal ulcers, the most dangerous are gram-negative bacteria and fungi because of the potential for rapid progress of the ulcer with ultimate corneal rupture. It is of critical importance to assess the depth of the ulcer, because deeper ulcers may quickly become full-thickness defects and lead to iris prolapse and globe collapse. Deep ulcers often necessitate surgical intervention, especially if there are no corneal blood vessels to the region.

KEY POINT

Examine behind the third eyelid for the presence of a foreign body that may be producing ulceration—more is missed by not looking than not knowing!

DIFFERENTIAL DIAGNOSIS

- Corneal foreign bodies
- Stromal abscess
- Conjunctivitis
- Severe uveitis
- Immune mediated keratouveitis (equine recurrent uveitis)

TREATMENT

KEY POINT

Topical corticosteroids should never be used in the treatment of corneal ulcers because of the likelihood of worsening the ulcer and leading to rupture of the cornea.

Bacterial Keratitis

- Topical medical therapy (q6h) is effective for most uncomplicated superficial ulcerations. The antibiotics of first choice are chloramphenicol 1% or a neomycin-polymyxin B-bacitracin combination. Some horses may react to neomycin and develop blepharitis and blepharospasm.

KEY POINT

Initial antibiotic therapy should be based on cytologic results and Gram stains at the time of examination.

- Ocular pain can be managed using a cycloplegic such as atropine (every 2-4 hours for the first 24-48 hours) until there is mydriasis. Ideally atropine should be used enough to cause dilation but not enough to cause systemic side effects, such as colic. In addition to the atropine, systemic NSAIDs are used to control ocular pain. The agent of choice is flunixin meglumine administered at a dose rate of 1 mg/kg IV once or twice daily. In our experience, phenylbutazone is not as effective as flunixin meglumine to control ocular pain and anterior uveitis.

KEY POINT

In using atropine, beware of systemic effects, such as increased heart rate, gastrointestinal stasis, and dilation of the opposite pupil. When using flunixin meglumine for long periods, monitor the plasma total protein concentration. A decrease in protein, indicative of gastrointestinal ulceration, is one of the first signs of NSAID toxicosis.

- Chronic or progressive ulceration requires anticollagenase therapy due to the production of autolytic enzymes by bacteria/fungi and local inflammatory cells that can cause rapid corneal rupture. Agents such as 20% acetylcysteine (Mucomyst, Mead Johnson, Evansville, IN) diluted to 5 to 8% with artificial tears and EDTA plasma have been used successfully in combination with other therapeutic strategies. Serum is also useful as an anticollagenase.
- In treating corneal ulcers, a nasolacrimal or subpalpebral lavage system should be used to avoid irritation and pain associated with the administration of drops or ointments.
- If there is necrotic debris associated with the ulcer, it may be necessary to debride the necrotic material.

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- Systemic antibiotics may be indicated in severe ulcers, or where there are corneal stromal infections if corneal blood vessels have progressed to the affected site.
- In some corneal ulcers, a partial lateral tarsorrhaphy (suturing the eyelids) is helpful to provide protection for the cornea, while still allowing vision medially.
- More extensive ulcers may require a conjunctival flap. These flaps provide mechanical protection and also increase vascularity and supply of anticollagenase to the region.
- Pupillary dilation and a reduced degree of corneal edema are good indicators of the control of the intraocular disease.

Mycotic Keratitis

- Fungal keratitis is uncommon but sometimes is found after combined topical antibiotic/corticosteroid therapy that have allowed fungi to proliferate.

KEY POINT

Combined antibiotic/steroid therapy is rarely indicated in veterinary ophthalmology.

- The common fungal species include *Aspergillus* spp., *Penicillium* spp., *Fusarium* spp., and *Alternaria* spp. Similar signs are found as with bacterial keratitis, and diagnosis is made on the basis of cytology (central and peripheral scrapings), culture, and histopathology.
- Loss of the eye is a common sequel to fungal infection if aggressive therapy is not instituted. Agents that can be considered include natamycin ophthalmic preparation and miconazole (10 mg/ml IV solution applied directly to the eye) at least every 6 hours for up to 6 weeks. Most topical antifungal agents are fungostatic, and usually for healing to occur the cornea relies on the ingrowth of corneal blood vessels to allow phagocytes to kill the fungi present.
- Aggressive cycloplegic, anti-inflammatory, and anticollagenase therapy described above must be instituted.

KEY POINT

The prognosis for recovery in the case of fungal keratitis is guarded.

- After initiating antifungal therapy, anticipate an initial exacerbation of clinical signs as fungal death stimulates a strong inflammatory reaction.
- Medical management typically takes 6 to 8 weeks of treatment with corneal scarring common.
- Surgical resection of the affected cornea and a conjunctival graft often are needed, because the associated anterior uveitis cannot be controlled sufficiently in the time needed to allow the ingrowth of the corneal blood vessels.

Anterior Segment Trauma

Because of the fractious nature of horses and their prominent eyes, trauma to the cornea is common. This may be blunt or sharp in origin. The ocular damage that ensues is usually a result of corneal endothelial damage, anterior uveitis, lens luxation, and retinal detachments. Prompt recognition of the underlying disease and appropriate treatment of traumatized eyes can yield successful outcomes.

HISTORY AND PRESENTING SIGNS

- Often the owners of the horse will be aware of an acute injury necessitating attention
- Photophobia
- Blepharospasm
- Epiphora
- Corneal edema
- Possible iris prolapse

CLINICAL FINDINGS AND DIAGNOSIS

- As with corneal ulceration, the clinical signs seen with anterior segment trauma are present in a variety of ocular diseases.
- It is important to establish globe integrity, because sharp and blunt trauma can result in perforations of the cornea and sclera. Be sure to evaluate the sclera as much as possible (i.e., heavily sedate the horse and use an auriculopalpebral nerve block to elevate the eyelids, allowing close inspection of the sclera).
- Signs may be compatible with anterior uveitis, lens luxation, and retinal detachments (see relevant sections below).

TREATMENT

- This mainly involves identification and treatment of the underlying disease problem that has resulted from the trauma. If a rupture of the globe is present, in either the cornea or sclera, surgical intervention is necessary. Usually aggressive NSAID drug therapy and atropine are required to control the associated anterior uveitis.

Uveal Diseases

Diseases of the uveal tract (iris, ciliary body, and choroid) may occur as primary or secondary disor-

ders. Uveitis may occur secondary to severe keratitis but is most common as a primary immune-mediated disorder.

Immune-Mediated Uveitis (Recurrent Uveitis, Periodic Ophthalmia, Moon Blindness)

KEY POINT

Immune-mediated uveitis is the most common uveal disease of horses, and its recurrent nature can result in progressive loss of vision and ultimate blindness.

It is the most common cause of blindness. The inciting causes are not known but this is a multifactorial disease with *Onchocerca* and *Leptospira* spp. implicated. In most cases, the cause of the uveitis cannot be determined, but treatment of signs may be effective, at least in the short term.

HISTORY AND PRESENTING SIGNS

- Excessive lacrimation
- Cloudy eye
- Blepharospasm

CLINICAL FINDINGS AND DIAGNOSIS

- In the acute phase of uveitis, there are signs of intense ocular pain with excessive lacrimation and photophobia.
- A cloudy eye (corneal edema) is a typical finding, and there is usually ciliary and conjunctival injection. In some cases there also may be aqueous flare or even hypopyon, but the latter is unusual. Peripheral corneal vascularization is common.

KEY POINT

Miosis (constricted pupil) is a consistent clinical sign, and careful examination of the iris may show adhesions or synechiae, particularly when there have been several episodes of disease. Eyes with synechiae and uveitis dilate poorly or not at all in response to mydriatics such as 1% tropicamide or 1% atropine solution.

- *Anterior synechiae* are adhesions between the iris and cornea and are easily seen with a light source when viewed from the lateral aspect of the eye. *Posterior synechiae* are adhesions between the anterior lens and the iris and are suggested if the pupil is an abnormal shape on pupil dilation.
- A decrease in globe size is often the result of chronic uveitis. This may be detected in some cases by observation or by ultrasonography through the eyelids with a 7.5- to 10-MHz probe and comparing the axial length to the other eye.
- Tonometry of such eyes reveals low intraocular pressures.
- Cataracts occur in most long-standing cases.
- Fundic examination shows areas of depigmentation usually around the optic nerve in a classic "Butterfly wing" appearance. However, variations of this are possible.
- Laboratory tests are rarely diagnostic. However, blood profile, titers for leptospirosis, toxoplasmosis, and brucellosis may be indicated as are conjunctival biopsy for *Onchocerca* and fecal examination for parasites.

DIFFERENTIAL DIAGNOSIS

- Keratitis
- Conjunctivitis
- Septic uveitis
- Traumatic uveitis

TREATMENT

KEY POINT

The aims of therapy are to reduce the inflammation and reduce pain, which should be maintained until the clinical signs have abated.

- Topical corticosteroids, either prednisolone acetate 1% (Econopred ophthalmic solution, Alcon, Fort Worth, TX) or dexamethasone acetate drops or ointment should be used every 6 to 12 hours depending on the severity of the lesions, and provided the cornea does not retain fluorescein. Treatment should continue until clinical signs resolve.
- Topical NSAIDs including flurbiprofen 0.03% and suprofen 1% ophthalmic solution (Profenal, Alcon Surgical Inc., Fort Worth, TX) can be used.
- Recently an ocular immunomodulator, cyclosporine 2% (Optimmune ointment, Schering Plough, Kenilworth, NJ) has been marketed for veterinary ophthalmic use. Preliminary results are encouraging, but this drug may not fully replace other modes of treatment.
- Systemic NSAIDs are the agents of choice, but systemic glucocorticosteroidal antiinflammatory drugs may be used. Flunixin meglumine should be given at 1 mg/kg every 12 to 24 hours PO or IV, or phenylbutazone PO at a dose rate of 2.2 mg/kg every 12 hours for a period of 4 to 7

days. Aspirin also has been used at dose rates of 35 to 100 mg/kg every 24 hours PO for treatment of immune-mediated uveitis.
- Cycloplegics should be applied to produce pupillary dilatation. They are useful to treat the pain associated with ciliary spasm and may prevent synechiae formation. Atropine (1-2%) ophthalmic drops or ointment is effective but must be used at frequent (q4h) intervals until there is pupillary dilatation. Note that this frequency of atropine administration may result in signs of colic and therefore, care must be taken. After dilation has been established, the frequency of atropine administration can gradually be reduced. This may require 1 to 2 weeks, or even longer.

KEY POINT

Owners should be warned of the likelihood of recurrence, and anti-inflammatory therapy should be restarted as soon as clinical signs are apparent.

- Horses with a diagnosis of equine recurrent uveitis should be noted on a prepurchase examination, because blindness may result over the long term.

Miscellaneous Uveal and Anterior Segment Conditions

- *Iris cysts* are brown/black grape-like spheres that may be up to 0.5 cm diameter and may float free in the anterior chamber. (Do not confuse these with the corpora nigra found on the iris at the pupil margin.) Treatment is usually not required, but can include surgical removal or laser ablation.
- *Iris tumors* are unusual but do occur, especially iris melanomas in gray horses. These may metastasize or cause local destruction and may necessitate surgical intervention.
- *Hyphema.* The presence of blood in the anterior chamber of horses is usually secondary to trauma to the eye. Possible associated sequelae such as globe rupture, lens luxation and retinal detachments must be evaluated. Ultrasonic evaluation is useful in these cases, because the blood present often obscures any view of the posterior segment. Treatment involves the use of topical steroids if anterior uveitis is suspected secondary to a traumatic incident. Confining the horse with minimal excitement is also important. Systemic and topical NSAIDs should be avoided, because they may cause an exacerbation of the bleeding. If there is no ongoing hemorrhage, the blood will usually resorb by itself.

Glaucoma

Glaucoma, an optic nerve disease usually associated with elevations in intraocular pressure, is being increasingly diagnosed in horses due to the more common use of tonometry. Initial clinical signs are subtle, and it is often only late in the disease that a horse is presented for treatment. It is usually secondary to equine recurrent uveitis, so this condition must be treated in addition to the glaucoma. This disease behaves somewhat differently to glaucoma in other species.

HISTORY AND PRESENTING SIGNS

- Corneal edema and lines on the cornea
- Buphthalmia (swollen globe)
- Visual deficits

CLINICAL FINDINGS AND DIAGNOSIS

KEY POINT

Ocular pain rarely is demonstrated by horses with glaucoma.

- Dilated fixed pupil
- Lens displacement
- Depressed, dark optic nerve in the latter stages of the disease
- Blindness
- Elevated intraocular pressure

KEY POINT

Because of the strong extraocular muscles that may elevate intraocular pressure artificially, auriculopalpebral nerve blocks and topical anesthesia are indicated before measuring pressure. Also, no digital pressure should be placed on the globe when retracting the eyelids.

TREATMENT

- Therapy is directed at lowering intraocular pressure by either reducing the production of fluid or increasing outflow. Medical and surgical treatments are used. In our experience surgical treatment offers the best alternative.

Medical Treatment

- Miotics such as pilocarpine 1 to 4% every 8 to 12 hours (Pilocarpine HC1 ophthalmic solution, Schein Pharmaceutical Inc., NJ), which may cause irritation for the first few days, and demecarium bromide 0.25% every 12 hours (Humorsol, Merck & Co, Inc., West Point, PA) can be used. However, both miotics may exacerbate uveitis.

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- Topical beta-blockers such as timolol maleate 0.5% every 12 hours (Timoptic 0.5%, Merck & Co., Inc., West Point, NY) may be effective.
- Topical prednisolone acetate 1% or dexamethasone acetate every 6 to 12 hours can be used.
- Topical NSAIDs such as suprofen 1 % (Profenal, Alcon Surgical Inc., Fort Worth, TX) or flurbiprofen 0.03% every 6 to 12 hours, and systemic NSAIDs, flunixin meglumine at 1 mg/kg every 24 hours IV or PO may be helpful.

KEY POINT

No medical treatment of equine glaucoma has been uniformly effective.

- Surgical treatment involves destruction of the ciliary body, which produces the aqueous fluid of the eye. This is achieved either by cryotherapy or laser ablation.

Diseases of the Lens

Cataracts

Cataracts may be either congenital or acquired and focal or diffuse.

KEY POINT

Congenital cataracts are more common and more amenable to surgical treatment than acquired cataracts.

There is some suspicion that cataracts may be heritable, particularly in the quarter horse and Appaloosa. Cataracts may occur unilaterally or bilaterally.

HISTORY AND PRESENTING SIGNS

- Visual deficits
- Cloudy appearance of the lens
- "Spooking" in sunlight, possibly due to light reflections off the cataract within the eye

CLINICAL FINDINGS AND DIAGNOSIS

- When the cataract is diffuse, there is little problem in diagnosis, the opaque lens being readily apparent. However, when the cataract is focal, a more careful examination is necessary.
- If there is diffuse disease, there will be disturbance to vision with absence of a menace response in the affected eye. The pupillary light reflexes should be normal if there is a functional retina and the test is performed with a bright light in a dark room. Lack of a pupillary light response is a poor prognostic sign.
- Ophthalmoscopy can be undertaken to confirm the diagnosis. It is important to determine whether there are signs of uveitis and/or lens luxation, particularly in horses with acquired disease.

KEY POINT

Pupillary dilation is essential to assess the extent of the cataract and to allow fundic examination to evaluate signs of equine recurrent uveitis.

DIFFERENTIAL DIAGNOSIS

- Synechiae
- Lens luxation
- Uveitis
- Nuclear sclerosis (a normal aging change of the lens; see section on ophthalmic examination of the lens)

TREATMENT

- If the cataract is focal and the client unwilling to opt for surgery, vision may be improved by using atropine as needed to dilate the pupil, allowing vision "around" the cataract.
- Good results have been obtained with cataract surgery in foals. The surgery is best undertaken when the foal is a few months of age. The success rates in adults usually are lower due to the associated uveitis.
- In a blind animal, the goal of therapy is to prevent pain, which in these horses results from leakage of highly inflammatory lens proteins that in turn induce uveitis (See treatment of uveitis.)

Miscellaneous Problems of the Lens

- *Lens luxations* in the horse are not common but when they do occur are usually secondary to either glaucoma or equine recurrent uveitis. Spontaneous luxations are rare. Treatment involves correcting the underlying disease process and possible surgical removal, as long as it is expected that the horse will be able to see postoperatively. Complete luxations are often accompanied by significant intraocular disease, which will not enable vision even if the lens is removed. If a subluxated lens is stable, then surgical intervention may be avoided.

Diseases of the Retina and Choroid

Retinal Diseases

Early retinal disease may be easily overlooked unless an appropriate fundic examination (with pupil dilation in a dark room and suitable light source) is performed. It is usually not until significant vision-compromising damage occurs that horses with retinal disease are presented for examination. Early intervention may help some animals, reinforcing the need to perform fundic examinations as part of a routine physical examination.

HISTORY AND PRESENTING SIGNS

KEY POINT

Horses that are blind, especially foals, learn to adapt to familiar environs extremely well. It is only when placed in a new environment that the extent of blindness becomes apparent. A careful history is therefore essential.

- Blindness
- Problems with night vision
- Pupil dilation (observant clients may note this)

CLINICAL FINDINGS AND DIAGNOSIS

- Horses with retinal disease usually present with disturbances to vision and inspection of the anterior segment of the eye reveals no major abnormalities.
- Detailed ophthalmoscopic examination is required. This may reveal a pale optic nerve, depigmentation in the nontapetum, blood vessel thinning, and hyperreflectivity of the tapetum.
- Pupillary light reflexes are often poor, and the pupil may be dilated even in bright light.
- If an inflammatory etiology is suspected, a full physical examination and possibly an investigation for systemic disease is indicated.

DIFFERENTIAL DIAGNOSIS

- Retinal detachment—congenital (Standardbreds and Thoroughbreds) or acquired (secondary to uveitis)
- Equine recurrent uveitis
- Night blindness—Appaloosas, also with normal ophthalmoscopic findings
- Chorioretinitis—discrete lesions in the nontapetal zone with signs of visual deficit

TREATMENT

- If an inflammatory basis is suspected, then systemic anti-inflammatory drugs may be useful. Retinal disease that causes visual deficits generally carries a guarded prognosis.

Retinal Detachments

Most retinal detachments result from inflammatory or traumatic episodes. This is a common sequelae to trauma in foals, where the condition may be unilateral only.

HISTORY AND CLINICAL SIGNS

- Acute onset of blindness
- Pupillary dilation or pupils that respond slowly to light

CLINICAL FINDINGS AND DIAGNOSIS

- Associated anterior segment (in front of the lens) disease, such as hyphema, uveitis, and corneal abnormalities
- Lack of menace reflex
- Poor pupillary light reflexes
- Poor results when performing an obstacle test

KEY POINT

Blindfold each eye individually to perform the vision test.

- Fundic examination reveals a gray, translucent membrane extending from the optic disc into the vitreous.
- Ultrasonography, necessitated by an opaque anterior segment, may show a hyperechoic line on B scan ultrasonography (7.5-MHz transducer) extending from the optic disc.

TREATMENT

- At this time, anti-inflammatory medications are the only drugs that may be of benefit. It has been suggested that diuretics and carbonic anhydrase inhibitors may help, but in our experience this has not been the case.

Optic Nerve Disease

Optic nerve disease may be unilateral or bilateral. Unilateral optic nerve disease may be revealed as slight mydriasis, monocular visual impairment or blindness, occasional stumbling, and poor pupillary light reflexes in the affected eye. The consen-

sual pupillary light reflex may also be slower and less complete. If both optic nerves are affected, blindness may be evident.

HISTORY AND PRESENTING SIGNS

- Head trauma (as a result of rearing over backwards)
- Profound hemorrhage elsewhere in the body (such as complications with castration)
- Inflammatory episode, such as septicemia or bacteremia
- Guttural pouch disease with subsequent ligation of the internal/external carotid arteries and palatine artery
- Severe disturbances to vision
- Normal external appearance of the eye

CLINICAL FINDINGS AND DIAGNOSIS

- Ophthalmoscopy is essential to determine changes in appearance of the optic disc. In some cases there may be atrophy and decreased vascular supply, whereas in other cases of head trauma there will be edema of the optic disc and sometimes hemorrhage of the optic nerve head.
- Dilated pupils unresponsive to light are typically noted in horses with optic nerve disease.

DIFFERENTIAL DIAGNOSIS

- Hypoplasia of the optic nerve (present since birth)
- Optic neuritis
- Traumatic optic neuropathy
- Posthemorrhagic/ischemic optic neuropathy

TREATMENT

- There is no known treatment for any of the problems involving the optic nerve, apart from lesions resulting from head trauma. In these situations, high doses of systemic glucocorticosteroids, such as dexamethasone, are recommended for a few days after the traumatic event, then tapered off.

ACKNOWLEDGMENTS

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CHAPTER 12

Hemolymphatic System

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The hemolymphatic system is involved commonly in a secondary capacity in many disease states, with primary disease occurring rarely. However, collection of blood samples for evaluation of systemic disease is perhaps the most common ancillary diagnostic procedure undertaken by equine practitioners. Whether there is primary or secondary disease involving the hemolymphatic system, clinical signs are highly variable and depend on which of the numerous components of the system are affected. Presenting complaints may vary from exercise intolerance to weight loss and fever. A careful physical examination, concentrating on the mucous membranes, cardiovascular and alimentary systems, and peripheral lymph nodes, is important in suspected hemolymphatic disease.

EXAMINATION OF THE HEMOLYMPHATIC SYSTEM

History

The following questions may be pertinent when obtaining a history for a horse with suspected hemolymphatic disease:

- Has there been a change in attitude or demeanor and, if so, has it been sudden or insidious?
- Have there been recent changes in appetite?
- Are there signs of decreased exercise capacity?
- Has there been any change in urine color?
- Has there been evidence of bleeding from the nose?
- Have any swellings been noticed in any region of the horse's body?
- If swellings have appeared, have these been related to episodes of minor trauma?
- Has there been weight loss or signs of abdominal pain?
- Has the horse's breathing changed at all, either at rest or during or after exercise?
- Has the horse recently delivered a foal?

Physical Examination

A detailed physical examination of all body systems is indicated. In hemolymphatic disease, the nonspecific nature of clinical signs emphasizes the importance of a careful physical examination. Extensive use of clinicopathologic tests often is required.

KEY POINT

The most common presenting signs in horses with hemolymphatic disease are signs of depression, inappetence, fever, and weight loss.

The following areas should be given careful attention when performing a physical examination:

Mucous Membranes—In a range of hemolymphatic diseases, abnormalities in the mucous membranes may be a key clinical feature. Oral (including sublingual), conjunctival, nasal, or vulval mucosae and the inner side of the pinnae are usual sites for examination. Normal mucous membranes vary in color from pale to darker pink, but anemia can exist without excessive pallor being evident. Other important findings include jaundice due to accumulation of bilirubin in connective tissue, which may indicate hemolysis. However, mild jaundice commonly is observed in horses with inappetence. Also, the presence of petechial hemorrhages is particularly important because it may

indicate thrombocytopenia, severe platelet dysfunction, or vasculitis or, if it occurs in a horse with septicemia/toxemia, may herald the onset of disseminated intravascular coagulation (DIC). In addition, observation of muddy red-brown mucous membranes may suggest decreased functional capacity of erythrocytes from methemoglobinemia in response to wilted red maple leaf or onion toxicity.

The Circulation—Should be carefully assessed with particular attention paid to palpation of the jugular and other visible veins to assess signs of pain, thickening, and the possibility of thrombosis. Thrombosis may be a clinical manifestation of coagulation dysfunction. An elevation in heart rate may indicate compensation for decreased oxygen delivery (as a result of anemia), and various systemic disorders such as septicemia, alterations in fluid balance, and pain. A pansystolic heart murmur also may accompany tachycardia in severe anemia (packed cell volume [PCV] <0.15 L/L) in which there is reduced blood viscosity. The presence of ventral and/or limb edema that is nonpainful and pitting may indicate circulatory problems as a result of congestive heart failure, decrease in plasma albumin concentrations, or localized lymphatic obstructions. In contrast, the presence of hot painful inflammatory edema involving limbs, ventral abdomen, and head may be a sign of vasculitis or lymphangitis. Lesions of the skin in response to vasculitis often progress to fissures that exude serum, whereas edema in other body sites may cause dyspnea, colic, diarrhea, or lameness.

The Lymphatics—Normally, it is difficult to palpate lymph nodes in the horse, and those most frequently enlarged are located near the head. Lymph node enlargement can be localized or generalized and may be the result of reactive hyperplasia or immune stimulation, inflammation (e.g., *Streptococcus equi* abscessation), or primary tumors of the lymphoma complex. However, with diseases such as lymphoma, it is unusual to find enlargement of superficial lymph nodes. Generalized lymphadenopathy may result in lymphatic obstruction with peripheral edema, pleural effusion, or ascites. In particular, limb edema frequently is observed when lymphatic vessels are obstructed or infected. As space-occupying lesions, enlarged lymph nodes also may obstruct the pharynx, airways, or intestinal tract, resulting in signs such as dyspnea or abdominal pain, respectively.

Urinary System—Evaluation is important, particularly determination of the presence of hemoglobinuria as a consequence of intravascular hemolysis.

Rectal Examination—Is an important part of the work-up of many cases of suspected hemolymphatic disorders to determine the presence of internal abscesses or neoplasia. Further details on rectal examination are provided in Chapter 7.

DIAGNOSTIC AIDS

Collection of Venous Blood

Blood samples are most easily collected in evacuated tubes (e.g., Vacutainer, Becton Dickinson, Rutherford, NJ) containing various anticoagulants. For most routine hematology determinations (e.g., complete blood count), potassium EDTA is the anticoagulant of choice (purple-top Vacutainer tubes). For coagulation tests, sodium citrate tubes (blue-top Vacutainer tubes) are used. For plasma biochemical measurements, blood samples are usually taken in tubes containing lithium heparin as an anticoagulant (green-top Vacutainer tubes). For serum biochemical measurements (and for serum iron and serology for equine infectious anemia [EIA], equine viral arteritis [EVA], and piroplasmosis), samples are collected in tubes containing no anticoagulant (red-top Vacutainer tubes). Sample tubes should be filled to capacity; otherwise, the blood to anticoagulant ratio will be incorrect with the potential for changes in red cell size or clot formation. Also, collected blood should be immediately and thoroughly mixed with anticoagulant by gentle inversion. In general, citrate, fluoride-oxalate, and heparin anticoagulants are unsuitable for routine hematology.

Blood samples may be collected from any free-flowing vein such as the jugular, preferably with the horse in a relaxed state. After collection of the blood sample, evaluation of cytology should be performed within 1 to 2 hours. Longer periods, particularly if the blood is kept above room temperature, can result in changes in the morphology of cells, the most common being leukocyte vacuolation. Storage of the blood in ice or a refrigerator increases to ~24 hours the time available for accurate total leukocyte counts. Blood samples should not be frozen and should be transported chilled if possible.

KEY POINT

Ideally, if a blood sample is taken in the late afternoon and cannot be processed until the next morning, an air-dried blood smear should be made before refrigeration of the sample. However, blood smears should not be chilled.

For optimum results, thin blood smears must be prepared on clean glass slides with a steady and even spreading technique. If available, a Romanovsky stain (e.g., Diff-Quik) should be used before submission of smears to a laboratory for cytologic evaluation.

Interpretation of the Hemogram

The reference values for the hemogram of adult horses are presented in Table 12-1. Also, some details of the hemogram in foals are given in Chapter 9.

Of particular note is that normal hematologic values for the horse vary depending on breed, age, and training status or level of activity. For example, "hot-blooded" horses (Arabians, Thoroughbreds, light horses) tend to have higher values for red blood cells (RBC), hemoglobin (Hb) content, PCV, and lower neutrophil:lymphocyte (N:L) ratios than "cold-blooded" horses (ponies and draught breeds). Also, RBC variables only reach the adult normal range at 2 years of age, whereas from 8 months through the first 1 to 2 years, N:L ratios are approximately 1:1. As well, handling, excitement, and exertion may elevate red cell indices because red cells are mobilized from the spleen under the influence of catecholamines. This effect may last for approximately 60 minutes depending on the level of stress. Catecholamine release also frequently results in a physiologic leukocytosis primarily due to increased numbers of lymphocytes and neutrophils. Thus, care must be taken when interpreting results from a horse in which there has been apprehension during sample collection or in recently exercised horses. Such blood samples usually will have values at the high end of the reference range.

TABLE 12-1. Reference Range of Hematology Values in the Adult Horse

Hematology Value	Reference Range
Packed cell volume (PCV), L/L	0.30-0.48 (30%-48%)
Hemoglobin (Hb), g/L	110-160(11-16 g/dL)
Erythrocytes (RBC), $x10^{12}$/L	7.5-11.0(7-11 $x10^{6}$/LμL)
Mean corpuscular volume (MCV), fl	41-49
Mean corpuscular hemoglobin concentration (MCHC), g/L	300-360 (30-36 g/dL)
Mean cell hemoglobin (MCH), pg	13-16
Leukocytes (WBC), $x10^{9}$/L	6.0-11.0 (6000-1 l,000/μL)
Neutrophils, $x10^{9}$/L	2.5-7.0 (2500-7000/μL)
Band neutrophils, $x10^{9}$/L	0.0-0.24 (0-240/μL)
Lymphocytes, $x10^{9}$/L	1.6-5.4 (1600-5400/μL)
Neutrophiklymphocyte Ratio	1.5:1.0
Monocytes, $x10^{9}$/L	0.0-0.7 (0-700/μL)
Eosinophils, $x10^{9}$/L	0.0-0.5 (0-500/μL)
Basophils, $x10^{9}$/L	0-0.3 (0-300/μL)
Thrombocytes, $x10^{9}$/L	100-300 (100,000-300,000/μL)

Note: Standardbreds usually have lower mean red cell indices than Thoroughbreds, and horses used for endurance riding will tend to be at the low end of the normal range. Ponies and other "cold-blooded" horses (eg., draughthorses) also may have lower mean red cell indices often below the reference range given above.

Interpretation of the Erythrogram

Before discussion of changes in erythrocyte variables in disease (i.e., anemia), a number of features unique to equine erythrocytes should be emphasized.

- Evaluation of erythrocyte regeneration in the horse is difficult because equine erythrocytes are not released into the circulation until mature, even when there is intense erythropoiesis. Therefore, reticulocytosis, nucleated red cells, polychromasia, macrocytosis, and marked anisocytosis, common in the other domestic species, are not present in equine regenerative anemias.

KEY POINT

For this reason, erythrocyte indices rarely are useful in the diagnosis of the type of anemia in horses. Regenerative or nonregenerative anemias in the horse are best determined by examination of bone marrow aspirates, although performing serial measurement of PCV, Hb, RBC, and plasma total protein (TP) (e.g., once per day in severe anemias and once per week in mild anemias) also may be helpful to determine the erythropoietic response.

- A moderate increase in mean corpuscular volume (MCV; particularly in hemolytic anemia) and red cell distribution width (RDW; particularly in blood loss anemia) as measured by automated hematology analyzers have been reported to occur 2 to 3 weeks after the onset of anemia. As well, increased mean corpuscular hemoglo-

bin (MCH) values may be indicative of the presence of free hemoglobin and of hemolysis.
- The spleen is capable of inducing up to a 50% increase in the red cell count when stimulated to contract. This may accompany not only excitement and exercise but also disorders such as blood loss or endotoxemia.

KEY POINT

The response of the spleen to hemorrhage with elevation of the PCV means that estimation of the magnitude of blood loss using this variable is unreliable until at least 24 hours after hemorrhage has occurred.

- Howell-Jolly bodies may be found in a small percentage of normal horses and are not indicative of increased erythropoiesis as in other species.
- The small size and lack of central pallor of equine erythrocytes makes spherocytosis, indicative of immune-mediated hemolytic anemia, difficult to appreciate on equine blood films. Also, the tendency for rouleaux formation may be confused with autoagglutination often noted in immune-mediated hemolytic anemia.

Interpretation of the Leukogram

NEUTROPHILS

Neutrophilia is defined as the presence of more than 7 x 10^9 (7000/μL) neutrophils/L of blood. Mild to moderate but transient increases in neutrophil numbers (7-15 x 10^9/L or 7000-15,000/μL) are noted in horses after epinephrine-mediated shifts from the marginal pool to the circulating pool as a result of excitement, fear, and exercise. Also, neutrophilia of this magnitude may occur with stress-induced endogenous corticosteroid release or exogenous corticosteroid administration and in chronic inflammation. Corticosteroid-mediated responses in horses may occur after short-term intense or endurance exercise and during long-term inflammatory disease. Severe neutrophilia (>15 x 10^9/L or 15,000/μL) occurs usually as a result of noninfectious and infectious inflammation or leukemia. The most sensitive indicator of infectious inflammatory disease is the presence of increased numbers of circulating immature neutrophils (left shift). Generally, the magnitude of the left shift and the degree of toxic changes in neutrophils (vacuolization, toxic granulation, and Dohle bodies) indicate the severity of disease. In horses, toxic neutrophils are most often observed when there is gram-negative bacterial infection or gastrointestinal abnormalities (e.g., salmonellosis, colitis, intestinal strangulation or obstruction) resulting in endotoxemia.

KEY POINT

In general, the total leukocyte count usually correlates with the absolute neutrophil count. However, neutrophilia without an increase in the total leukocyte count always suggests the possibility of inflammation.

Neutropenia (<2.5 X 107L or 2500/μL) occurs when marked tissue demand for neutrophils and/or endotoxin-induced sequestration of neutrophils exceeds bone marrow production. Leukopenia and neutropenia are most often associated with the acute stages of gastrointestinal and septicemic disorders in horses (e.g., salmonellosis, strangulation) and may be accompanied by marked left shift. Neutropenias of more than 3 days duration may reflect a guarded prognosis. Neutropenia also may occur with acute viral diseases (e.g., EVA, EIA, equine herpes virus 1), ehrlichiosis, and with bone marrow neoplasia such as lymphosarcoma.

LYMPHOCYTES

Lymphocytosis (>5.4 x 10^9/L or 5400/μL) occurs most commonly in excited or recently exercised horses in which accelerated emptying of thoracic duct contents into blood may occur. Counts as high as 14 x 10^9/L (14,000/μL) have been reported. Diseases that can result in lymphocytosis include acute and long-standing bacterial or viral infections and rarely lymphoid leukemia.

Lymphopenia (<1.6 x 10^9/L or 1600/μL) frequently is associated with corticosteroid-induced responses with counts less than 1 x 10^9/L (1000/ML), suggestive of severe systemic stress. Other causes of lymphopenia include acute systemic infections, viral diseases, loss of lymph into body cavities, lymphosarcoma, and lymphoid hypoplasia.

NEUTROPHIL: LYMPHOCYTE RATIO

Many veterinarians use changes in the N:L ratio as an indication of disease in athletic horses. The normal N:L ratio is about 1.5:1, and if the ratio is inverted, this is taken as evidence for maladaptation to training. Changes in N:L ratio will mirror changes in plasma Cortisol, which undergoes diurnal variation, the N:L ratio being higher when Cortisol concentrations are elevated.

KEY POINT

Considerable care should be exercised in interpretation of the N:L ratio because

various physiologic processes may result in temporary changes in the ratios of the two major cell types in the peripheral circulation.

MONOCYTES

Monocytes are present in relatively low numbers in the peripheral blood and are the precursors of macrophages. *Monocytosis* may accompany acute and chronic diseases (e.g., tissue necrosis, suppuration, granulomatous disease, hemolysis, and rarely glucocorticoid-mediated disorders). Monocytosis usually occurs in association with neutrophilia.

EOSINOPHILS

Eosinophilia (>0.5 x 10^9/L or 500/μL) usually is indicative of significant internal parasite migration or allergic disease, but in horses a cause is not always found. Some fungal and neoplastic diseases of tissues rich in mast cells (e.g., skin, lung, gastrointestinal tract, and female genital tract) and eosinophilic leukemia sometimes may result in eosinophilia.

Eosinopenia (<0.1 X 10^9/L or 100/μL) is most often a result of increased circulating corticosteroid concentrations and of acute infectious or endotoxic disease. The appearance of eosinophils on sequential hemograms of horses with severe disease often is a good prognostic sign.

Bone Marrow Aspiration

Although uncommonly performed in practice, bone marrow aspiration may be useful in characterizing anemias (i.e., regenerative versus nonregenerative), evaluating iron stores, and explaining the presence of abnormal blood cells (e.g., leukemia) or pancytopenia. Also, unexplained hyperproteinemia or hypercalcemia may result from marrow neoplasia (e.g., multiple myeloma or lymphoma) and warrant consideration of marrow evaluation.

KEY POINT

Bone marrow evaluation should be considered if a hemolymphatic disorder is identified but cannot be diagnosed from information gathered by history, physical examination, and routine laboratory tests.

Bone marrow aspiration is relatively easy to perform and is associated with minimal risk of complications in horses. In the adult horse, the usual sites for collection are the cranial sternum, the wing of the ilium, and the dorsal third of the ribs. However, aspirates for cytology are most reliably collected from the sternum because sternal hemopoietic activity persists throughout life, the bones are not covered by a large muscle mass, and the marrow cavity is covered by a thin layer of bone only. In foals, the wing of the ilium is the preferred site.

KEY POINT

Although a good bone marrow aspiration is sufficient for most purposes, a core biopsy is essential to subsequently confirm suspected cases of generalized marrow suppression, hypocellularity, myelofibrosis, or necrosis. In general, collection of core biopsies is the method of choice when sampling from the rib or ilium.

Ideally, bone marrow aspiration and core biopsies are performed in standing horses placed in stocks and restrained with nose twitch and, if necessary, sedation. An area of skin on the ventral midline, between the front legs to just caudal to the points of both elbows, is clipped and appropriately disinfected. Local anesthetic (5-7 mL of 2% lidocaine; Treatment No. 67) is infused subcutaneously and close to the sternum at a site determined by palpation. With the operator wearing sterile surgical gloves, a small stab incision is made through the skin with a scalpel blade. A special bone marrow collection needle with stylet (Fig. 12-1) or a 16- to 18-gauge, 8.75-cm (3.5-inch) spinal needle should then be inserted through the skin incision to the sternal bone surface. While grasping the hub firmly with one hand and stabilizing the needle with the other, the needle is

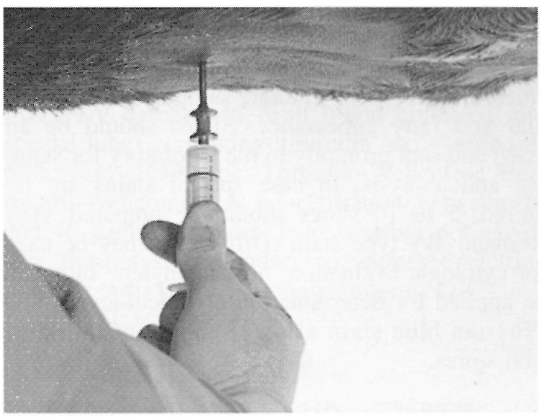


Figure 12-1. Bone marrow collection needle in position for collection of a bone marrow aspirate from the sternum.

slowly rotated and pushed in an upward and medially angled direction until firmly seated about 1 to 1.5 cm within the bone. The stylet then is removed and a 10- to 20-mL syringe containing 1 mL of 2 to 3% liquid tripotassium EDTA (anticoagulant) is attached to the aspiration needle (see Fig. 12-1). While stabilizing the needle in the bone, rapid and repeated suction on the syringe should be applied to obtain a sample of stromal marrow particles with as little blood contamination as possible. As soon as blood becomes evident in the syringe, the needle and syringe together should be removed from the bone and the contents of needle and syringe immediately transferred to a watch glass or Petri dish containing 10 to 15 mL of 2 to 3% tripotassium EDTA to prevent coagulation. Alternatively, smears can be made directly onto clean glass slides. Occasionally, the needle may require greater depth or redirection in the bone if initial attempts at aspiration are unsuccessful. To ensure an adequate sample is obtained, marrow spicules should be visualized in the watch glass.

KEY POINT

Blood contamination of the sample is difficult to avoid; however, even if contamination does occur, the marrow sample can still be useful for interpretation. Aspirates deposited into a watch glass containing anticoagulant allow marrow spicules to be separated from excess blood and easier processing of samples.

To preserve cell morphology, smears must be thin and prepared rapidly. Using a Pasteur pipette, marrow spicules are removed individually from the watch glass and placed on clean prelabeled glass slides. Excess fluid is sucked away using the pipette. A squash preparation is made by placing a second slide directly over the top of the spicules and, with application of light pressure, the two slides are slowly and evenly pulled apart. Alternatively, a technique similar to one used for a blood smear may be used. Suitable samples have a granular and fatty appearance. Slides should be air dried and sent promptly to the laboratory for staining and analysis. In case special stains are required, 5 to 10 slides should be prepared. Any Romanovsky type stain (Diff-Quik) may be used for cytologic evaluation, new methylene blue can be applied for determination of reticulocytes, and Prussian blue stain allows semiquantitation of iron stores.

KEY POINT

Although clinicians can perform bone marrow aspiration in the field and can prepare good air-dried smears without difficulty, accurate histologic interpretation of aspirates generally requires the experience of a specialist veterinary pathologist.

Apart from determination of cellularity, megakaryocyte numbers, and abnormal cell types, the myeloid:erythroid ratio (M:E) is determined by counting 500 cells. Also, counting the number of reticulocytes per 1000 erythrocytes on a new methylene blue-stained slide and/or counting the number of polychromatic macrocytes after Romanovsky staining may provide a more accurate assessment of erythropoietic regeneration. The normal M:E ratio is in the range 0.5 to 1.5. An M:E ratio <0.5, a reticulocyte count >50/1000 erythrocytes, and the presence of >5 polychromatic macrocytes suggest an adequate regenerative response in hemolytic or blood loss anemias. In contrast, the M:E ratio tends to increase in nonregenerative anemias. The M:E ratio and hence bone marrow response to disease must be interpreted in conjunction with results of a complete blood count. For example, a decreased M:E ratio in the presence of a decreased PCV and a normal leukogram suggests erythroid hyperplasia, whereas a decreased M:E ratio in the presence of neutropenia and a normal PCV suggests granulocytic hypoplasia.

Lymph Node Biopsy

When there is lymphadenopathy, lymph node biopsy may be indicated. Because a representative sample is essential for histopathologic examination, the best technique is surgical removal of an affected node. If this is not possible, use of a Tru-Cut biopsy needle (Travenol Laboratories Incorporated, Deerfield, IL) may provide an adequate sample for diagnosis. Fine-needle aspiration from enlarged lymph nodes is not recommended. Although this can provide a quick, easy, and inexpensive way to evaluate peripheral lymphadenopathy, misinterpretation of results is common. Because lymph nodes typically exfoliate cells easily, this frequently results in highly cellular aspirates. The exception is abscessation of lymph nodes where fine-needle aspiration can be effective and useful in obtaining samples for bacteriology. Smears of aspirates or impression smears of biopsies should be prepared on dry, clean, glass slides, air dried, and then sent to the laboratory for staining and interpretation.

Ultrasonography

Ultrasonography is a useful technique for evaluation of various vascular diseases including throm-

boembolism of the jugular veins and to assess the terminal aorta/iliac arteries. Also, ultrasonography may be useful for investigating flow in a vessel and for determining the presence of a bloody thoracic effusion or presence of cardiac abnormalities (refer to Chapters 3 and 6). A 5- or 7.5-MHz linear array transducer is most suitable for investigations of vascular structures.

Evaluation of Immunoglobulins

Quantitation of immunoglobulin concentration is an indirect way of assessing the humoral immune system and B-cell function in the horse. The total serum globulin concentration can be determined by subtracting albumin concentration from the total serum protein concentration. However, both globulins and albumin can be more accurately quantified by serum protein electropheresis. Electropheresis allows for separation and quantitation of albumin, α-globulins (antitrypsin, lipoproteins, and acute phase proteins), β-globulins (transferrin, coagulation proteins [complement, plasminogen], and fibrinogen), and γ-globulins (immunoglobulins; e.g., IgG, IgM, IgA, and IgE). Assessment of each of these fractions may provide valuable information about total immunoglobulin concentration, acute and chronic inflammatory states, hypogammaglobulinemia, polyclonal gammopathy, or monoclonal gammopathy. Polyclonal gammopathy has been reported in horses in association with chronic inflammatory diseases, immune-mediated diseases, and some lymphoid neoplasms. Monoclonal gammopathies also have been documented in horses (e.g., multiple myeloma) and are the result of malignancy of a single plasma cell clone. Concentrations, particularly deficiencies of individual immunoglobulin classes (principally IgG, IgM, IgE, and IgA), can be evaluated specifically using immunoelectropheresis, single radial immunodiffusion, enzyme-linked immunosorbent assay (ELISA), and radioimmunoassay.

Evaluation of Hemostasis and Platelets

Abnormal hemostasis may involve alterations in coagulation or fibrinolysis or both. The minimum laboratory database readily available to the practitioner to evaluate hemostasis in horses includes a platelet count, plasma fibrinogen determination, prothrombin time (PT) (or one-stage prothrombin time), activated partial thromboplastin time (aPTT), and serum fibrin degradation product (FDP) determination. Also, bleeding time is a simple though imprecise test of hemostasis that may be used. For accurate results, blood should be collected into Vacutainers containing the appropriate anticoagulant with the needle inserted into the vein in one motion to avoid contamination by tissue fluids that may activate coagulation. Ideally, the first tube of blood collected should be discarded, thus ensuring that the sample for testing does not contain tissue fluids. Also, tubes should be allowed to fill until the vacuum has been expended, because the ratio of anticoagulant to blood is critical for accurate laboratory analysis.

KEY POINT

Because some clotting factors are labile in vitro, blood samples are best placed on ice or refrigerated and delivered to the laboratory for evaluation within 4 hours (preferably 2 hours) of collection. If delay is expected beyond this time, the plasma should be harvested from samples within 30 minutes and frozen.

Blood for platelet counts is collected into tubes containing EDTA (purple top). However, if excessively low platelet counts are obtained from either manual counts from smears or by electronic cell analyzers, repeat evaluation should be performed on blood collected into tubes containing sodium citrate. This is because, on occasion, EDTA causes platelet clumping and subsequent pseudothrombocytopenia. Blood samples for PT and aPTT should be collected into sodium citrate (blue top) tubes. PT measures the integrity of the extrinsic pathway of coagulation and detects deficiency of one or more of the specific coagulation factors II (prothrombin), V, VII, X, and fibrinogen. Disorders in which PT is prolonged beyond 13.5 seconds include vitamin K deficiency (e.g., warfarin and sweet clover toxicity), DIC, and advanced, usually terminal, liver failure. In contrast, aPTT assesses the intrinsic clotting pathway and detects deficiencies of the specific coagulation factors II, VIII, IX, X, XI, XII, and fibrinogen. An extended aPTT beyond 64 seconds indicates DIC or hemophilia. FDPs are determined from blood collected into special tubes containing thrombin and e-aminocaproic acid (FDP tube; Thrombo-Wellcotest, Burroughs-Wellcome). A concentration between 10 and 40 μg/mL suggests increased but not excessive fibrinolysis secondary to increased clotting whereas >40 (μg/mL strongly suggests DIC.

KEY POINT

Whenever assessing a coagulation profile, samples from a disease-free horse should be used to establish "normal" or control values for that particular assay.

Bleeding time is used to crudely evaluate platelet number and function and the function of blood vessels in the hemostatic pathway. A small skin puncture is made using a 19-gauge needle inserted to ~4 mm on a clipped area on the side of the neck. Timing is begun when blood is first observed with blood drops removed every 30 seconds using filter paper that should not touch the puncture site. Timing stops when blood no longer appears. Bleeding time will be prolonged beyond 5 minutes in thrombocytopenia or when platelet function defects and vitamin K deficiency are present.

WHOLE BLOOD TRANSFUSION

Situations necessitating blood transfusion often arise as life-threatening emergencies and may result from acute hemorrhage from trauma, surgery, and rupture of large internal arteries or from severe hemolysis.

When to Transfuse. Transfusion is warranted when acute blood loss or hemolysis results in persistent reduction of the PCV to 0.12 L/L (12%) or less or in chronic blood loss when PCV falls to 0.08 L/L (8%). A low but stable PCV (0.12-0.20 L/L or 12-20%) with evidence of regeneration on serial hemograms and bone marrow aspiratation normally does not necessitate transfusion.

KEY POINT

Blood transfusion is a temporary therapeutic procedure, because transfused erythrocytes are removed from the circulation by the mononuclear phagocyte system within 2 to 6 days.

Volume to Transfuse. In general, a transfusion volume of 15 mL/kg is recommended. This represents 6 to 8 L for an adult horse (400-525 kg). To ensure that circulatory volume overload does not occur, the volume of any given transfusion should not exceed 20% of blood volume (i.e., 20% of ~ 8% of body weight [kg], which equates to ~8 L in a 500-kg horse).

Donor Selection. In field situations where an immediate transfusion is needed to sustain life, blood may be collected from any available donor (preferably a gelding of similar breed) and administered without delay. Initial transfusions rarely are associated with adverse reactions because horses infrequently produce strongly reactive natural erythrocyte alloantibodies. Antibodies against transfused cells develop within 3 to 10 days in 50% of horses after a single transfusion. If a second transfusion is necessary, the risk of reaction still remains low because it usually is given within this time. Ideally, however, an equine blood donor should be in good body condition, with a normal PCV and plasma TP concentration, be seronegative for equine infectious anemia virus, has never received a blood or plasma transfusion, and if female, has never had a foal. If time allows, compatibility testing (major and minor cross-matching, Coombs' antiglobulin test, and hemolytic testing) may be performed to select the most appropriate blood donor.

KEY POINT

Identifying potential blood (and plasma) donors in the practice area well before need arises may be done by blood typing and screening for plasma alloantibodies, in particular anti-Aa and anti-Qa. Blood typing laboratories will assist in this process.

Collection. Blood is most easily collected into 3-L, high-quality, blood-grade, plastic bags (with or without a connecting 2-L plasma reservoir bag). The anticoagulants acid-citrate-dextrose (ACD) or citrate-phosphate-dextrose (CPD) are recommended. A volume of 15 mL of ACD or CPD is needed for every 100 mL of collected blood. A healthy adult horse can donate up to 25% of blood volume (20 mL/kg) every 4 to 5 weeks without adverse clinical or physiologic consequences. The donor is restrained in stocks or tied up and may be lightly sedated with xylazine (Treatment No. 109) if necessary. An area over the midjugular vein should be clipped, surgically prepared, and local anesthetic infused subcutaneously. A 10- to 12-gauge, 8.75-cm (3.5-inch) catheter (Angiocath, Becton Dickinson, San Jose, CA) or large-bore needle is introduced retrograde into the jugular vein and secured in place using adhesive glue or electrical or duct tape. The anticoagulant primed collection tubing arising from the 3-L collection bag is attached to the catheter, allowing flow of blood into the bag via gravity. Bags should be mixed gently during collection. Bags are sequentially filled and the tubing clamped or tied when collection is complete.

Administration. In general, whole blood should be administered soon after collection using a blood administration set with an in-line filter. However, whole blood can be stored refrigerated (4°C) for up to 3 weeks. Transfusion should begin slowly at a rate of 0.1 mL/kg/min (i.e., slow drip) over 5 to 10 minutes to determine if there is a transfusion reaction. This usually is manifested by

trembling, sweating, restlessness, tachycardia, and tachypnea. If such signs occur, the transfusion should be stopped and flunixin meglumine (1.1 mg/kg IV; Treatment No. 52) administered. If there is further deterioration, epinephrine (adrenalin chloride, Parke-Davis, Morris Plains, NJ) at a dose rate of 1 to 1.5 mL/100 kg of a 1:1000 solution, given IM or SQ and intravenous polyionic fluids, should be administered. If no adverse reaction to blood infusion is observed, the infusion rate can be increased to 20 mL/kg/h.

Plasma Production. Plasma is obtained either commercially or harvested from blood collected as described above and then allowed to sediment. Ideally, to avoid large numbers of erythrocytes with the potential to sensitize recipients, blood should sediment for 12 to 18 hours at room temperature or preferably at 4°C. Plasma is siphoned or forced into the reservoir collection bag attached to the blood bag. Up to 60% of the total volume of blood collected may be harvested as plasma and subsequently stored in a household freezer (-4 to -20°C). Plasma harvested from blood sedimentation can be stored frozen at —4°C for at least a year or at —20°C for up to 5 years, retaining all constituents except coagulation factors VIII and V Preservation of all clotting factors in plasma for 1 year is possible if plasma obtained by commercial plasmapheresis enterprises is frozen to at least —20°C within 6 hours of collection. Plasma should be thawed at temperatures less than 37°C to avoid significant protein denaturation and administered in a manner similar to that described for blood transfusions.

Hemolymphatic Diseases

Anemia

Anemia is a common clinical sign in horses and is characterized by decreased RBC count, decreased PCV, and, except in cases of hemolysis, decreased Hb below the lower end of the reference range. Methodical determination of the underlying disease process producing the anemia is essential if appropriate management is to be instituted.

KEY POINT

Because the circulating red cell mass in the horse is extremely labile due to the effects of breed, age, level of activity, and the unique role of the spleen, accurate assessment of anemia may be difficult.

The reference range for hematology in adult horses is relatively wide (see Table 12-1). Although a small drop in red cell mass could have a significant impact on the performance of a competitive racehorse, a single blood sample taken from such a horse is likely to show values within reference range. In athletic horses presented for poor performance, a single measurement of PCV, Hb, and RBC count with values at the lower end of the normal reference range may or may not indicate a significantly reduced red cell mass as the cause of the problem. Sequential blood samples are more likely to provide definitive information as the individual reference range is narrower than that for the breed or use of horse. The rapidity of development and extent of erythrocyte loss determine the clinical signs of anemia, which can be due to a wide range of diseases.

HISTORY AND PRESENTING SIGNS

- Obvious external blood loss (e.g., trauma, surgery, epistaxis, melena)
- Increased respiratory excursions
- Decreased exercise capacity
- Decreased appetite and weight loss
- Signs of depression and weakness
- Colic
- Ventral and/or limb edema
- Intermittent deworming history
- Ingestion of wild onion *(Allium canadense)* and wilted red maple leaves *(Acer rubrum)*
- Recent administration of penicillin, dimethyl sulfoxide (DMSO), hypertonic solutions, or phenothiazine anthelmintics
- Geographic area in which horse ticks or horse flies are prevalent

CLINICAL FINDINGS

Acute Anemia

- Acute anemia generally is the result of blood loss or hemolytic processes, with signs dependent on the rate at which anemia develops.
- Signs include mucous membrane pallor, tachycardia, tachypnea, and a systolic heart murmur associated with decreased blood viscosity and increased blood flow turbulence. Horses that have ingested wilted red maple leaves generally have gray or brown mucous membranes because of methemoglobin accumulation in erythrocytes.
- Fever and mature neutrophilia often accompany rapid reduction in red cell mass. Renal hypoxia or pigment-induced nephropathy (i.e., from hemoglobin) may cause acute renal failure with azotemia, serum electrolyte, and urinary biochemical abnormalities.
- Horses with acute severe loss of more than 30% of blood volume (24 mL/kg or 12 L of blood

for a 500-kg horse) may develop hypovolemic shock, characterized by a weak thready pulse, pallor of the mucous membranes, prolonged capillary refill time, marked tachycardia, and poor jugular filling.
- Acute external blood loss from epistaxis, hematuria, or trauma is usually obvious, whereas acute intrathoracic or intrabdominal hemorrhage may cause dyspnea or ileus and colic, respectively. Also, coagulopathy may be manifested as frank bleeding, hematoma formation, and petechial and/or ecchymotic hemorrhages.

Chronic Anemia

- Chronic anemia usually is the result of long-term blood loss, low-grade hemolysis, and reduced erythropoiesis.
- Signs are more obscure because of physiologic adaptation by the horse. Pallor of the mucous membranes, exercise intolerance, and signs of depression are common. Although vital signs often are normal at rest, dramatic increases in heart and respiratory rates may be observed if the horse is subjected to stress.
- Icterus and hemoglobinuria may occur in association with chronic intravascular hemolysis, whereas anemia secondary to chronic inflammatory diseases and concomitant decreased erythrocyte production may be accompanied by anorexia, weight loss, fever, and dullness of haircoat.

DIFFERENTIAL DIAGNOSIS AND DIAGNOSIS

The approach to identifying the cause of anemia depends on history (e.g., external evidence of hemorrhage, exposure to hemolytic toxins or drugs), a thorough physical examination (in particular, attempts to identify evidence of chronic inflammatory disease), and the initial findings on hematologic and biochemical analyses. In general, decreased PCV and plasma TP may be interpreted to suggest *blood loss anemia.* A decreased PCV, normal TP, and marked increases in serum total bilirubin concentration with normal liver enzymes indicate *hemolytic anemia.* A decreased PCV, normal or increased TP, with or without increased globulin and fibrinogen concentrations and an inflammatory leukogram may be interpreted to suggest anemia due to *inadequate erythropoiesis.*

Blood Loss Anemia

- Blood loss anemia may either be acute or chronic and occur as a result of either external or internal hemorrhage. Common causes of external hemorrhage include complications from castration, accidental trauma, urogenital neoplasia, epistaxis associated with nasal trauma or tumors, guttural pouch mycosis, or severe exercise-induced pulmonary hemorrhage. Internal hemorrhage occurs most often as a result of severe soft tissue trauma, splenic rupture, spontaneous middle uterine artery rupture in postpartum mares, aortic rupture, pulmonary artery necrosis secondary to abscessation or neoplasia, endoparasitism, gastric ulcers, and gastric squamous cell carcinoma. Rarely, coagulopathy may be a cause of blood loss.
- In *acute blood loss* (up to 24 mL/kg of blood), the loss of red cells and plasma accompanied by the release of stored erythrocytes into circulation via catecholamine-induced splenic contraction results in little change in erythrocyte variables for 1 to 2 days. Within 6 hours of hemorrhage, significant translocation of fluids into the intravascular space leads to measurably decreased TP. However, anemia usually is not evident until at least 12 hours posthemorrhage with the true severity of blood loss known only after 36 to 48 hours, when erythrocyte variables reach lowest values. The PCV may fall to less than 0.20 L/L (<20%) and the TP may decrease to ~45 g/L (4.5 g/dL).

KEY POINT

The rate of erythropoietic recovery in the horse is slow, with an estimated increase in PCV of 0.0032 to 0.0042 L/L (0.32-0.42%) per day in response to severe acute hemorrhage.

- Depending on the severity, a regenerative response by the bone marrow is demonstrable from 3 to 42 days posthemorrhage, with complete restoration of red cell mass requiring between 1 and 3 months. However, replacement of plasma proteins occurs more rapidly with albumin taking 5-10 days and globulins requiring 3-4 weeks to return to pre-blood loss concentrations.
- Weekly measurement of PCV and TP may be of value to determine whether regeneration is occurring (i.e., increasing PCV and TP), if hemorrhage has stopped (i.e., stable PCV, increasing TP), or is ongoing (i.e., PCV and TP remain low).
- Auscultation, percussion, centesis of body cavities, and percutaneous ultrasonography are useful diagnostic aids to determine presence of thoracic, abdominal, or intramuscular hemorrhage.
- If blood loss is suspected in the absence of an obvious cause, a coagulation profile and platelet count may be indicated to rule out presence of coagulopathy.

- In *chronic blood loss*, physiologic adaptations tend to mask clinical signs until the PCV drops below 0.12 L/L (12%). Total protein concentration may be normal, because of liver compensation, or reduced.
- Examination of feces, urine, and the peritoneal cavity for detection of blood; a fecal egg count; gastroscopy to examine for the presence of severe gastric ulcers or neoplasia; and cytologic examination for hemosiderin-laden macrophages in body cavity effusions can be valuable in evaluating chronic blood loss anemia.
- Loss of iron in chronic external blood loss may rarely result in decreased iron stores, reflected by decreased serum and marrow iron and a microcytic hypochromic anemia. However, iron deficiency anemia is uncommon in horses because dietary iron generally is abundant.

Hemolytic Anemia

- Hemolytic anemia in horses may be acute or chronic and most often is the result of oxidative injury, immune-mediated processes, and infectious diseases.
- Characteristic clinicopathologic changes in both acute extravascular and intravascular hemolysis include marked bilirubinemia, bilirubinuria and normal TP, and liver enzyme activities. Jaundice appears approximately 12 hours after an initial hemolytic crisis and is most pronounced in the sclera. In contrast, acute severe intravascular hemolysis causes hemoglobinemia with discoloration of plasma and an increased MCH and hemoglobinuria if the renal threshold for hemoglobin is exceeded. Urinalysis is therefore an important diagnostic aid in hemolytic anemia.

Oxidant-Induced Hemolysis

- This can be caused by ingestion of wilted (not fresh) red maple leaves, wild onions, rape, kale, and phenothiazine anthelmintics. Severe oxidative damage to erythrocytes is characterized by methemoglobinemia, resulting in gray-brown mucous membranes, chocolate-brown colored blood, and signs of intravascular hemolysis.

KEY POINT

Identification of Heinz bodies near the cellular margin of erythrocytes stained with new methylene blue and detection of eccentrocytes (erythrocytes with a pale zone along one margin of the cell) are considered confirmatory of the diagnosis.

- Horses may die peracutely, within 12 to 18 hours, after ingestion of wilted red maple leaves.

Immune-Mediated Hemolytic Anemia (IMHA)

- Usually occurs secondarily when immune complexes attach to antigen coating the surface of erythrocytes, resulting in cell destruction. Reported sources of antigens that coat erythrocytes include bacteria (e.g., streptococci, *Clostridium perfringens)*, viruses (e.g., EIA), parasites (e.g., *Babesia equi)*, neoplasms (e.g., lymphosarcoma), and drugs (e.g., penicillin). Also, foals may develop an isoimmune hemolytic anemia (neonatal isoerythrolysis) when alloantibodies against the foals' red cells are present in the mares' colostrum (see Chapter 9).

KEY POINT

Blood films from blood diluted with saline (1:4) may show erythrocyte agglutination, spherocytosis, and anisocytosis, whereas a positive direct Coombs' test is strong evidence for IMHA. False-negative results are possible with a Coombs' test, in particular if there has been prior corticosteroid therapy.

- Horses with acute or chronic IMHA usually present with signs referable to a primary underlying disease.

Infectious Diseases

- *Equine infectious anemia* (EIA) is a viral (Lentivirus) disease with a worldwide distribution. Transmission is primarily by biting hematophagous insects (e.g., horseflies), although transplacental and iatrogenic spread between horses via repeated use of needles, dental floats, and other contaminated equipment has been reported.

KEY POINT

A radial immunodiffusion test (Coggin's test) is used for diagnosis of EIA, and serologically positive animals are infected for life.

- Horses with acute EIA show fever, signs of depression, thrombocytopenia, and anemia. Chronic carriers are clinically normal, although cyclic recrudescence of signs with transmission to other horses is possible.
- *Equine piroplasmosis* is a parasitic infection with the intraerythrocytic hemoprotozoan parasites *Babesia caballi* and *Babesia equi* and results in severe acute hemolytic anemia. Equine piroplasmosis has a worldwide distribution wherever the tick vector lives. The incubation period is 5 to 30 days.

KEY POINT

Diagnosis of piroplasmosis is by observation of the organism on blood smears stained with Giemsa or methylene blue and by use of serologic testing.

- Although horses can remain asymptomatically infected for long periods and suffer relapse of disease, infection with *B. equi* may result in death within 24 to 48 hours of onset of signs.
- *Equine ehrlichiosis* is caused by the rickettsial organism *Ehrlichia equi.* The exact mode of transmission is unknown, although ticks are implicated and disease most commonly is noted during late autumn, winter, and spring. Incubation ranges from 3 to 10 days.

KEY POINT

Observation under oil immersion of granular inclusion bodies in the cytoplasm of neutrophils and occasionally eosinophils in blood smears stained with Giemsa correlates closely with the onset of clinical signs of ehrlichiosis. Inclusion bodies remain visible for up to 10 days.

- Horses over 3 years appear to be most severely affected, but mortality is low and animals are immune to reinfection for at least 2 years.

Miscellaneous Causes

- Miscellaneous causes of hemolytic anemia include microangiopathic hemolytic anemia, heavy metal toxicosis (e.g., chronic consumption of lead, arsenic, copper, selenium), DMSO and lecithinases from bacteria (e.g., *Staphylococcus aureus* and *Leptospira interrogans)* and snake venom.
- Microangiopathic anemia commonly occurs secondary to some other disease process in which erythrocytes are damaged while passing through abnormal vasculature or as a result of turbulent blood flow. Examples are DIC, acute fulminant hepatic failure, hemolytic uremic syndrome, vasculitis, hemangiosarcoma, or arteriovenous shunts.

Inadequate Erythropoiesis

- Inadequate bone marrow production of erythrocytes usually occurs secondary to some other chronic condition and results in a slowly developing nonregenerative anemia with nonspecific clinical signs.

Anemia of Chronic Disease

KEY POINT

Anemia of chronic disease associated with infectious, inflammatory, or neoplastic disorders is the most common form of anemia in horses.

- Chronic disease processes result in shortened erythrocyte lifespan (<150 days), bone marrow suppression, and sequestration into and decreased release of iron from the reticuloendothelial system.
- The anemia generally is mild (PCV of 0.20-0.30 L/L or 20-30%), TP is normal or increased, and often there is leukocytosis and elevated fibrinogen concentrations.
- A careful and thorough clinical and laboratory examination may indicate where the chronic disorder is localized. Apart from hematology and fibrinogen determination, serum biochemical analyses to evaluate organ function, serum protein electropheresis, and investigation of the thoracic and abdominal cavities may be warranted.
- Bone marrow examination may show a nonregenerative response. Also, other causes of inadequate erythropoiesis specifically involving the bone marrow (e.g., hypoplasia, aplasia, or neoplastic infiltration) can be determined.
- *Aplastic anemia* results from congenital or acquired failure of marrow stem cells to differentiate. Most reported cases are considered idiopathic, although phenylbutazone has been implicated as a rare cause in horses. *Myelophthisic anemia* occurs when normal marrow is invaded by neoplastic tissue, resulting in a decrease in all marrow-derived elements. Pancytopenia is characteristic of both conditions. Because the lifespan of platelets and leukocytes is shorter than that of erythrocytes, clinical signs of thrombocytopenic hemorrhage, infection, and fever typically precede those of anemia.

TREATMENT

Blood Loss Anemia

Acute Blood Loss

- The source of the hemorrhage should be controlled and hypovolemia corrected. Direct pressure or ligation of large vessels assists in control of external bleeding. Little can be done to directly control internal hemorrhage apart from reducing physical and environmental stress.
- Hemorrhagic shock must be promptly treated with isotonic electrolyte solutions (e.g., lactated Ringer's solution), administered intravenously at an initial rate of up to 80 mL/kg/h (or one blood volume per hour, i.e., 8% of body weight [kg] or 40 L for a 500-kg horse). To achieve this flow rate, large-bore catheters (10-12 gauge) are needed in both jugular veins. However, 20 mL/

kg/h (i.e., 10 L/h for a 500-kg horse) is probably more practically achievable and, even in severe blood loss, will result in improvement in the horse's condition. If blood loss (external) can be roughly estimated, a guide for replacement is three times the estimated loss with the crystalloid isotonic fluid.

- Careful monitoring of vital signs and TP and appropriate adjustment of fluid rate are necessary to prevent dilution of the blood and exacerbation of anemia and hypoproteinemia. Reduction of TP below 45 g/L (4.5 g/dL) may allow fluid to move out of the circulation and into interstitial spaces, and therefore crystalloid fluids should be discontinued at this stage.
- Small volumes (2-4 mL/kg or 1-2 L) of hypertonic saline (7% NaCl) may be useful to rapidly improve blood volume and cardiovascular function, particularly if high-volume crystalloid solution replacement is not practical. However, because some evidence indicates that use of hypertonic saline in horses with uncontrolled hemorrhage increases blood loss and mortality, these solutions should be used with caution and preferably when bleeding has been controlled.

KEY POINT

Blood transfusion is recommended if the PCV decreases to less than 0.12 L/L (12%) over 24 to 48 hours, if there is uncontrolled bleeding, or if there is poor clinical response to crystalloid therapy. The latter is manifested as persistent tachycardia, tachypnea, prolonged capillary refill time, mucous membrane pallor, and weak pulse pressure.

- Although anecdotal, there are reports that antifibrinolytic agents such as e-aminocaproic acid (Amicar, Wyeth-Ayerst, Pearl River, NY; 5 g IV once), tranexamic acid (Cyklokapron, Pharmacia, Mississauga, Ontario; 1 g IV once), naloxone hydrochloride (Narcan, DuPont Pharmaceuticals, Manati, Puerto Rico; 8 mg IV once), and 10% buffered formalin (10-30 mL added to 500 mL of 0.9% NaCl administered rapidly IV) can be beneficial and appear to be without adverse effects to treat postpartum uterine artery hemorrhage.
- Once the acute problems are resolved, with stabilization of the circulation and reestablishment of oxygen delivery, it is necessary to monitor for signs of renal and hepatic hypoxia. A diet containing adequate iron (in cases of external blood loss; see Chapter 18) so that normal erythropoiesis is possible is indicated. However, iron supplementation rarely is required, because most diets are rich in iron.

Chronic Blood Loss

- Management requires identification and treatment of the source of the blood loss. For example, appropriate anthelmintics should be used for gastrointestinal parasitism, whereas gastric ulceration can be managed by discontinuing use of ulceragenic medications (e.g., nonsteroidal anti-inflammatories) and administering treatment for ulcers (see Chapter 7).
- If true iron deficiency is diagnosed as a consequence of chronic blood loss, oral ferrous sulfate (2 mg/kg q24h) can be given, although normal diets contain iron in excess of normal nutritional recommendations.

KEY POINT

Despite the widespread use of oral and intravenous iron supplements by many horse trainers, parenteral administration is not recommended because iron deficiency anemia is extremely rare and there is the possibility of serious adverse reactions.

Hemolytic Anemia

- Regardless of the cause of hemolytic anemia, in severe acute cases (PCV <0.12 L/L), whole blood transfusion, balanced IV fluid therapy, close monitoring for renal failure induced by hemoglobin or hypoxia, supportive therapy, and avoidance of stress may be warranted as described for blood loss anemia. However, administration of new erythrocytes via transfusion, to horses with IMHA, may exacerbate the hemolytic process.

Noninfectious

- Elimination of the source of toxin from the diet, reduction of further toxin absorption using activated charcoal via nasogastric tube, and general supportive care are the primary goals of treatment of *oxidant-induced anemia.*
- Vitamin C (30 mg/kg ql2h in IV fluids) and dexamethasone (0.05-0.2 mg/kg IV, Treatment No. 30) may be beneficial in stabilizing cellular membranes. Methylene blue therapy is mostly ineffective in horses and even may exacerbate hemolysis.

KEY POINT

Horses recovering from acute oxidant injury may develop laminitis.

- Specific therapy for *IMHA* includes identification and elimination of the antigenic source. Inhibition of the immune response with dexamethasone at dose rates of 0.05 to 0.2 mg/kg

q24h IV or IM for 1 to 3 days (Treatment No. 30) is indicated, maintaining with oral prednisolone (Treatment No. 93) at dose rates varying from 0.4 to 2 mg/kg/day. The aim is to reduce the dosage to around 0.4 to 0.6 mg/kg every other day.

Infectious

- There is no specific means of eliminating *EIA* virus, and no vaccine currently is available. Options for management include palliative therapy, with permanent quarantine and EIA identification branding, donating the animal to an approved research facility, or euthanasia.
- In cases of *equine piroplasmosis, B. caballi* infection can be effectively eliminated using imidocarb (Burroughs Wellcome, Research Triangle Park, NC). The dose rate is 2 mg/kg q24h IM for two treatments. Side effects include salivation, colic, diarrhea, and injection site myositis. Donkeys may die if treated with imidocarb.
- A single dose of buparvaquone (Coopers Animal Health, Berkhamstead, UK) at 4 to 6 mg/kg IV or IM is effective at reducing clinical signs of *B. equi* but not at eliminating infection.
- Horses with *ehrlichiosis* usually recover with appropriate supportive care and administration of oxytetracycline at 7 mg/kg IV once daily for 7 days.

Reduced Erythropoiesis

- Treatment for *anemia of chronic disease* and *aplastic anemia* is directed at therapy for the primary disorder.
- Horses with anemia of chronic disease have normal iron stores, with the iron sequestered in macrophages, and do not require iron supplementation.
- Corticosteroids and anabolic steroids may enhance erythropoiesis in aplastic anemia, although the prognosis for both aplastic and myelophthisic anemia generally is grave.

Disseminated Intravascular Coagulation

DIC is a complex systemic syndrome characterized by concurrent hypercoagulation and hyperactivity of the fibrinolytic system (anticoagulation). Diffuse microvascular thrombosis leads to ischemic organ failure and consumption of platelets and clotting factors with the subsequent development of widespread hemorrhages.

KEY POINT

DIC always is secondary to any severe systemic disease. The clinical manifestations may range from a thrombotic crisis to a hemorrhagic diathesis. However, because of the dynamic nature of DIC in the horse, coagulopathy usually occurs in an intermittent or compensated form and rarely is associated with overt hemorrhage.

Endotoxemia related to bacterial sepsis and ischemic or inflammatory gastrointestinal diseases are the most common underlying causes of DIC in horses. The major predisposing factors include intestinal strangulating obstruction, thromboembolic infarction, and severe colitis. These induce mucosal disruption, allowing endotoxins to gain access to the peripheral circulation. Endotoxins promote DIC by directly activating the extrinsic clotting pathway or by damage to the vascular endothelium with activation of intrinsic coagulation. DIC also has been reported in association with disseminated neoplasia, protein-losing enteropathy, renal and hepatic failure, viremia, hemolytic anemia, and snake envenomation.

HISTORY AND PRESENTING SIGNS

- There are few specific presenting signs of DIC.
- There is greater risk of DIC secondary to any severe inflammatory or gastrointestinal disorder in which there are signs of endotoxemia (e.g., discoloration of mucous membranes, pyrexia, signs of depression, and dehydration), renal compromise (e.g., oliguria or anuria), gastrointestinal bleeding, or lameness associated with laminitis.
- A greater tendency for venous thrombosis occurs, in particular of the jugular vein, soon after venipuncture or insertion of catheters.

CLINICAL FINDINGS AND DIAGNOSIS

- The primary disorder is usually the main presenting feature early to midway through the course of DIC. Microvascular thrombosis may occur early in the disease, and this can result in ischemia to vital organs such as the kidneys, gastrointestinal tract, and the digit. Clinical signs include oliguric renal failure, ileus, colic, occult fecal blood loss, and laminitis.

KEY POINT

Although contributing to the morbidity and mortality of the primary disease process, the signs of microvascular thrombosis are nonspecific. Consequently, DIC usually is not recognized until late in its course when hemorrhage may become a prominent feature.

- Mucosal petechial or ecchymotic hemorrhages and a tendency to bleed after venipuncture or after minor trauma occur when depletion of clotting factors and platelets intensifies and generation of fibrinolytic byproducts (FDPs) exceeds mononuclear phagocyte system (MPS) clearance. However, uncontrolled bleeding, such as epistaxis, hyphema, and melena, is uncommon in horses with DIC.
- Occasionally, horses develop a chronic, intermittent, and compensated form of DIC with no apparent clinical signs. Localized gram-negative infections, neoplasia, and immune-mediated disorders (e.g., vasculitis and anemia) are common initiating diseases. However, compensation may become imbalanced by stress or worsening of the primary disease, resulting in overt DIC.

KEY POINT

The most important step in recognizing DIC in the horse is to be constantly aware of the particular clinical situations that may predispose to it.

- When DIC is suspected and diagnosis imperative, repeated hemostatic testing may be indicated because no one test consistently and specifically provides a definitive diagnosis. Serial analyses revealing thrombocytopenia (<50 x 10^9/L or 50,000/ML), with trends toward mild to moderate prolongation of PT and/or aPTT, strongly suggest DIC. Reduction (<60%) in antithrombin III (AT-III) concentrations and elevations in FDPs in the late stages of DIC further support the diagnosis. However, these tests generally are relatively complex and are not available in many laboratories. Hypofibrinogenemia is rare in horses with DIC.

DIFFERENTIAL DIAGNOSIS

- Septicemia
- Endotoxemia and shock
- Immune-mediated thrombocytopenia
- Vitamin K deficiency from warfarin or mouldy sweet clover toxicosis
- Inherited coagulation abnormalities
- Protein-losing nephropathy or enteropathy
- Severe hepatic dysfunction
- Vasculitis

TREATMENT

- Early, aggressive treatment of the primary disorder is essential. Septic conditions should be treated with appropriate antimicrobial agents. If there is an abdominal crisis, ischemic intestine should be surgically resected and the deleterious effects of endotoxemia treated.
- Large volumes of balanced intravenous fluids are crucial to assist in correction of shock and reduced tissue perfusion.
- Nonsteroidal anti-inflammatory drugs, particularly flunixin meglumine (Treatment No. 52), are useful to assist in combating the effects of endotoxemia (see Chapter 18). Aspirin (Treatment No. 9) has antithrombotic effects and may be given intravenously at a dose rate of 20 to 40 mg/kg every other day.
- Glucocorticosteroids are contraindicated because they enhance the vasoconstrictive effects of circulating catecholamines and reduce the phagocytic function of the MPS.
- Although restricted by cost and availability, administration of fresh plasma (15-30 mL/kg) may be of value to increase the AT-III concentrations and to replace consumed or lost clotting factors. However, once DIC has progressed to the stage at which hemorrhage predominates, the prognosis is grave.
- Although controversial and in general not recommended in fulminant DIC, there is some evidence that administration of heparin (80-120 IU q4h for 12 h followed by 20-40 IU/kg subcutaneously q12h for 3 days) may be of value in severe gastrointestinal diseases that have a high risk of coagulopathy or in suspected cases of chronic DIC. When possible, heparin should be administered in combination with fresh plasma to ensure adequate circulating AT-III activity and discontinued if the PCV undergoes a sudden decrease.
- Other treatments with reported clinical benefit include DMSO (100 mg/kg IV slowly q8h as a 10% solution) as a free radical scavenger, acepromazine (0.006-0.01 mg/kg IM or SQ q8-12 h) as a peripheral vasodilator to improve digital and renal perfusion, and commercial antisera enriched with antibodies directed against gram-negative bacteria to directly neutralize circulating endotoxin.

Hemophilia A

Hemophilia A in horses is due to an inherited disorder of the intrinsic coagulation pathway in which there is a deficiency of factor VIII coagulant activity. It is a rare condition and is the result of a recessive gene, described in Thoroughbred, Standardbred, Quarter horse, and Arabian colts.

KEY POINT

Hemophilia A is an X-linked chromosomal abnormality, and clinical signs are found only

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in males usually within the first few weeks of life.

HISTORY AND PRESENTING SIGNS

- Young horses (<6 months of age, although there is one reported case in a 3-year-old Thoroughbred gelding)
- Presented after trauma or after injections, for intramuscular or subcutaneous hematomas
- Recurring shifting lameness with or without multiple joint swelling (due to hemarthrosis)
- Signs of depression, abdominal pain, and dyspnea (due to hemorrhage into body cavities)

CLINICAL FINDINGS AND DIAGNOSIS

- Hemophilia A may result in spontaneous bleeding episodes, the severity of which depend on the degree of factor VIII deficiency in each affected individual. Minor trauma or surgical procedures increase the propensity for bleeding. Hemarthroses and intramuscular and subcutaneous hematomas most commonly are found. Epistaxis, hemoptysis, and hemorrhage into body cavities resulting in sudden death are observed rarely.
- Routine clotting profiles and platelet count show normal values apart from an increased aPTT. A specific assay for factor VIII coagulant activity on blood collected into citrate tubes should be performed to confirm suspected hemophilia A.

DIFFERENTIAL DIAGNOSIS

- Trauma
- Abscesses
- DIC
- Thrombocytopenia
- von Willebrand's disease
- Prekallikrein deficiency

TREATMENT

- Palliative treatment involving the administration of fresh blood or fresh or fresh frozen plasma is possible in the short term to control bleeding episodes. Unfortunately, this involves large volumes of plasma (up 10 L initially for an adult horse followed by 5 L every 12-24 h for 3-5 days) and is seldom practical outside a university teaching hospital. Also, because of the recurrent and progressively debilitating nature of the disease, most animals die or are euthanized in the first year of life.
- Because it is an inherited disorder in which the mare is a carrier, it is important to ensure that the mare does not continue breeding, because male offspring are likely to be affected and female offspring will act as carriers.

Leukemia

Leukemia occurs rarely in horses and involves unregulated proliferation of one or more hemopoietic cell types in the bone marrow. For example, *myeloproliferative leukemia* involves granulocytes, monocytes, erythrocytes, megakaryocytes, and mast cells, whereas *lymphoproliferative leukemia* involves lymphocytes and plasma cells. Acute and chronic forms occur, although the more aggressive acute leukemias of the granulocytic cell series are reported most commonly. Affected animals are reported to range in age from 10 months to >16 years, with no breed or sex predilection.

HISTORY AND PRESENTING SIGNS

- Acute or chronic course of disease
- Pitting hindlimb and ventral edema
- Inappetence and weight loss
- Signs of depression
- Enlarged lymph nodes
- Infrequently presented for signs of respiratory tract infection or exercise intolerance

CLINICAL FINDINGS AND DIAGNOSIS

- Clinical signs usually are nonspecific. However, the presence of hindlimb edema with or without ventral edema, mucosal petechial hemorrhages, and signs of depression are typical features in reported cases of leukemia. Less common signs are fever, infection (often respiratory), and colic.

KEY POINT

Results of routine hematologic examinations usually are what alerts the clinician to the diagnosis of leukemia. Anemia, thrombocytopenia, and abnormal cell types on blood smears are invariably found, whereas neutropenia and gammopathy are common.

- Definitive diagnosis is possible only by bone marrow aspirate or biopsy with further classification of the type of leukemia made on the basis of histochemical staining.

DIFFERENTIAL DIAGNOSIS

- Idiopathic or immune-mediated thrombocytopenia
- Vasculitis

- Granulomatous enteritis
- Pleuritis
- Peritonitis
- Internal abscessation
- Equine infectious anemia
- Other neoplasia (e.g., lymphosarcoma)

TREATMENT

- Although chemotherapy using cytosine arabinoside (10 mg/m^2 q12h for 3 weeks) has been attempted, there is no treatment that is economically or practically feasible for leukemia in horses.

Lymphangitis

Inflammation of lymphatic vessels and regional lymph nodes resulting in lymphatic stasis and obstruction may occur in association with infection, neoplasia, or other unknown factors. *Sporadic lymphangitis* is a relatively common lymphatic obstruction affecting the hindlimbs of horses. This disorder does not have an infectious etiology in most cases. *Ulcerative lymphangitis* is a bacterial or fungal infectious lymphatic obstruction often associated with wounds and is more frequent in conditions of poor hygiene. Identified causative organisms include *Corynebacterium pseudotuberculosis, Pseudomonas aeruginosa, Streptococcus* and *Staphylococcus* spp., *Sporothrix schenckii,* and *Pythium insidiosum.*

HISTORY AND PRESENTING SIGNS

- Poor hygienic conditions and presence of a wound
- Lameness
- Limb edema
- Multiple nodular limb swellings

CLINICAL FINDINGS AND DIAGNOSIS

- The earliest sign in *sporadic lymphangitis* is acute lameness, followed by whole limb edema often with serum oozing from taut skin surfaces. If persistent for more than 7 days, severe interstitial fibrosis may cause permanent swelling, disfigurement, and reduced function.
- In contrast, the infections associated with *ulcerative lymphangitis* may result in multiple foci of nodular abscessation along lymphatic tracts, which erupt to discharge serum/pus. Multiple limbs usually are affected and there is a tendency for exacerbation of clinical signs after innocuous cuts and abrasions.

KEY POINT

Samples of pus, fine-needle aspirates of tissue fluid, or biopsy for cytology, culture, and histopathology are essential for diagnosis. However, culture frequently is unrewarding.

DIFFERENTIAL DIAGNOSIS

- Vasculitis
- Trauma
- Cellulitis of the limbs due to staphylococcal infection
- Hypoalbuminemia
- Photosensitization
- Contact hypersensitivity
- Cutaneous glanders *(Pseudomonas mallei)* and epizootic lymphangitis *(Histoplasma farciminosus)* (limited to Africa and Asia)

TREATMENT

- Hydrotherapy, massage, and bandaging and forced hand walking may reduce soft tissue swelling and encourage vascular and lymphatic circulation.
- Administration of dexamethasone (0.04 mg/kg q24h IV reducing to 0.02 mg/kg q24h for up to 5 days; Treatment Nos. 29 and 30) may be beneficial in cases where infection is *not* a feature.
- In ulcerative lymphangitis, nonsteroidal anti-inflammatory agents such as phenylbutazone at 2.2 to 4.4 mg/kg q12h orally (Treatment No. 89) combined with prolonged antimicrobial therapy often is effective.
- Initial broad-spectrum combinations such as procaine penicillin (15-20 mg/kg or 15,000-20,000 IU/kg q12h IM; Treatment No. 84) and gentamicin (6.6 mg/kg q24h IV; Treatment No. 56) for 7 days, followed by oral trimethoprim/sulfa (15-30 mg/kg q12h PO; Treatment No. 108) for 3 weeks may be used.
- Erythromycin (10 mg/kg IV or 25 mg/kg PO; Treatment Nos. 39 and 40) has been reported to be more effective in confirmed cases of *C. pseudotuberculosis* infection.

Lymphosarcoma

A range of neoplastic disorders may involve the hematopoietic system, but the most common is lymphosarcoma (lymphoma). Most cases occur in middle-aged and older horses with no breed or sex predilection.

KEY POINT

Lymphosarcoma may exist in localized forms (e.g., cutaneous or alimentary) or may be

generalized. However, metastases with subsequent multiple organ system involvement are common.

HISTORY AND PRESENTING SIGNS

- Weight loss
- Colic
- Increased respiratory rate and effort
- Ventral edema
- Enlarged lymph nodes
- Signs of depression, exercise intolerance
- Inappetence or anorexia

CLINICAL FINDINGS AND DIAGNOSIS

- Clinical signs relate to the sites of tumor involvement and are therefore diverse and nonspecific, including features common to any chronic inflammatory or neoplastic disease. However, regardless of site, the most frequent signs include weight loss, inappetence, signs of depression, fever, and ventral edema.
- Clinically evident enlargement of peripheral lymph nodes (lymphadenopathy) is unusual in lymphosarcoma.

KEY POINT

Additional clinical signs reflect dysfunction of affected organs, and the course of the disease typically is rapid after the onset of clinical signs.

- Although most lymphosarcomas appear in lymph nodes or in other tissues rich in lymphoid cells such as the spleen, bone marrow, pharynx, and gastrointestinal tract, lymphosarcoma may occur in any tissue, including the skin. Four anatomic categories of the disease have been described. Combinations of these forms of lymphosarcoma occur in approximately 50% of cases.

  Generalized (Multicentric)—The most common form typically involving tissues such as lymph nodes (in particular mandibular, caudal cervical, retropharyngeal, mesenteric, and colonic), liver, spleen, intestine, and kidney. Clinical signs may include icterus, upper airway obstruction, and dyspnea.

  Alimentary—A common acute form of lymphosarcoma in horses resulting in rapid deterioration of clinical signs. Diffuse involvement of the small intestine and associated lymph nodes most frequently occurs. Apart from the nonspecific signs listed above, diarrhea and colic may occur.

  Mediastinal—Enlargement of mediastinal lymph nodes may produce cardiorespiratory signs (e.g., pleural effusion, tachypnea, dyspnea, and jugular distension).

  Cutaneous—Results in multiple, firm, nonpainful, subcutaneous nodules, commonly affecting the shoulder, perineum, and axillary regions. Tumors may develop rapidly or may be present for extended periods before the onset of systemic signs.

- Other extranodal sites include the eyes or adnexa and the central nervous system.

KEY POINT

A thorough, persistent, and systematic approach is vital for successful diagnosis. Establishing a database in these cases (see Chapter 2, Weight Loss, Edema) involves repeated physical and organ system examinations.

- If there are signs referable to the abdominal cavity, rectal examination should be performed to detect lymph node enlargement and splenomegaly. Abdominocentesis occasionally may provide diagnostic exfoliative cytology, whereas oral D-glucose and D-xylose absorption curves are frequently flattened, indicating malabsorption (see Chapter 7). Rectal biopsy in some cases may confirm diffuse alimentary involvement. Masses or intestinal infiltration also may be noted if an exploratory celiotomy is performed. Diagnosis is confirmed after excisional biopsy.
- Careful thoracic auscultation and percussion, thoracic ultrasonography, and thoracocentesis will aid in determining involvement of the thoracic cavity.
- Findings on clinicopathologic analysis frequently are nonspecific, although in up to 50% of cases there is anemia of chronic disease, neutrophilia, elevated globulin and fibrinogen concentrations, and hypoalbuminemia. Rarely, immune-mediated anemia with a positive Coombs' test, thrombocytopenia, lymphocytosis, and hypercalcemia are present.

KEY POINT

The observation of neoplastic lymphocytes in peripheral blood is rare and may be a late manifestation of the generalized form, indicating dissemination and bone marrow involvement.

- The presence of neoplastic lymphocytes in tissues, body cavity fluids, or bone marrow is the

main criteria for definitive diagnosis. If access is possible and there is enlargement of one or more superficial lymph nodes, fine-needle aspiration, biopsy, or total excision is indicated. Bone marrow biopsy is preferable to aspiration, because there may be difficulties when attempting to make a diagnosis.

DIFFERENTIAL DIAGNOSIS

- Nodular skin disease
- Granulomatous enteritis (inflammatory bowel disease)
- Pleuritis
- Peritonitis
- Internal abscessation
- Vasculitis
- EIA
- Other chronic inflammatory or neoplastic disease

TREATMENT

- Horses with the cutaneous form of lymphosarcoma without internal involvement and in which dexamethasone (0.02-0.2 mg/kg IV, IM or PO q24h) is administered have been reported to survive for several years. Spontaneous recovery of cutaneous lesions sometimes with intermittent recurrence also has been documented.
- There is no successful treatment for other forms of lymphosarcoma, although antineoplastic agents (various combinations of cytosine arabinoside, chlorambucil, cyclophosphamide, L-asparaginase, and prednisone) have been used, with about 50% of horses with multicentric lymphosarcoma going into remission for several months to a year.

Plasma Cell Myeloma

Plasma cell myelomas are rare in the horse. The most common form is multiple myeloma, characterized by malignant proliferation of plasma cells, primarily within the bone marrow. The cells produce excessive amounts of either a complete immunoglobulin or immunoglobulin fragment (paraprotein), which is detectable using serum and/or urine protein electropheresis.

HISTORY AND PRESENTING SIGNS

- No sex or age predilection but reported in hotblooded horses more frequently
- In appetence
- Weight loss
- Limb edema
- Lameness or ataxia

CLINICAL FINDINGS AND DIAGNOSIS

- Signs of disease are variable and are related to organ invasion by neoplastic cells or to systemic effects of the immunoglobulin produced. Consistent findings include weight loss, anorexia, fever, and limb edema.
- Pneumonia, hindleg paresis/ataxia, epistaxis, and palpable lymphadenopathy are less commonly reported.
- Lameness or neurologic deficits may occur secondary to bone pain, bony invasion by myeloma cells, or osteolysis due to an osteoclast-activating factor produced by neoplastic cells. Also, circulating paraproteins may contribute to renal disease and interfere with clotting factors and platelets, resulting in bleeding disorders.
- Anemia, hyperglobulinemia, and hypoalbuminemia are the most common abnormal laboratory findings.
- Leukopenia, thrombocytopenia, circulating plasma cells, azotemia, and hypercalcemia are found infrequently. A monoclonal gammopathy is detected in nearly all cases by serum protein electrophoresis with most paraproteins in the IgG class. Urinalysis also may reveal proteinuria, although confirmation of Bence-Jones protein is rare.
- Support for the diagnosis is by demonstration of typical "punched-out" radiolucencies on survey radiographs of long bones or cervical vertebrae. Also, plasma cell infiltration of enlarged lymph nodes is commonly observed in biopsy specimens or smears from fine-needle aspirates.

KEY POINT

Definitive diagnosis of multiple myeloma requires demonstration of at least 10% plasma cells in bone marrow aspirates or biopsy in addition to monoclonal gammopathy.

DIFFERENTIAL DIAGNOSIS

- Any chronic inflammatory process (e.g., granulomatous enteritis, pleuritis, pneumonia, peritonitis, and internal abscessation)
- EIA
- Vasculitis
- Other neoplasia

TREATMENT

- There is no reported successful treatment for plasma cell myeloma in horses. Most horses die

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or are euthanized within 4 months of developing clinical signs. However, two cases have been reported in which treatment with antineoplastic agents (melphalan, prednisone, and cyclophosphamide) resulted in longer survival times.

Thrombocytopenia

Platelet numbers less than 100 x 10^9/L (<100,000/μL) indicate thrombocytopenia. Most cases are associated with increased use or consumption of platelets that occurs most frequently with DIC and acute inflammation associated with infection or endotoxemia. Thrombocytopenia due to increased platelet destruction usually is immune mediated. Immune-mediated thrombocytopenia may be primary (idiopathic) or secondary to other diseases, including EIA, IMHA, lymphosarcoma, and certain drugs (e.g., phenylbutazone, aspirin, heparin, sulfonamides, and penicillin). Thrombocytopenia caused by decreased or ineffective production of platelets can occur with neoplastic diseases of the bone marrow (e.g., lymphosarcoma, plasma cell myeloma, or bone marrow aplasia), toxic insult to the marrow (e.g., phenylbutazone and estrogens), and immune-mediated damage to platelet-producing precursors. In addition, mild transient decreases in platelet numbers may occur as a result of splenic sequestration, severe vasculitis, or severe acute external hemorrhage.

KEY POINT

Because blood collected into EDTA sometimes can produce platelet aggregation, any low platelet counts should be repeated after collection of a second blood sample, preferably using citrate (blue-top Vacutainer) as the anticoagulant.

HISTORY AND PRESENTING SIGNS

- Signs of primary systemic disease
- Hematoma formation after minor trauma or venipuncture
- Few signs of disease in idiopathic cases

CLINICAL FINDINGS AND DIAGNOSIS

- Affected horses suffering idiopathic thrombocytopenia are generally bright and alert.

KEY POINT

Clinical signs generally are confined to those caused by the primary disease process, but if platelet counts are sufficiently low (<30-40 x 10^9/L; i.e., <30,000-40,000/μL), they also might include signs specifically related to thrombocytopenia (e.g., mucosal petechial and ecchymotic hemorrhages and bleeding from injection or surgical sites).

- Spontaneous hemorrhage (epistaxis, hyphema, hematuria) is rare unless the platelet count is less than 10 to 20 x 10^9/L (i.e., < 10,000-20,000/μL). However, cases with serious underlying disease processes may show evidence of spontaneous bleeding even in the presence of greater platelet numbers.
- Tests including a Coggins test, Coombs test, and bone marrow evaluation should be used to rule out secondary immune-mediated thrombocytopenia. In addition, review of recent drug therapy and thorough investigation for other infectious or neoplastic conditions should be undertaken. Normal or increased numbers of megakaryocytes should be present in bone marrow.
- Bone marrow aspiration or biopsy also may identify aplastic, neoplastic, and infiltrative marrow disorders.
- Anemia, leukopenia, or pancytopenia frequently accompanies bone marrow diseases.
- Although bleeding time consistently is prolonged, PT and aPTT are prolonged only in cases of DIC.
- If the underlying condition has been present long term, there may be anemia and hypoproteinemia.

KEY POINT

True idiopathic thrombocytopenia is a diagnosis of exclusion, and response to immunosuppressive therapy usually is diagnostic.

DIFFERENTIAL DIAGNOSIS

- Chronic inflammatory disease (e.g., pneumonia, abscessation, cellulitis)
- DIC
- Septicemia or toxemia
- Vasculitis
- Immune-mediated anemia
- EIA
- Ehrlichiosis
- Myeloproliferative bone marrow disorders
- Lymphosarcoma

TREATMENT

- With elimination of the primary cause, platelet counts usually improve. If the cause of thrombo-

cytopenia is thought to be associated with drug administration, treatment should be stopped, and in many cases there will be spontaneous recovery.
- Appropriate therapy for DIC should be begun if indicated.
- Where platelet counts are particularly low ($<10 \times 10^9$/L or 10,000/μL) and there is significant hemorrhage, administration of whole fresh blood, fresh platelet-rich plasma, or platelet concentrates is indicated to temporarily increase platelet numbers. Platelet-rich plasma and platelet concentrates are produced using plasmapheresis technology by commercial enterprises that market equine plasma products.
- Idiopathic thrombocytopenia may respond to systemic corticosteroid treatment. Of the corticosteroids available, the best response is likely to occur with administration of dexamethasone (Treatment Nos. 29 and 30) at dose rates of 0.05 to 0.2 mg/kg given IV or IM q24h. Initially, higher dose rates (0.2 mg/kg) should be used for up to 1 week until platelet numbers increase above 100×10^9/L (i.e., 100,000/μL). After this, the dosage can gradually be reduced by 0.01 mg/kg/day as long as platelet counts remain normal over a further 10 to 16 days. When the dosage of dexamethasone is less than 0.04 mg/kg/day, oral prednisolone therapy (Treatment No. 93) may be given at equivalent anti-inflammatory dose rates (0.4 mg/kg PO once daily in the morning), decreasing to an every-other-day dosage regimen.
- Most horses with idiopathic thrombocytopenia have a good prognosis and usually recover after 10 to 21 days of therapy. However, if there is no response to corticosteroids, azathioprine (Imuran, Burroughs Wellcome Co., Research Triangle Park, NC) may be used at 3 mg/kg PO q24h for 24 days with the dosage gradually reduced after 7 days.

Vasculitis

Equine vasculitis is a syndrome of diseases manifest by inflammation of blood vessels regardless of size, location, or cause. Usually, vasculitis results from type III hypersensitivity reactions secondary to systemic infections, neoplasia, and occasionally drug administration. Small vessels of the skin primarily are involved, although other organs occasionally may be affected. In rare cases, the vasculitis may be due to direct damage to vessel walls associated with some viruses (e.g., equine viral arteritis), chemicals, and endotoxins.

KEY POINT

The major pathogenic mechanism for immune-mediated hypersensitivity vasculitis is deposition of antigen-antibody complexes in vessel walls. Activation of complement and neutrophil chemoattraction result in release of proteolytic enzymes that cause vessel wall dysfunction with subsequent edema, hemorrhage, and ischemia of supplied tissues.

Vasculitis syndromes characterized in the horse include equine purpura hemorrhagica, EIA, EVA, infection with *Ehrlichia equi,* photoactivated vasculitis, and certain drug-induced vasculitides (e.g., quinidine). Purpura hemorrhagica is the most common form of vasculitis and is thought to be associated with hypersensitivity to *Streptococcus equi* var. *equi* ("strangles") antigens. Other causes of purpura include equine influenza virus, other *Streptococcus* species, or other antigenic stimulation. However, in approximately 50% of cases of vasculitis in horses, the etiology and clinical course are poorly defined.

HISTORY AND PRESENTING SIGNS

- *S. equi* var. *equi* infection 2 to 4 weeks previously; recent strangles vaccination or exposure to strangles without clinical signs
- Localized or generalized edema
- Skin necrosis
- Reluctance to move
- Signs of depression

CLINICAL FINDINGS AND DIAGNOSIS

- Although the severity of clinical signs varies greatly depending on the etiology, the most consistent feature is the sudden appearance of localized dermal and subcutaneous edema. The limbs, ventral abdomen, trunk, and face are most often affected.
- In cases of photoactivated vasculitis, the disease affects only the nonpigmented portions of the lower limbs.
- Lesions are not necessarily symmetric and often are warm, painful, and pit on palpation.
- In severe cases, areas of edema can proceed to skin necrosis, exudation, and eventual sloughing.
- Petechial and ecchymotic hemorrhages associated with the mucosa (oral, nasal, ocular, and, in mares, vulval) also are common with mucosal ulceration less frequently observed.
- Fever, anorexia, and signs of depression are variable, whereas tachycardia and tachypnea due to pain are common.

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- Although peripheral manifestations are most common, vasculitis involving the alimentary and respiratory tracts, kidneys, central nervous system, joints, and muscles also may occur. Consequently, signs of colic, diarrhea, dyspnea, azotemia, ataxia, and lameness often are noted.
- Hematologic and biochemical findings are nonspecific. Because most cases are due to systemic disease, there often is neutrophilic leukocytosis, increased plasma fibrinogen and globulin concentrations, and mild anemia. The platelet count usually is normal. However, lymphopenic leukopenia occurs in EVA and ehrlichiosis, whereas thrombocytopenia is common in EIA and ehrlichiosis.
- Histologic examination and direct immunofluorescence testing of skin punch biopsies (see Chapter 13) provide the opportunity for confirming the diagnosis. Tissue samples collected early in the disease process (within 12 hours of their appearance) are most likely to be diagnostic. Also, changes may not be found unless a large number of biopsies are collected.
- Urinalysis may show hematuria and proteinuria suggestive of glomerulonephritis, whereas muscle involvement is reflected by increased serum activities of creatine phosphokinase and aspartate aminotransferase.
- Antibodies against M protein can be detected using ELISA and may support the possibility of a previous infection with *S. equi* var. *equi.*
- Blood samples collected into EDTA (purple top) or lithium heparin (green top) tubes are suitable for virus isolation and serologic tests for diagnosis of EVA, whereas the Coggins radial immunodiffusion (RID) test is available for definitive diagnosis of EIA.

DIFFERENTIAL DIAGNOSIS

- Purpura hemorrhagica
- EIA
- EVA
- Ehrlichiosis
- DIC
- Septicemia
- Thrombocytopenia
- Hypoalbuminemia
- Appendicular inflammatory disorders such as lymphangitis, cellulitis, and immune-mediated diseases

TREATMENT

- There is no specific treatment for vasculitis syndromes, but providing good supportive care is necessary together with appropriate anti-inflammatory and antibiotic therapy.
- Underlying diseases should be treated aggressively. Edema can be minimized by frequent hydrotherapy, hand walking, and pressure bandages to the limbs. Maintenance of electrolyte and fluid balance using intravenous or oral polyionic fluids is essential for horses that fail to eat and drink.
- Nonsteroidal anti-inflammatory drugs such as phenylbutazone (Treatment No. 89) at dose rates up to 2.2 mg/kg IV or PO q12h; or flunixin meglumine (Treatment No. 52) at dose rates up to 1 mg/kg IV, IM, or PO q24h; or ketoprofen (Treatment No. 66) at 1 to 2 mg/kg, IV only, q24h, may provide analgesia and reduce inflammation.
- Procaine penicillin (Treatment No. 84) at a dose rate of 15 to 20 mg/kg (15,000-20,000 IU/kg) IM q12h should be used for at least 2 weeks, particularly if streptococcal infection is considered to be active.
- Antimicrobial therapy also is indicated to reduce the incidence or severity of secondary infections such as cellulitis, pneumonia, and thrombophlebitis. The spectrum of antimicrobial therapy should be broadened if gram-negative organisms also are involved.
- Clinical experience indicates a good response in many cases when systemic corticosteroids are administered. Dexamethasone (Treatment Nos. 29 and 30) is the corticosteroid of choice and should be given at a dose rate of 0.05 to 0.2 mg/kg IV or IM once daily, in the morning. Prednisolone (0.5-1 mg/kg IM or PO q12h) may be used but may not be as effective as dexamethasone. With all corticosteroid therapy, the concept is to gradually reduce the dose rate (by 0.01 mg/kg daily over a period of 7-21 days) once clinical response has occurred. Morning administration of dexamethasone results in less adrenal suppression than with afternoon treatment. Horses frequently require 4 to 8 weeks of glucocorticoid therapy. In addition, antimicrobial therapy should be continued throughout the period of glucocorticoid administration to reduce the incidence of secondary sepsis.

KEY POINT

Some cases respond well to therapy, but a proportion of horses either respond poorly or relapse, in particular when steroid administration is reduced. Others develop secondary complications such as laminitis, renal failure, pneumonia, and colitis.

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- The treatment for EVA, EIA, and ehrlichiosis is palliative. Glucocorticoids are contraindicated in both EVA and EIA because there may be increased risk of exacerbating viral replication and clinical disease. Horses with ehrlichiosis should be treated with oxytetracycline (7 mg/kg IV q24h for 7 days), whereas cases with photoactivated vasculitis should be stabled during the day to prevent further exposure to sunlight.

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CHAPTER 13

Dermatology

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Skin diseases in the horse are very common and often present a diagnostic challenge for the clinician. Different skin diseases can present with similar clinical signs; for example, a number of conditions can cause pruritus and alopecia, and similarly, a wide range of conditions can cause nodular skin disease. A thorough approach to investigation is extremely important, and a complete physical examination and detailed history are essential. Vital clues to indicate the most appropriate diagnostic approach may be missed with cursory examinations. Knowing when to use available diagnostic tests greatly increases a clinician's diagnostic skills.

EXAMINATION OF THE SKIN

History

The signalment (breed, age, sex, and use of the horse) may provide important diagnostic clues and should always be accurately recorded (see Chapter 2). A standardized historical questionnaire for investigating skin disease is useful and should include the following questions:

- What is the main problem from the client's viewpoint?
- When did the problem start?
- Where on the body did the problem start?
- What did the initial lesions look like?
- Is the problem static, waxing and waning, or progressive?
- Is there any evidence of pruritus and, if so, was this noticed before, after, or concurrently with the onset of lesions?
- Is there any prior history of skin disease?
- If the problem is chronic, is it seasonal or present all year?
- If there are other horses, are they in contact and do they have any skin disease?
- If there are other horses affected, is there any sharing of grooming equipment?
- Is the horse showing any generalized signs of ill health?
- What treatment has been given for the condition to date, and what response was there to each treatment?
- What topical shampoos or other products are currently used?
- What is the current diet of the horse and are any supplements given?
- Have there been any alterations to diet, stabling, exercise, and so on, and, if so, did the changes seem to influence the skin disease?

Important background information to note in considering the history and presenting signs is as follows:

Geography. Location of the horse will influence the range of possible skin diseases. It is important to know past and present locations of the horse and the prevalence of disease in your geographic location. For example, blastomycosis, coccidioidomycosis, and pythiosis are confined to specific regions.

Season. Some diseases are more common at different times of the year. For example, insect hypersensitivity is more common in summer/autumn with warmth and moisture, and dermatophilosis is more common during the wetter winter periods.

Local Environment. If the horse is stabled, attention must be paid to the type of bedding and

feed, the management of the horses in the stables, use of grooming brushes and cloths, and general care of the horse. If the horse is at pasture, the pasture types, extent of shade, water and feeding facilities, and fencing should all be examined because of possible relevance to the presenting problem.

Physical Examination

Examination of localized skin problems is often performed without a detailed examination of the whole skin or the rest of the body. When the clinician is presented with a large mass on the limb or an area of alopecia on the thorax, it is easy to bypass a more detailed examination that could reveal other relevant abnormalities, such as other skin involvement, lymph node enlargement, evidence of anemia, or elevated body temperature. It is important to examine the whole animal with a general physical examination, as outlined in Chapter 1. In particular, regions such as the oral cavity, mucous membranes of the nose, and the conjunctivae should be carefully inspected. A careful physical examination of the skin should next be made, starting at the head and ending at the tail. Examination of the coronary band and hooves is important because they may provide evidence of a more generalized problem. Note should be taken of changes in the quality of the hair coat (e.g., dry, brittle, dull, or easily epilated) or to the general character of the skin (e.g., changes in elasticity, extensibility, or thickness). The distribution of the lesions should be noted, and the lesions should be described accurately and classified as primary or secondary when possible.

KEY POINT

A thorough search for primary skin lesions should be made. Primary lesions are often less prevalent and less obvious than secondary lesions but provide the best diagnostic clues and are the best sites for taking samples for diagnostic testing.

This is because primary lesions are often directly caused by or are a direct reflection of the underlying disease. Primary lesions include papules, pustules, vesicles, nodules, macules, and wheals. Secondary lesions occur when the skin is altered by factors such as self-trauma, infections, and response to therapy. Secondary lesions do not directly relate to the causal disease process. They include ulcers, erosions, excoriations, fissures, epidermal collarettes, lichenification, and scars. Lesions, either primary or secondary, include scale, crusts, pigmentary changes (e.g., hyperpigmentation), alopecia, comedones, and follicular casts.

DIAGNOSTIC AIDS

For investigation of skin problems, little sophistication is needed to perform the basic diagnostic tests, with many tests being quick, easy, and inexpensive to perform. Nonetheless, many clinicians facing a skin disease will opt for treatment rather than trying to make a specific diagnosis.

Skin Scrapings

- Skin scrapings are always an important part of the diagnostic workup in a horse with skin disease. If possible, areas of primary lesions such as papules or alopecia without evidence of self-trauma should be scraped using a no. 10 or 12 scalpel blade and a small amount of mineral oil, as shown in Figure 13-1. The type of mite you could expect to find should always be kept in mind, because this will dictate the depth, number, and location of scrapings needed. For example, demodectic mites reside deep in the hair follicles and are mostly plentiful if causing disease; therefore, one deep scraping producing capillary bleeding is usually diagnostic. In contrast, chorioptic mites live very superficially, feeding on epidermal debris, and are usually easily found in one superficial scraping. Sarcoptic mites also live superficially but are often scarce, so multiple superficial scrapings are indicated if these mites are suspected.
- Samples should be placed directly onto glass slides with a few drops of mineral oil placed on top and can then be transported if needed inside

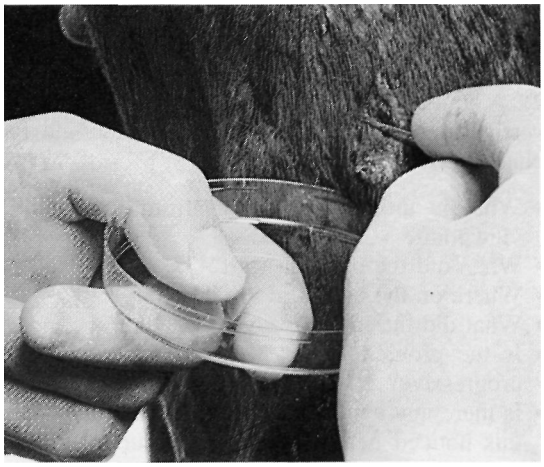


Figure 13-1. Skin scraping using a scalpel blade.

Petri dishes. To examine these samples under a microscope, cover slips should be placed on top of the slides to ensure a uniform layer, and a systematic search of all scraped material should then be made. If the microscope condenser is lowered, there is an increase in contrast that allows mites to be seen more easily.

Cytology

KEY POINT

Cytologic examination of the skin is another simple and fast diagnostic test that is greatly underused in evaluating skin disease. Very useful information can be obtained that may confirm a diagnosis, identify secondary infections requiring specific treatment regardless of the underlying problem, or narrow the differential diagnoses and guide further diagnostic workup.

- Cytologic examination can reveal the presence of bacteria, yeasts, fungal hyphae, neoplastic cells, different types of inflammatory cells, and acantholytic cells, which are vital clues to the underlying disease. For example, large numbers of eosinophils may be seen with parasitic or allergic disease. Degenerate neutrophils suggest infection, and bacteria being phagocytosed by inflammatory cells confirm bacterial infection. Acantholytic cells and nondegenerate neutrophils in the absence of bacteria suggest pemphigus foliaceus. Cells may be seen that display characteristics of neoplasia. Samples can be collected in a number of ways, depending on the types of lesions present.
- With superficial lesions, direct impression smears, swab smears, or sticky tape preparations are mostly used. Direct impression smears are made by firmly pressing the surface of a glass slide onto the skin. They can be made on moist areas, including the underside of crusts, the surface of the skin beneath crusts, and areas with exudate or discharge. They can also be made after gently opening the surface of pustules, papules, or vesicles. These can be stained with simple stains such as modified Wright's stain (Diff-Quik) or new methylene blue. Dermatophilosis is easily diagnosed in this manner.
- Dry superficial areas can be examined by performing swab smears or sticky tape preparations. Swab smears are made by moistening a cotton-tipped applicator with saline and rubbing it briskly over the area to be examined. Ear canals are also frequently examined this way. The swab is then rolled over the surface of a glass slide, which is then stained as for impression smears.
- Sticky tape preparations are made by firmly pressing clear acetate tape (e.g., 3M Scotch No. 602 tape) directly onto the skin in a number of places in the affected area and then placing the tape sticky-side down onto a microscope slide. Staining is performed by placing a few drops of blue stain (e.g., methylene blue or Diff-Quik blue) directly onto the slide before gently pressing the tape in place, then wiping the surface to distribute the stain underneath, or by performing a complete Diff-Quik stain of the tape and then attaching it to a slide. Sticky tape preparations are easier and faster to prepare than swab smears and may provide a better representation of cells and organisms from dry areas, but they can be more difficult for the clinician unfamiliar with their interpretation. Organisms and cell types present in large numbers can usually be readily identified, but subtle changes can be harder to recognize and interpret.
- Cytology samples from deeper lesions are frequently obtained by fine-needle aspiration and then immediately sprayed onto a microscope slide and stained as for an impression smear.

Bacterial Culture

- Superficial bacterial infections frequently have predictable antibiotic susceptibilities and also often respond to topical antibacterial treatment alone. Bacterial culture may be unnecessary.

KEY POINT

Bacterial culture should be considered if there are nodular, granulomatous, or draining lesions; if cytologic examination shows unusual bacteria; or if there has been a poor response to previous appropriate antibiotic therapy.

- Samples for bacterial culture can be tissue biopsy specimens or aspirates from pustules. Swabs taken from purulent tracts are often of little value for bacteriologic study because a mixed growth of secondary and invading organisms will mostly be found.
- Before aspirating from a pustule or collecting a biopsy specimen for culture, the area should be disinfected, and collection of samples must be performed aseptically. The surface of biopsy samples can also be carefully removed with a scalpel after collection to further decrease the risk of culturing nonsignificant surface bacteria. Transport to the laboratory on transport media is required. Cytologic examination should always be performed before attempting bacterial culture to ensure that results are significant.

Fungal Examination

- If fungal infection is suspected, isolation and identification of the species involved is essential. Despite this, confirmation of fungal infection can sometimes be made at the time of sampling without having to wait for culture results. Fungal hyphae or spores may be identified on cytologic examination of tissue or discharge. With dermatophytes, Wood's light examination may reveal yellow-green fluorescence along hair shafts, or fungal spores may be visualized on hairs or keratin by direct microscopic examination. Clearing techniques for hair and keratin examination are sometimes recommended; however, these are tedious to perform and may not increase diagnostic yields. Suspension of the hair and keratin debris in mineral oil can be equally successful. Hair shaft examination requires practice and expertise and will not always show fungal spores despite fungal infection. Fungal culture together with topical antifungal treatment while awaiting results is often the preferred option.

KEY POINT

Dermatophyte infection can be confirmed by direct microscopic demonstration of fungal spores on hair shafts but cannot be ruled out if no spores are seen.

- Similarly, positive fluorescence on Wood's light examination is diagnostic. However, most dermatophytes commonly infecting horses do not fluoresce.

Fungal Culture

- Samples for fungal culture in suspected dermatophytosis should include hair and superficial keratin from the periphery of lesions, obtained via skin scrapings and hair pluckings. For deeper fungal infections (systemic or subcutaneous mycoses), tissue samples obtained by biopsy are preferred, although exudates when cultured can be positive for fungi.
- Special media are needed for fungal culture, and the laboratory should be informed of the possible diagnoses when samples are submitted. Histopathologic examination of affected tissue is advised if deeper fungal infections are suspected, because fungal culture may be unreliable or take many weeks.

Skin Biopsy

- Skin biopsy is a simple diagnostic aid that can be carried out easily with a minimum of equipment. Unfortunately, biopsies frequently are performed without thought to the major differential diagnoses for that case and without the prior performance of more basic diagnostic tests.

KEY POINT

Diagnostic yield from biopsies can be greatly enhanced by selecting cases to biopsy more carefully, performing prior diagnostic tests or trials if indicated, treating secondary bacterial infections first if appropriate, discontinuing therapies that may alter results before sampling (e.g., prednisolone therapy), and having definite diagnostic goals in mind.

- Biopsies always should be recommended with severe or very unusual skin disease, if malignant or aggressive neoplasia is possible, or if there is persistent ulceration. They are also indicated if diseases requiring immunosuppressive or expensive treatment are suspected, if the differential diagnoses being considered have a very different prognosis, and if there has been poor response to apparently appropriate therapy and previous diagnostic tests have not been conclusive.
- Biopsy may not be very useful with chronic problems associated with secondary infections, secondary changes from self-trauma, and/or changes due to recent therapies.
- Allergic skin diseases also are frequently unrewarding to biopsy, because histopathologic changes are mostly very similar for the different types of allergy. Allergies are often strongly suspected based on the history, clinical findings, and initial diagnostic tests (negative skin scrapings, supportive cytologic findings), and further diagnostic effort and money can often be better directed at differentiating between the possible allergic causes using skin testing, an insect control trial, and/or an elimination diet. Biopsy may be useful if the case is atypical and confirmation of an underlying allergy is desired before proceeding with further diagnostic tests or trials.
- To obtain biopsies, disposable punch biopsy needles are convenient and usually suitable. An 8-mm-diameter biopsy punch should be used where possible. Careful selection of biopsy sites will increase the diagnostic yield from biopsies. Primary lesions should be sampled wherever possible, and a number of samples from a representative range of lesions should ideally be taken.
- Normal skin should not be included in a punch biopsy of affected skin. This is because when the biopsy is sectioned for processing, it is possible to miss affected areas and end up with only normal skin in the sample, requiring se-

quential cuts to find the desired section. If it is desirable to biopsy the edge of a lesion and include normal skin, a wedge-shaped excisional biopsy sample is required, which will always be sectioned longitudinally and will thus always include affected and normal areas. Excisional wedge biopsy is also indicated with large, deep, or fragile lesions such as large pustules, nodules, or vesicles.

- Biopsy specimens can frequently be collected using physical restraint and local anesthesia. A small bleb of local anesthetic should be placed beneath the skin at the biopsy site. Ideally, there should be no skin surface disinfection performed. At most, gentle soaking with 70% alcohol is permitted. Hair can be gently clipped away if needed. Using a biopsy punch, the punch is rotated in one direction with firm pressure to penetrate the skin down to the subcutaneous tissue (Fig. 13-2). The piece of skin is then gently removed using a pair of sterile forceps and a scalpel blade (Fig. 13-3). Samples are fragile, and care should be taken to handle them carefully to avoid crushing artifacts. Holding with large tissue forceps should be avoided. A single suture of nonabsorbable material can be placed to close the biopsy site.
- The fixative most widely used for skin biopsies is 10% buffered formalin. If special procedures are being performed, special fixatives may be required, and the laboratory handling the samples should be contacted for advice.

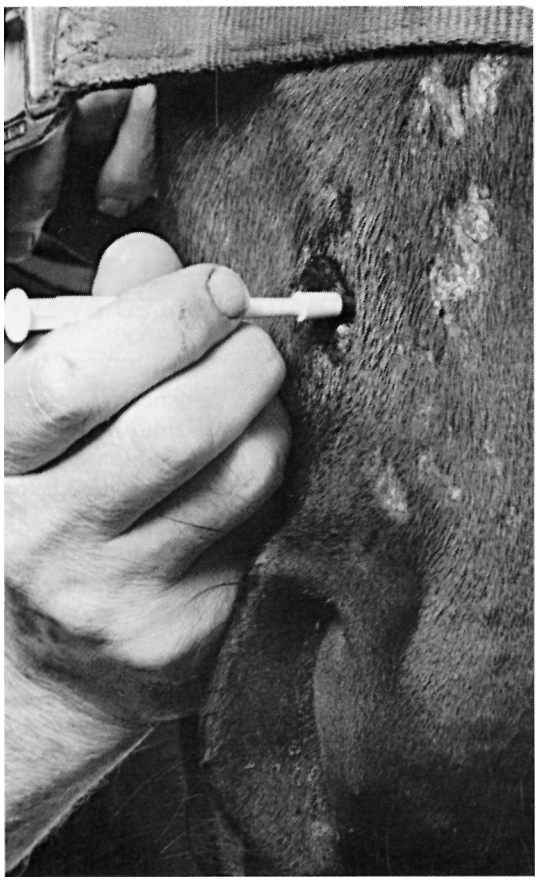


Figure 13-2. For collection of a skin biopsy specimen, an 8-mm biopsy punch is rotated through the skin after injection of local anesthetic.

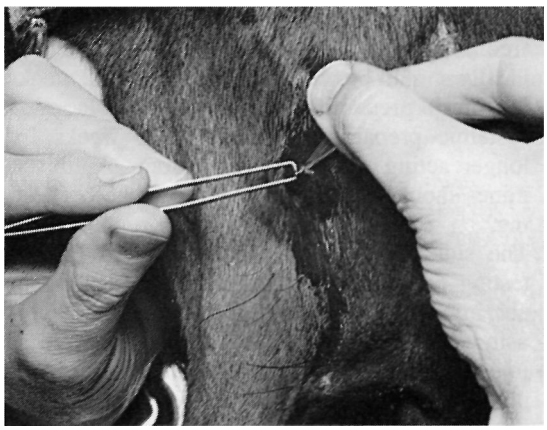


Figure 13-3. After the biopsy punch has been rotated through to the subcutaneous tissue, a scalpel blade and forceps are used to remove the piece of skin. Single interrupted or cruciate sutures are placed in the biopsy site.

Allergy Testing

- *Intradermal skin testing* is becoming more widely available and currently is thought to be the most specific test for identifying airborne allergens causing atopy in horses. Testing involves injecting a panel of allergens (e.g., pollens, molds, dust mites) intradermally. If a horse is presensitized to an allergen, it will have IgE antibodies specific for that allergen bound to dermal mast cells in the skin. Intradermal injection of that allergen results in cross-linking of the IgE antibodies, which induces degranulation of mast cells with release of many inflammatory mediators. This reaction, often referred to as a type I hypersensitivity reaction, produces an inflammatory wheal in the skin.
- Allergens causing insect hypersensitivity can also be identified with an intradermal skin test, and more recently, testing is being used to help evaluate some forms of respiratory disease, especially chronic obstructive pulmonary disease (see Chapter 5).
- Testing can be interfered with by certain drugs,

including sedatives, anesthetic agents, glucocorticoids, and antihistamines. Glucocorticoids ideally should be withdrawn for at least 2 weeks for topical preparations, 4 weeks for short-acting systemic preparations, and 4 to 8 weeks for longer acting systemic preparations. Antihistamines should be withdrawn 2 weeks before testing.

- The side of the neck usually is used for the testing procedure, and after clipping, allergens are injected *intradermally* at premarked spots using 26- or 27-gauge needles. Histamine is injected as a positive control and saline as a negative control. Wheals produced at each injection site are scored compared with the positive and negative controls for wheal size, induration, and erythema using a standard scoring system. Scores are recorded after 15 to 30 minutes, then ideally at 4 to 6 hours, and then at 1 and 2 days after the intradermal injection.
- Performance of intradermal skin testing and interpretation of results require expertise and are best delegated to specialist dermatologists. Allergens used in the test must be selected appropriately for locality. It is essential to rule out other potential differential diagnoses before relying on test results, because false-positive results can occur where sensitization to certain allergens has occurred but is not the important factor causing the current disease.

Serum Testing

- Radioallergosorbent test (RAST) or enzyme-linked immunosorbent assay (ELISA) test are available in some areas for diagnosing and formulating therapy for atopy in horses. However, the value of such tests has not been firmly documented. These tests detect relative levels of allergen-specific IgE in serum rather than in the skin, as in intradermal testing. Serum testing has been used more commonly in humans and more recently in dogs for the diagnosis of atopy, but false-positive reactions are reported to be more common than with intradermal testing, and serum test results may not correlate well with those of skin testing. Technical difficulties with the serum tests mean that results can vary widely between different tests and also when the same test is run at different laboratories. If skin testing is not available, serum testing is an option for the evaluation of atopy, but care must be taken to rule out other possible diagnoses first (e.g., parasitic dermatoses, food or insect hypersensitivity) and to use a test provided by a reliable laboratory that can document results from its testing in normal horses. The test should use single rather than grouped allergens, and it has been advised that a good deworming program be in place for 3 months before testing to limit elevations of IgE due to parasitic burdens.

Skin Diseases

The skin is the largest organ in the body, and although it can be affected by a wide variety of diseases, it can react to disease in only a limited number of ways. Thus, many diseases can appear similar and cause similar presenting signs yet have vastly different etiologies. Some skin diseases have predictable clinical features, whereas others can have a variety of clinical forms. However, with a logical and thorough investigative approach, a diagnosis can often be made that allows the most appropriate treatment. To discuss the common skin diseases in this chapter, they are categorized based on their major presenting signs:

1. Skin disease characterized by pruritus
2. Nonpruritic skin disease with alopecia, scaling, and/or crusting
3. Nodular skin disease with ulceration and/or exudation
4. Nodular skin disease without ulceration or exudation
5. Miscellaneous skin diseases
6. Traumatic skin disease

SKIN DISEASE CHARACTERIZED BY PRURITUS

The following diseases characteristically are very pruritic, but pruritus may range from minimal to intense among horses with the same disease. Some owners may not recognize pruritus despite it being an important feature of the disease.

Insect Hypersensitivity ("Queensland Itch," "Sweet Itch," "Summer Itch")

KEY POINT

Hypersensitivity reactions to biting insects are one of the most common causes of pruritic skin disease throughout the world.

Hypersensitivity to the biting midge *Culicoides* is the best documented insect hypersensitivity in the horse, but hypersensitivity reactions to other biting insects are thought also to occur frequently (e.g., horse flies, black flies, stable flies, mosquitoes, sandflies).

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HISTORY

- Insect hypersensitivity tends to be a seasonal problem, being more common during the summer months, except in the tropics, where insects are active throughout the year.
- Disease most commonly first occurs within the first 4 years of life but can start at any age and usually worsens with age. If the affected horse is part of a herd, most others will be unaffected. However, because insect hypersensitivities are common, appear to have genetic predilections, and are seen only when the causal insects are present, there may be a number of individuals affected at the same time.

KEY POINT

Pruritus is a key feature and is often intense.

- Pruritus occurs at the sites where the insects feed, which varies with the insect species.
- Even with *Culicoides*, different species are found in different geographic locations, and the preferred feeding site and thus predominant areas of pruritus on affected horses vary with locality.

CLINICAL FINDINGS

- Lesions frequently are caused by self-trauma due to the pruritus.

KEY POINT

Lesions commonly seen include alopecia, excoriations, hair matting, scaling, crusting, lichenification, and hyperpigmentation. The most likely affected areas are the dorsal midline, ears, mane, and tail.

- In some localities, a more ventral distribution of lesions is seen, with chest, ventrum, and legs being affected. It is important to know lesion distributions most commonly seen in your region and to see if the bites from insects known to occur in your region can account for the distribution of lesions seen.
- Urticaria occasionally is seen as the sole clinical finding.

DIAGNOSIS

KEY POINT

The classic distribution of lesions, pruritus, and seasonal incidence are suggestive of insect hypersensitivity.

- Skin scrapings will be negative. Skin biopsies will be consistent with allergies of any cause and are rarely of use diagnostically. Intradermal skin testing or insect control trials (see Treatment) can be used to confirm the diagnosis.

DIFFERENTIAL DIAGNOSIS

- Food allergy/adverse reaction
- Atopy
- Onchocerciasis
- Ectoparasites (e.g., louse, mite, or tick infestation)
- Dermatophilosis
- Dermatophytosis

TREATMENT

KEY POINT

Insect control is the crucial part of management of insect hypersensitivity and if done effectively will allow complete control of clinical signs. The regular use of an insect repellent, together with attention to environmental control where appropriate, often provides the most effective control.

- Permethrin is one of the most effective insect repellents but often needs to be used daily for full effect. Knowing habits of the offending insects helps greatly with control. For example, *Culicoides* spp. are active in the evening hours, before and after dusk when there is little or no breeze. They breed in stagnant water, decaying vegetation, and manure; thus, stabling overnight with fans, stabling and fine-meshing all areas of access (normal commercial insect meshes will not suffice because of the very small size of the midges), removing stagnant water pools, and/or spraying insect repellent daily before dusk can aid greatly with control. Stable flies breed in decaying vegetation and manure, feed during daylight hours, and may prefer strong light; thus, good sanitation is important for control, together with use of insect repellents applied in the mornings and possibly stabling in dull light during the day.

KEY POINT

An insect control trial can be used as a diagnostic trial to help confirm or disprove the diagnosis of insect hypersensitivity but must be done very thoroughly to be useful.

- If *Culicoides* hypersensitivity is suspected, an insect control trial could involve a thorough daily permethrin spray before dusk, removal of the horse from obvious insect breeding sites and

rugging and/or stabling with fans. Considerable improvement in clinical signs should be seen within 4 weeks if control is effective, which helps to confirm a diagnosis of insect hypersensitivity. Insect control can then be tapered to the minimum effective level for ongoing treatment.
- Superficial bacterial infections can occur concurrently and contribute significantly to the pruritus. They are not always suspected based on appearance of the skin and can be identified using cytologic examination. Treatment with topical antibacterial shampoos often is effective, but systemic antibiotics occasionally may be needed.
- The oral use of prednisolone may be helpful in controlling the clinical signs of insect hypersensitivity. However, it is important to remember that not all horses will respond, and long-term therapy can cause a variety of side effects. Insect control always should be the preferred treatment option. Prednisolone is used at an anti-inflammatory dose of 1 mg/kg twice daily initially and if effective is then tapered to the lowest effective dose given every second day. Dexamethasone can also be used intramuscularly at a dose of 0.1 mg/kg daily initially and then tapered to every third or fourth day, which is often more effective.

Food Allergy/Adverse Reaction

Allergic or idiosyncratic reactions to components of the diet can cause pruritic skin disease in the horse. It is strongly suspected but not proved that an allergic reaction occurs. The offending diet has often been previously fed for months to years without causing problems.

HISTORY

- Skin reactions to food are classically nonseasonal, but they may be seasonal if the offending food is fed only at certain times of the year.
- Horses of any age can be affected.
- Pruritus is often a key feature of the disease but may be absent.

CLINICAL FINDINGS

- Food allergy/adverse reaction may appear identical to insect hypersensitivity.
- Pruritus and alopecia frequently are seen with or without signs of self-trauma such as excoriations, lichenification, and hyperpigmentation.
- Any area of the body can be affected; however, an increased suspicion of food allergy should be raised if the tail alone is affected or affected areas are less likely to be associated with insect hypersensitivity (e.g., flanks, lateral thorax).
- Urticaria also can be seen, either alone or in combination with other signs.

DIAGNOSIS

- The only reliable way of establishing a diagnosis of food allergy/adverse reaction is by performing an elimination diet, which involves feeding a restricted diet for a period of 4 to 6 weeks. Specific foods known to induce pruritus in horses include barley, bran, alfalfa, and supplements. Ideally, the elimination diet should consist of foods the horse has previously not been fed. In practice, this is often difficult, and the trial may need to be performed in different stages. All supplements and unusual foods should be discontinued as a minimum. If grains are fed, they may be discontinued or a novel grain substituted. Feed mixes should be stopped. Horses at pasture can have all additional foods stopped during the diet, and if there is no improvement after 4 to 6 weeks, no pasture feeding is then allowed for the next 4 to 6 weeks. Dietary changes should be made with care, however, and may need to be introduced gradually to avoid gastrointestinal disease. Significant improvement should be evident after 4 to 6 weeks of avoidance of the offending food. Complete resolution may take longer.

KEY POINT

Confirmation of diagnosis requires exacerbation of signs on rechallenge with the previous diet, followed by improvement back on the test diet.

- Return of pruritus is usually seen within 2 to 3 days of rechallenge but may take up to 2 weeks. Once the diagnosis of food allergy/adverse reaction is made, a sequential rechallenge, in which foods are reintroduced one at a time, is performed to identify the offending food(s).

DIFFERENTIAL DIAGNOSIS

- Insect hypersensitivity
- Atopy
- Onchocerciasis
- Ectoparasites (e.g., louse, mite, or tick infestations)
- Dermatophilosis
- Dermatophytosis
- Oxyuris (pinworm) infestation (if only tail base affected)

Atopy

Atopy is an inherited predisposition to form antibodies to environmental allergens, such as pollens, molds, insects, and dust components. It has been well documented and studied in humans and to a lesser extent in dogs but is poorly defined in horses.

HISTORY

- Pruritus is a key feature of the disease. It may be seasonal or not, depending on what the offending allergens are. It tends to start in young adult horses and worsen with age.

CLINICAL FINDINGS

- May appear identical to insect hypersensitivity or food allergy/adverse reaction.
- Pruritus, alopecia, excoriations, lichenification, hyperpigmentation, and urticaria may be seen.

DIAGNOSIS

- Intradermal skin testing is the diagnostic test of choice. It is important to rule out insect hypersensitivity and food allergy/adverse reaction before skin testing.

DIFFERENTIAL DIAGNOSIS

- Insect hypersensitivity
- Food allergy/adverse reaction
- Onchocerciasis
- Ectoparasites (e.g., louse, mite, or tick infestation)
- Dermatophilosis
- Dermatophytosis

TREATMENT

- Hyposensitization based on intradermal skin test results is the treatment of choice. This is a fairly complex and long-term treatment, and the treatment protocol needs to be adjusted to best suit each individual patient, and thus it is often best performed by a specialist dermatologist.
- All other treatment options are symptomatic and need to be used continually while the offending allergens are present. Options include the use of fatty acid supplements, antihistamines, and glucocorticoids, all of which are variably effective at alleviating signs. The antihistamine of choice in horses is hydroxyzine given at a dose of 1-2 mg/kg orally twice to three times daily. Fatty acid supplements that can be given on a trial basis include linseed oil, evening primrose oil, flaxseed oil, and fish oil; however, controlled studies evaluating efficacy and dosages are lacking.

Contact Dermatitis

Contact allergies occur in horses after long-term exposure to tack, plants, bedding, and topically administered agents (e.g., shampoos, insecticides, sprays). These are delayed or type IV hypersensitivity reactions. Direct irritation from substances in contact with the skin can also occur.

HISTORY

- Rapid onset of localized inflammation and irritation occurs.

CLINICAL FINDINGS

- Papules, vesicles, and erythema may be seen initially, which progress to crusting, scaling, alopecia, and if severe to erosions and ulcers. Permanent scarring sometimes occurs. Localized urticaria sometimes is seen.

KEY POINT

In the early stages there is severe irritation at the contact site, with horses showing evidence of pain and sometimes pruritus.

- The muzzle or pasterns and fetlocks frequently are affected if plants are involved. Saddle or girth lesions can occur from tack. Topical products affect areas of application.
- Moisture allows more direct contact with allergens/irritants and decreases the barrier function of the skin; thus, there is an increased chance of contact dermatitis when horses are sweating.

DIAGNOSIS

- Confirmation of contact allergy requires patch-testing, but this procedure is difficult to perform and is currently not standardized for horses. Resolution of lesions with avoidance of suspect substances, followed by recurrence after reexposure, will confirm that direct irritation or allergy exists.

DIFFERENTIAL DIAGNOSIS

- Insect hypersensitivity
- Food allergy/adverse reaction

- Atopy
- Ectoparasites (e.g. louse, mite, or tick infestation)
- Onchocerciasis

TREATMENT

- Removing the offending agent is obviously critical to effective treatment. Topical antibiotic/glucocorticoid ointments or systemic glucocorticoids may be useful to decrease the initial discomfort.

Ectoparasites

KEY POINT

A number of ectoparasites can cause pruritus and alopecia in horses.

Louse infestation is relatively common, particularly in herds of horses housed indoors during the winter months. Two types of lice are found: biting lice *(Damalinia equi)* and sucking lice *(Haematopinus asini).* **Mite** infestations are less common, and responsible organisms include *Sarcoptes scabei* var. *equi, Psoroptes equi, Chorioptes equi,* forage mites (e.g., *Pyemotes tritici),* poultry mites *(Dermanyssus gallinae),* and trombiculids. *Demodex cabilli* and *Demodex equi* are normal inhabitants of the skin in horses and can rarely be associated with skin disease. **Tick** infestations can also occur in horses but seldom cause signs of pruritus.

HISTORY

- Lice—often seen in winter; horses are mostly not well-groomed
- *Sarcoptes* and *Psoroptes*—rare; geographic variation in prevalence
- *Chorioptes*—increased risk in horses with well-feathered fetlocks (e.g., draft horses) and during winter
- *Demodex*—part of normal skin flora in small numbers; demodicosis is seen with increased numbers of mites and thought to be associated with immunosuppression (e.g., debilitating illness or glucocorticoid therapy)
- Trombiculidiasis—disease caused by trombiculid larvae feeding in skin and seen with access to heavily infested pasture areas, mostly in summer and autumn when larvae are active
- Forage mites—live in straw and grain; contact with contaminated foodstuffs may cause irritation of contact areas
- Poultry mites—adult mites live and lay eggs in bird nests or poultry houses and occasionally cause dermatitis in horses

CLINICAL FINDINGS AND DIAGNOSIS

Lice

- Lice are commonly located around the mane and tail, and horses will often show evidence of intense pruritus—rub at their manes, bite at their flanks, and rub their tails. Examination of the coarse mane hairs usually demonstrates eggs, and sometimes the lice can be seen.

Chorioptic Mange ("Leg Mange")

- Mites mostly affect the legs. Alopecia, erythema, and/or crusting of the pasterns and fetlocks may be seen. Occasionally, horses will stamp their feet because of the irritation.
- Some horses have asymptomatic infestations. Numerous mites are usually seen on superficial skin scrapings.

Psoroptic Mange

- The common sites for the mites are the mane, forelock, and tail.
- Pruritic otitis externa, probably caused by a different species of mite, is also reported. Extreme pruritus is found, with lesions ranging from papules to scaling and crust formation. There is variable alopecia.
- Mites are easily seen in superficial skin scrapings.

Sarcoptic Mange

- Pruritus is characteristically intense. Lesions start on the head and neck and may progress to involve the whole body; however, the mane is usually not affected. These mites tend to burrow into the epidermis, unlike the other types of mange mites. There may be severe excoriation of affected skin with extensive alopecia.
- Multiple skin scrapings taken from over a wide area and then soaked in mineral oil are generally recommended; however, mites are usually few in number and may be difficult to demonstrate.

Demodectic Mange

- Demodicosis is a noncontagious disease. Clinical signs are similar to those of disease in other affected species and include alopecia, scaling, erythema, and excoriations. Pruritus may occur but often is absent.
- Diagnosis is made with deep skin scrapings, and mites usually are plentiful.

Trombiculidiasis ("Chiggers")

- Papules and wheals progressing to crusting and excoriations are seen, mostly on distal limbs,

face, and muzzle. Larvae are visible as orange dots in the center of papules early in the infestation. Pruritus may be intense or absent. Diagnosis is easy early in disease when larvae are visible, but they feed for only a few days, so diagnosis can be difficult in chronic cases.

Forage and Poultry Mites

- Papules and crusts are seen in areas of contact—mostly limbs, muzzle, and ventrum.
- Diagnosis is made on demonstration of mites in skin scrapings and/or environment.

DIFFERENTIAL DIAGNOSIS

- Insect hypersensitivity
- Food allergy/adverse reaction
- Atopy
- Onchocerciasis
- Dermatophilosis
- Dermatophytosis

TREATMENT

- Lice and ticks respond to treatment with topical organophosphate or pyrethrin products.

KEY POINT

Lice eggs are unaffected by topical treatments, hatching after 10 to 14 days; thus, treatment must be repeated two or three times at 14-day intervals. It is also important to spray all the gear associated with the horse, including rugs, cloths, and the stable or other accommodation.

- Sarcoptic, psoroptic, and chorioptic mites and sucking lice can be treated with ivermectin administered at a dose rate of 0.2 mg/kg and repeated at 14-day intervals for a total of three treatments. Chorioptic mange has been reported to be resistant to ivermectin in some cases, and topical insecticides as used for lice and ticks are an alternative treatment.
- Treatment for demodectic mange in the horse is not well evaluated. Lesions may regress spontaneously. Amitraz is used to treat demodicosis in dogs but *must not be used* in horses because it is toxic. A search for underlying disease should be made or glucocorticoid administration stopped.
- Trombiculids and forage and poultry mites can be easily killed with organophosphate or pyrethrin sprays applied to the affected skin and to the environment. Contaminated forage should be removed. Lesions are self-limiting if parasites are removed.

Onchocerciasis

Onchocerca cervicalis is a nematode parasite that infects horses worldwide. Adult worms live within the ligamentum nuchae. Microfilariae migrate through connective tissue and then localize in the skin, most frequently in the region of the ventral abdomen.

KEY POINT

Pruritic skin lesions may occur in response to localization of the microfilarial stage in the skin, but most horses are asymptomatic.

It is most likely that the clinical signs of disease are related to a hypersensitivity reaction. *Culicoides* species serve as intermediate hosts for the microfilariae. *Culicoides* hypersensitivity may occur concurrently, and differentiation between these two diseases is important, especially in areas where ventral pruritus is typically seen with *Culicoides* hypersensitivity.

HISTORY

- Disease is most common in warm to hot weather.

CLINICAL FINDINGS

- Skin lesions are variable and include alopecia, depigmentation, erythema, and crusting. A circular lesion of alopecia, depigmentation, and scaling, commonly called a "bull's eye" lesion, may be seen in the center of the forehead. This lesion is sometimes considered specific for onchocerciasis but may be seen with other dermatoses also. Pruritus is variable but may be intense.

KEY POINT

Most lesions are localized to the ventral abdomen or chest but also occur on the head and neck.

DIAGNOSIS

- Microfilariae can be demonstrated by taking a skin biopsy, chopping the sample into fine pieces, and incubating it in 0.9% saline at 37°C (98.6°F) for 10 to 15 minutes. The microfilariae can be seen in the solution. However, because the microfilariae of *Onchocerca* are commonly found in asymptomatic horses, the presence of microfilariae does not indicate a clinical problem.

- Histopathologic examination of skin biopsies may reveal microfilariae in the superficial dermis with a surrounding eosinophilic inflammatory infiltrate.
- Diagnosis can also be confirmed by response to treatment with ivermectin. Total resolution of clinical signs is expected within 2 to 3 weeks; however, initial exacerbation of symptoms may occur.

DIFFERENTIAL DIAGNOSIS

- Insect hypersensitivity
- Ectoparasites
- Dermatophilosis

TREATMENT

- Ivermectin administered orally at a dose rate of 0.2 mg/kg is effective in eliminating the microfilariae but does not kill the adult *Onchocerca*. Repeated treatments are necessary at 3-month intervals. The death of microfilariae causes intense pruritus in some horses.

KEY POINT

A thorough ophthalmoscopic examination should be performed before treatment because uveitis may be seen as another manifestation of onchocerciasis, and it may be markedly worse after microfilaria are killed.

- Reactions to treatment may be decreased by the prior administration of a short-acting glucocorticoid such as dexamethasone disodium phosphate at a dose of 0.1 mg/kg IV or IM.

Oxyuris Equi (Pinworms)

These nematodes parasitize the cecum and large colon of horses. The female nematode crawls out of the anus and deposits eggs on the skin of the perineum, inducing pruritus.

HISTORY

- An inadequate anthelmintic program will exist.

CLINICAL FINDINGS

- Constant tail rubbing causes breakage and loss of hair at the base of the tail ("rat tail").

DIAGNOSIS

- A tape sample from the anus and perineum reveals characteristic operculated eggs.

DIFFERENTIAL DIAGNOSIS

- Food allergy/adverse reaction
- Insect hypersensitivity

TREATMENT

- Routine anthelmintic use and good stable hygiene.

NONPRURITIC SKIN DISEASES WITH ALOPECIA, SCALING, AND/OR CRUSTING

Most of these diseases may sometimes be pruritic; however, if pruritus is present, it is usually mild and not a key feature of the disease. Occasionally there will be exceptions to this generalization, so all historical features and clinical findings must be considered together when formulating differential diagnoses.

Dermatophilosis (Rain Scald)

Dermatophilosis is an infectious disease caused by the gram-positive anaerobe *Dermatophilus congolensis.* The source of the organism is thought to be carrier animals, crusts from affected animals, and/or the soil. Prolonged exposure to rain is a key component in the development of the disease. However, many other factors, including loss of skin barrier function, also are involved.

HISTORY

KEY POINT

Dermatophilosis is a common condition affecting outdoor horses during wet weather and is characterized by moist crusty lesions affecting skin over the dorsum.

- Some horses seem predisposed to developing disease and will be affected in repeated seasons, whereas other horses historically remain unaffected.

CLINICAL FINDINGS

- Early lesions are multiple small crusts at the base of matted hair, which progress to larger crusts with purulent exudate and annular erosions beneath them.

KEY POINT

The classic features of dermatophilosis are matted tufts of hair and crusting along the

dorsum. Lesions are nonpruritic but may be painful.

- In some cases, dermatophilosis can affect the muzzle and distal limbs. White areas are often more severely affected.

DIAGNOSIS

- Clinical findings are suggestive of this disease, and diagnosis is confirmed using cytologic examination of impression smears collected from the underside of crusts or the surface of an erosion. Crusts can also be minced and mixed with a few drops of sterile water on a glass slide. Gram or Diff-Quik staining reveals typical branching filaments with parallel rows of gram-positive cocci, giving a classic "railroad track" appearance.

DIFFERENTIAL DIAGNOSIS

- Pemphigus foliaceus
- Dermatophytosis
- Bacterial folliculitis
- Photosensitization (if only white areas affected)

TREATMENT

KEY POINT

The most important aspect of treatment is removing exposure to moisture. Most cases regress spontaneously if the horse can be kept dry.

- Topical antibacterial products such as chlorhexidine, 1% povidone-iodine solution, or povidone-iodine shampoo will hasten resolution of lesions. Soaking for at least 10 minutes during treatment will loosen crusts and facilitate their removal. Crusts are infectious to other animals at risk, so careful removal and disposal of crusts are advised. Treatment should be continued daily for the first 7 days and then twice weekly until lesions are resolved.
- Removal of any other obvious predisposing factors will help with resolution also (e.g., following good anthelmintic programs, treating concurrent external parasites, and ensuring adequate nutrition).
- For severe cases or when topical treatment is impractical, systemic antibiotics can be used. Penicillin G and potentiated sulfonamides are reported to be effective.

Dermatophytosis (Ringworm)

Dermatophytosis is a fungal infection of the superficial layers of the skin and hair fibers with organisms that utilize keratin.

HISTORY

KEY POINT

Dermatophytosis is one of the most common skin diseases affecting stabled horses. It is found most often in young horses and is caused by two main fungal species, Trichophyton *and* Microsporum.

- There seems to be a seasonal pattern of the disease in the Northern Hemisphere, with most cases occurring in winter and fall, probably associated with close indoor confinement.
- Infection is common in racing stables, particularly in yearlings and 2-year-old horses.
- Dermatophytosis can spread via direct contact but more commonly by contaminated grooming equipment, blankets, and cloths.
- The incubation period may be quite prolonged, with some cases taking up to 4 weeks to show clinical signs.

CLINICAL FINDINGS

- Dermatophytosis most commonly presents as multifocal circumscribed areas of alopecia, scaling, and crusting; however, lesions can be highly variable. In the early stages, lesions can appear like urticaria, with hairs standing erect and exudation on the surface.
- Lesions are commonly found in the girth/saddle region.
- Pruritus may be present, particularly during the early stages of the infection, but in most cases is absent.

DIAGNOSIS

- Fungal culture is the diagnostic test of choice. Hairs can be submitted to a diagnostic laboratory and/or inoculated onto dermatophyte test medium.

KEY POINT

Apart from infections in isolated individual horses, determination of the specific dermatophyte species involved is advised, especially if cases are multiple, recurrent, or chronic. This directs the search for the source of the infection, which is vital to treatment and to prevention of reinfection.

- Wood's lamp examination often yields negative results because the commonly involved species do not fluoresce. Direct microscopic examination of hair shafts or keratin debris may demonstrate fungal spores; however, the absence of spores does not rule out dermatophytosis, and the procedure requires some expertise and time to perform.

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FUNGAL ORGANISMS CAUSING EQUINE DERMATOPHYTOSIS

- *Trichophyton equinum*—most commonly involved, nonfluorescing, zoophilic; infection most commonly transmitted from horse to horse
- *Microsporum canis*—occasionally involved, some strains fluoresce, zoophilic; infection most commonly transmitted from dogs and cats but also from horse to horse
- *Trichophyton mentagrophytes*—occasionally involved, nonfluorescing, zoophilic; infection most commonly transmitted from guinea pigs and rodents but also from dog and cat to horse
- *Microsporum gypseum*—occasionally involved, nonfluorescing, geophilic; infection mostly acquired from soil, causes highly inflammatory lesions

DIFFERENTIAL DIAGNOSIS

- Dermatophilosis
- Pemphigus foliaceus
- Bacterial folliculitis
- Occult (fiat) sarcoids

TREATMENT

- Dermatophytosis is often self-limiting, and most horses recover spontaneously after several months. Despite this, treatment is recommended to hasten resolution of lesions, to decrease the severity of disease, and to minimize infection of other animals and people.

KEY POINT

The condition may be spread to other animals and humans, and therefore advice should be given to personnel handling affected horses to help avoid a zoonotic infection.

- The condition may be highly contagious, and it is important that action be taken to limit the spread via isolation and disinfection of the affected horse's grooming equipment, blankets, tack, and stalls. Disinfection can be attempted with sodium hypochlorite (Clorox bleach), lime sulfur, or formaldehyde solutions.
- Topical antifungal agents should be used on affected and exposed animals. Agents include iodine (1% available iodine), chlorhexidine (2% solution), miconazole (2% shampoo), sodium hypochlorite (Clorox bleach-5% solution), and lime sulfur (5% solution). Solutions are applied to the full body daily for 5 to 7 days, and then once or twice weekly until 2 weeks after clinical remission.
- Enilconazole (Imaverol) is an imidazole, approved for use in some countries, that has been shown to be very effective for treatment in both small and large animals. It can be used topically on animals once weekly and as an aerosol for environmental treatment.
- Oral griseofulvin has been advocated at dose rates of 10 mg/kg/day for 1 to 2 months. However, no pharmacokinetic studies have been done in the horse, and its use at this time is not recommended.

Bacterial Folliculitis and Furunculosis

Staphylococcus species, and occasionally *Corynebacterium* or *Streptococcus* species, can cause infections in the hair follicles of the skin. Infections are almost invariably secondary problems. They can be caused by many factors, especially those that disrupt the barrier function of the skin such as warm and humid weather or anything causing trauma to the skin (e.g., pruritus, wounds).

KEY POINT

In horses, bacterial folliculitis and furunculosis are uncommon compared with dermatophilosis and dermatophytosis.

HISTORY

- Predisposing factors are usually present, and disease is seen more often in spring or summer.

CLINICAL FINDINGS

- Primary lesions are follicular papules and pustules, which progress to crusts and alopecia. If hair follicles rupture, furunculosis is seen, with nodules, draining tracts, and large crusted areas.
- Lesions are commonly in the saddle/girth region, but lesions around the pastern may occur, which can progress to cause a typical appearance of "greasy heel."

DIAGNOSIS

- Cytologic examination of impression smears or tape preparations from affected areas should reveal increased numbers of bacteria and neutrophils.

KEY POINT

The presence of bacteria within inflammatory cells on cytologic examination is diagnostic of bacterial infection.

- Bacterial culture can also be performed to help confirm the diagnosis; however, samples must be obtained in a sterile manner from intact pustules or deep aspirates for a positive culture of an organism that is part of the normal skin flora to be significant.

DIFFERENTIAL DIAGNOSIS

- Dermatophilosis
- Dermatophytosis
- Pemphigus foliaceus
- Photosensitization (if only white areas affected)

TREATMENT

- Topical antibacterials, such as benzoyl peroxide or chlorhexidine shampoos, can be very effective for superficial lesions. Weekly or biweekly shampooing should be performed for at least 3 weeks and may be continued on a regular basis to help prevent reinfection.
- Systemic antibiotics should be used for deep, poorly responsive, or recurrent infections. Penicillin (Treatment Nos. 83 and 84) is often effective; however, treatment needs to continue for a minimum of 3 weeks, so oral potentiated sulfonamides (Treatment No. 108) are preferred.
- Bacterial culture and sensitivity testing is advised only with severe cases or those that are poorly responsive to treatment.

KEY POINT

A search for underlying disease or predisposing factors should be made with any recurrent or poorly responsive bacterial infection.

Keratinization Defects (Seborrhea)

These are disorders due to altered keratinization and are characterized by excess scale and crust formation, with increased greasiness of the skin *(seborrhea oleosa)* or without it *(seborrhea sicca).* There is usually minimal pruritus. Keratinization disorders are poorly understood in the horse but are mostly secondary to some other skin disease, and generalized forms are rare. Primary seborrhea occurs rarely. The term "primary seborrhea" refers not to excess production of sebum by sebaceous glands but to what is thought to be a keratinization defect in the epidermis or the follicular epithelium, whereby normal growth and differentiation of epithelial cells do not occur.

HISTORY

- Lesions are most commonly localized and include scale and crusts, with or without greasiness of the skin.
- There is often a history of prior skin disease, and many other skin lesions may be present related to the underlying disease.

CLINICAL FINDINGS

- There are some specific forms of keratinization disorders noted to occur in horses:
  - *Generalized scaling*—may be seen in horses kept indoors with heating in winter
  - *Primary generalized*—rare, usually symmetric and truncal, oily or dry
  - *Mane and tail*—most common form recognized: moderate to severe scaling in the mane and tail can be oily or dry; usually little or no inflammation, pruritus, or alopecia
  - *Linear keratosis*—seen mostly in Quarter horses, may be congenital and possibly inherited; vertical bands of hyperkeratosis and lichenification on the neck and/or thorax, usually unilateral
  - *Cannon keratosis*—possibly localized keratinization defect; focal areas of scaling, crusting, and alopecia on the anterior surface of the rear cannon bone area, with or without inflammation

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS

KEY POINT

A search for underlying disease or predisposing factors should be made with all cases of seborrhea in the horse.

- Diagnosis of primary (idiopathic) seborrhea can be made only by ruling out other potential causes and with consistent biopsy findings. A wide variety of conditions can cause secondary seborrhea, including allergies, ectoparasites, pemphigus foliaceus, bacterial folliculitis, dermatophytosis, and dermatophilosis.

TREATMENT

- Treatment of the underlying cause in horses with secondary seborrhea will often lead to gradual resolution of lesions. With all forms, topical therapy will often help.
- For treating seborrhea oleosa, tar and sulfur shampoos, which are strongly keratolytic, can be effective.
- For seborrhea sicca, tar and sulfur shampoos are very drying and may worsen the scale. Emollient shampoos or mild keratolytic sulfur-salicylic acid shampoos are preferred.

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- Antiseborrheic shampoos can be followed by humectants, which rehydrate the skin and reduce scale formation.

Alopecia Areata

This is an idiopathic noninflammatory skin disorder seen in horses, characterized by well-circumscribed patches of alopecia. It occurs in many species, including humans, but the etiology is poorly understood.

HISTORY

- Disease is seen rarely in individual horses.

CLINICAL FINDINGS

- There may be focal or multiple patches of alopecia clearly demarcated from surrounding haired areas. Lesions may occur on the face, neck, and trunk. Pruritus is absent.

DIAGNOSIS

- Biopsy is needed for diagnosis, and characteristic histopathologic changes usually are seen early in the disease. With progression of disease, biopsies may be nondiagnostic.

DIFFERENTIAL DIAGNOSIS

- Dermatophilosis
- Dermatophytosis
- Bacterial folliculitis
- Demodecosis
- Occult sarcoid
- Anagen defluxion

TREATMENT

- Hair often will regrow without treatment within months to years. Topical remedies, including glucocorticoid creams, and systemic glucocorticoids have been used for treatment in horses, other animals, and humans with this disease, but there is no evidence any treatment alters the natural course of disease.

Photosensitization

Photosensitization can produce an inflammatory skin disease due to exposure of skin to ultraviolet (UV) radiation.

KEY POINT

The key features are the presence of a photoactivating substance in the skin, exposure of the skin to UV light, and absorption of the UV light by the skin, which is greatly facilitated by lack of pigment and hair.

There are different forms of disease relating to the origin of the photosensitizing agent. In *primary photosensitization,* the photosensitizing agent enters the body preformed or is produced metabolically within the body. It can be injected, ingested (e.g., phenothiazine drugs), or absorbed directly via contact with the skin (e.g., photosensitizing plants).

Secondary photosensitization occurs in liver disease when there is accumulation of phylloerythrin in the blood. Phylloerythrin is a porphyrin compound formed in the intestines that is normally conjugated in the liver and excreted in bile. When blood levels become elevated, phylloerythrin begins to accumulate in the skin.

KEY POINT

The hepatic form of photosensitization is more common than the primary form and is usually seen with ingestion of plants containing pyrrolizidine alkaloids (see Liver Disease, Chapter 7).

Once the photoactivating agent is present in the skin, absorption of UV light by the skin results in the release and/or formation of oxygen free radicals, which damage cells and cause inflammation.

HISTORY

- White areas that are sparsely haired or nonhaired are affected. Pigmented areas remain normal, because UV light is poorly absorbed in these areas.

CLINICAL FINDINGS

- Erythema, edema, and vesicles that progress to erosions and ulcers with crusting and scaling.
- Lesions are usually painful and may affect all white areas or white areas on distal limbs only.
- Lesions can be very severe, and horses may be systemically unwell.
- Affected areas of skin may slough.

DIAGNOSIS

- Diagnosis is suspected by the localized but severe skin lesions. Skin biopsy reveals superficial dermal blood vessel degeneration and thrombosis along with perivascular inflammation.

- Blood samples should be collected for hematologic studies and liver function tests in all cases (see Chapter 7) to allow differentiation of primary from secondary photosensitization. A thorough investigation of diet, pasture, and drug administration must be made to determine the source of the photoactivating agent or to help determine the cause of the hepatopathy if present.

DIFFERENTIAL DIAGNOSIS

- Contact dermatitis (if distal extremities and/or muzzle affected)
- Equine sarcoidosis
- Dermatophilosis
- Bacterial folliculitis
- Pemphigus foliaceus
- Dermatophytosis

TREATMENT

- A thorough investigation to identify and ideally remove the source of the photoactivating agent is needed. Prognosis is poor with secondary photosensitization unless the hepatopathy can be treated successfully.

KEY POINT

Stabling the horse to prevent exposure to the sun is important to prevent worsening of the problem.

- Topical application of antibiotic/glucocorticoid ointments can be helpful in decreasing the local inflammation.

Equine Sarcoidosis

This is a poorly understood disorder in horses characterized by generalized seborrhea, severe wasting, and sarcoidal granulomatous inflammation of multiple organ systems. It closely resembles sarcoidosis in humans.

CLINICAL FINDINGS

- Scaling, crusting, and alopecia are found initially on the face or limbs then progress to become generalized.
- Nodules may also occur and are occasionally the sole feature.
- Horses are also systemically ill and may show weight loss, lymphadenomegaly, pyrexia, inappetence, and lethargy.

DIAGNOSIS

- Skin biopsy is needed for diagnosis, revealing perifollicular and middermal sarcoidal granulomas, with many multinucleated giant cells and histiocytes. Similar granulomas may be present in multiple other organs.

DIFFERENTIAL DIAGNOSIS

- Exfoliative eosinophilic dermatitis and stomatitis
- Pemphigus foliaceus
- Primary seborrhea
- Systemic lupus erythematosus
- Dermatophilosis
- Dermatophytosis

TREATMENT

- Most horses develop progressive wasting and dermatitis, and the prognosis is guarded. Spontaneous remission rarely occurs. High-dose glucocorticoid therapy is sometimes successful.

Pemphigus Foliaceus

KEY POINT

Pemphigus foliaceus is a rare autoimmune skin disorder characterized by widespread vesicles, pustules, and crusts.

It occurs because of the production of antibodies directed against keratinocytes, resulting in loss of adhesion between cells.

HISTORY

- Generalized skin disease characterized by pustules and/or crusting occurs.
- Disease can occur in adult horses and also in foals, typically less than 1 year of age.
- Disease may wax and wane. Systemic illness (signs of depression, lethargy, and inappetence) occurs concurrently in greater than 50% of affected horses.
- Appaloosas may be predisposed.

CLINICAL FINDINGS

- Disease may commence with vesicles and pustules, which rapidly progress to crusting. There is variable scaling, alopecia, and erosions.
- Lesions may begin on the head, neck, and forelimbs but often quickly become generalized. Lesions may be restricted to the coronary bands occasionally.
- Pruritus and pain are variable.

DIAGNOSIS

- Cytologic examination of needle aspirates from intact vesicles or pustules may reveal numerous

acantholytic cells (rounded-up nucleated keratinocytes that have lost their intercellular junctions) and nondegenerate neutrophils with the absence of bacteria, which is strongly suggestive of the disease.
- Skin biopsy is needed to confirm the diagnosis; however, samples must be chosen carefully. Intact vesicles or pustules are ideal but may be difficult to find, and if possible, animals should be hospitalized to allow regular searching for these transient lesions. Crusted lesions are the next best lesions and may be diagnostic; however, it is vital that the crusts be left in place and included in the sections. Secondary bacterial infection can occur concurrently, which makes diagnosis more difficult and ideally should be addressed before biopsy.

DIFFERENTIAL DIAGNOSIS

- Dermatophytosis
- Dermatophilosis
- Bacterial folliculitis
- Exfoliative eosinophilic dermatitis and stomatitis
- Equine sarcoidosis

TREATMENT

KEY POINT

Therapy for adult-onset pemphigus foliaceus is difficult, requiring high doses of glucocorticoids and/or other immunosuppressive drugs and good owner compliance.

- Owners need to be committed to treatment, because therapy frequently needs to be adjusted and individualized to each patient, especially until the disease is brought under control. If treatment is successful in controlling the disease, therapy needs to be continued for life in most cases, and intermittent recurrence of symptoms can occur.

KEY POINT

In contrast to the disease in adult horses, pemphigus foliaceus in foals has a fairly good prognosis and responds well to treatment in most cases. Disease will sometimes resolve spontaneously.

- Oral prednisolone (Treatment No. 93) is the initial treatment of choice because it is inexpensive and there is generally a good response. Initially, a dose of 1 to 2 mg/kg twice daily should be used until lesions are regressing well, which typically occurs after 7 to 14 days. Dosing frequency is then decreased gradually, and once the disease is in remission, alternate-day treatment is used at the lowest dose that keeps the disease controlled.
- Sometimes initial treatment with dexamethasone (Treatment Nos. 29 and 30) is superior to prednisolone for inducing remission, and sometimes drugs such as injectable gold salts (aurothioglucose) need to be used with or instead of prednisolone to achieve good control or when side effects of glucocorticoid therapy (e.g., laminitis) restrict their use.

Equine Exfoliative Eosinophilic Dermatitis and Stomatitis

This disease is thought to be the result of a hypersensitivity reaction, but the initiating cause is not known. There is severe eosinophilic infiltration and granulomatous inflammation of affected areas.

HISTORY

- Disease more commonly begins in winter, and systemic signs of illness often occur. It is mostly found in Standardbreds and Thoroughbreds.

CLINICAL FINDINGS

- Scaling and crusting followed by alopecia, ulceration, and fissures are seen.
- Lesions usually commence on the face or coronary bands and progress to become generalized. Oral ulcers can also occur. As lesions progress, horses become systemically ill.

KEY POINT

In conjunction with skin lesions, weight loss often is found, despite good appetite. Animals may show signs of depression and may have diarrhea owing to colonic involvement.

DIAGNOSIS

- Skin biopsy is essential to make a diagnosis, and specimens reveal eosinophilic and lymphoplasmacytic dermatitis. Eosinophilic granulomatous inflammation may be present in other organs, including the gastrointestinal tract, liver, pancreas, salivary glands, and lungs.
- D-Glucose or D-xylose absorption testing usually shows flattened curves, indicating malabsorption.
- Rectal mucosal biopsy (for details, see Chapter

7) may be useful to aid diagnosis because eosinophilic infiltrates often are present.

DIFFERENTIAL DIAGNOSIS

- Dermatophilosis
- Dermatophytosis
- Pemphigus foliaceus
- Primary seborrhea
- Sarcoidosis
- Systemic lupus erythematosus

TREATMENT

KEY POINT

Most horses do not recover from this disorder, and the prognosis is grave.

- Some horses have been helped initially by glucocorticoid therapy. Dexamethasone disodium phosphate (Treatment No. 30) given initially intravenously for 2 to 3 days followed by oral prednisolone (Treatment No. 93) has been used. It will not cure the condition but may provide short-term relief.

NODULAR SKIN DISEASE WITH ULCERATION AND/OR EXUDATION

Nodular skin diseases have been divided into those frequently seen with ulceration and/or draining tracts and those without. There can be some overlap of clinical findings in most diseases, which is discussed where appropriate.

Exuberant Granulation Tissue ("Proud Flesh")

KEY POINT

Exuberant granulation tissue is a very common problem in horses. It is found most commonly on the distal limb in association with wounds in which poor skin mobility and frequent movement result in limited wound contraction. The granulation tissue bed proliferates because of a lack of epithelial cover.

HISTORY

- There should be a history of a previous skin wound that has not healed.

CLINICAL FINDINGS

- Exuberant granulation tissue appears as a proliferation of hemorrhagic granulation tissue. The surface may show evidence of infection and/or focal necrosis or may appear pink and healthy.
- No local irritation, pruritus, or pain is evident.

DIAGNOSIS

- Diagnosis is usually evident from history and clinical examination. However, if there is some doubt about the diagnosis or if the granulation tissue appears to be slightly different in color from the pink healthy appearance expected, a biopsy may be required to establish the diagnosis.
- If there is a chronic wound with excessive granulation tissue and an intermittently discharging sinus, radiographs should be taken because osteomyelitis can be found concurrently.

DIFFERENTIAL DIAGNOSIS

- Sarcoid
- Papillomatosis
- Habronemiasis
- Pythiosis
- Bacterial and/or fungal granulomas
- Squamous cell carcinoma

TREATMENT

- Wound contraction and epithelialization cannot proceed with the granulation tissue bed raised above the skin surface.

KEY POINT

Treatment first involves excising the excessive granulation tissue down to just below skin level.

- Because there are no sensory nerves in the granulation tissue bed, excision may be done without a local anesthetic. Although hemorrhage is often quite dramatic immediately after excision, it can be controlled easily with pressure bandaging.
- Once the excess granulation tissue is removed, the wound surface must be kept clean and free from infection to encourage rapid healing.
- Historically, various caustic substances, including copper sulfate, nitric acid, acetic/malic acid mixtures, and other preparations, have been used topically on the wounds to inhibit granulation tissue.

KEY POINT

These topical astringents may inhibit formation of granulation tissue, but they also discourage wound healing, and rapid wound

healing is the most important factor in preventing the recurrence of exuberant granulation tissue. Thus, the use of topical astringents is not advised.

- It is best to keep the wound firmly bandaged, which helps to prevent exuberant granulation tissue from forming. Recently, many new bandaging materials have become available for use, and there is much interest in the veterinary field in materials used in human medicine to encourage wound healing. Some preliminary studies have been performed in dogs, and products incorporating polymeric foams, hydrogels, and hydrocolloids appear to enhance wound healing compared with standard veterinary bandaging products that have a passive protective role only. Adherent dressings (e.g., wet-to-dry) are indicated only if necrotic tissue is present and debridement is still needed. Otherwise, dressings need to be nonadherent. Healing may be slow but if progressing can be allowed to continue. If healing fails to progress, skin grafting should be considered.

Habronemiasis ("Summer Sores")

Habronemiasis is caused by the larvae of nematode species, particularly *Habronema muscae* and *Habronema majus.* The adult nematodes live in the stomach of the horse and produce eggs and larvae that are passed in feces. The larvae are then ingested by maggots of the intermediate hosts (houseflies and stableflies) and can then be deposited on the skin, on open wounds, or on chronically wet areas. The lesions are thought to be the result of a hypersensitivity reaction to the larvae.

HISTORY

KEY POINT

Habronemiasis is a common cause of ulcerative granulomatous skin disease in the horse. It occurs particularly in warmer wet climates throughout the world, and it affects moist regions of the body or open wounds.

- Some horses have recurrent disease each summer, and there may be partial or complete remission in winter.

CLINICAL FINDINGS

- Single or multiple ulcerated nodules are seen that have a hemorrhagic or serosanguineous discharge.
- Exuberant granulation tissue is often present, and there may be small, yellow, nonbranching granules (which are composed of necrotic debris surrounding the larvae).
- Pruritus can be mild to severe.

KEY POINT

Lesions occur most commonly on the legs, ventrum, and prepuce; in the urethral process of the penis and medial canthus of the eye; and on wounds.

- Conjunctival habronemiasis is common, and there are usually yellowish gritty plaques on both palpebral and conjunctival surfaces. In severe cases, the eye will be painful, with blepharitis, photophobia, chemosis, and epiphora.

DIAGNOSIS

- Cytologic examination of deep scrapings or exudate may reveal larvae. Biopsy is advised to confirm the diagnosis regardless of cytologic findings, because *Habronema* larvae may invade the lesions of other ulcerative dermatoses, including sarcoids, squamous cell carcinoma, and infectious granulomas.
- Biopsies reveal a nodular to diffuse granulomatous dermatitis with large numbers of eosinophils and mast cells. Foci of coagulation necrosis, which may contain nematode larvae, are characteristically found.

DIFFERENTIAL DIAGNOSIS

- Exuberant granulation tissue
- Sarcoid
- Bacterial or fungal granuloma
- Squamous cell carcinoma (particularly for third eyelid lesions)

TREATMENT

- Organophosphates and ivermectin have been used systemically and topically in the past to treat habronemiasis, but because the disease results from a hypersensitivity reaction to the larvae, recent treatments have been aimed at decreasing the inflammatory reaction rather than at killing the larvae. Systemic glucocorticoids have been found to be effective as the sole systemic treatment. Excess granulation tissue should be surgically removed.
- Oral prednisolone is the least expensive and most effective therapy and is recommended at a dose of 1 mg/kg given once or twice daily for the first 10 to 14 days and then tapered.

KEY POINT

An important part of the treatment is to prevent reinfestation of the wound during healing.

- Organophosphate and anti-inflammatory pastes are applied topically to the wound daily until healed. The wound also can be bandaged. Fly control in the environment and on the horse and regular anthelmintic use to kill the adult worms in the stomach should be used to help prevent reinfestation.

Sarcoids

Sarcoids are unique to the horse and suspected to have a viral etiology (most likely a papilloma virus closely related to bovine papilloma virus).

KEY POINT

Sarcoids are the most common tumor affecting the skin of horses. They can be single or multiple, vary in appearance, and are most common on the head (around the eyes, lips, and ears) and on the legs or ventral trunk.

HISTORY

- Sarcoids are more common in horses less than 4 years old and tend to be familial. There may be a history of a previous wound at the site.

CLINICAL FINDINGS

KEY POINT

There are four basic types of sarcoids that are distinctly different in appearance: verrucous (wart-like), fibroblastic, mixed verrucous and fibroblastic, and occult (flat).

- Fibroblastic sarcoids have an ulcerated exudative surface and appear similar to exuberant granulation tissue. Verrucous forms may also have a similar surface but are more nodular and wart-like, and a combination of these forms also occurs. These forms can be very aggressive locally but do not metastasize. Some undergo spontaneous remission after a number of years.
- Occult sarcoids are flat annular areas characterized by alopecia, scaling, and sometimes crusting and are thus distinctly different in appearance from the nodular forms of sarcoids.

DIAGNOSIS

KEY POINT

Biopsy and/or excision for diagnosis is important. However, it is not recommended to biopsy occult or small verrucous forms if other diagnoses are very unlikely. This is because sudden increased growth and aggressive behavior can occur after surgical intervention.

- Histopathologic findings depend on the type of tumor present, with variable combinations of fibroblastic proliferation and epidermal hyperplasia. Occult forms may show focal epidermal hyperplasia and fibroblast proliferation at the dermoepidermal junction.
- Cytologic examination of impression smears, tape preparations, or fine-needle aspiration samples collected from lesions can be performed as with any lesion. Samples may reveal inflammation and should have no evidence of bacteria, fungi, other neoplastic cells, or other changes that would be more consistent with other diagnoses.

DIFFERENTIAL DIAGNOSIS

- Depends on the form of the sarcoid:
  - *Fibroblastic*
    - Squamous cell carcinoma
    - Exuberant granulation tissue
    - Habronemiasis
    - Bacterial granulomas
    - Pythiosis
  - *Verrucous*
    - Papillomatosis
    - Early squamous cell carcinoma
  - *Occult*
    - Dermatophytosis
    - Dermatophilosis
    - Alopecia areata
    - Focal onchocerciasis

TREATMENT

KEY POINT

Sarcoids can be very persistent and invasive tumors, and although surgical removal with adequate margins is the treatment of choice, this often is not possible owing to the size and location of the tumors. Recurrence after surgical excision is very common.

- Occult and small verrucous forms are best left alone and monitored.
- A number of alternative or adjunctive treatments to surgery have been used to help decrease recurrence rates. Some are still being evaluated, and some require expensive equipment and are not readily available. Treatments include cryotherapy, immunotherapy (using mycobacterial cell-wall products), radiofrequency hypothermia, carbon dioxide laser surgery, local radiation therapy, and topical therapies (5-fluorouracil [5-

FU, 5%], podophyllin in alcohol). Intratumoral injections of cisplatin used as a suspension in purified sesame oil (1 mg/cm^3 of tumor), repeated at 2-week intervals until tumor regression also appears to be safe and effective. If possible, surgical debulking is combined with most therapies.

KEY POINT

Cryotherapy is one of the most frequently used treatments, either as a sole therapy or in combination with surgical debulking. Recurrence rates are estimated at approximately 40%, compared with recurrence rates of 50 to 64% with surgery alone.

Squamous Cell Carcinoma

KEY POINT

Squamous cell carcinoma is a very common skin tumor in the horse. The head, mucocutaneous junctions, and genitalia are most commonly affected.

Tumors are very aggressive locally and can also metastasize. Occasionally, horses present because of symptoms related to metastases before those related to the primary tumors.

HISTORY

- Erosive lesions are commonly seen at mucocutaneous junctions. Older horses are most frequently affected, and the lesions can progress rapidly.

CLINICAL FINDINGS

- Lesions are usually solitary. They can begin as small verrucous nodules (especially around the genitalia) or as nonhealing and enlarging ulcerated masses. Necrosis and odor are common.

DIAGNOSIS

- Biopsy is critical to confirm the diagnosis. Cytologic examination of impression smears or fine-needle aspirates can be highly suggestive of squamous cell carcinoma and can be used to guide surgical approach before histopathologic confirmation.

DIFFERENTIAL DIAGNOSIS

- Sarcoid (verrucous or fibroblastic)
- Exuberant granulation tissue
- Papillomatosis
- Pythiosis
- Bacterial granulomas

TREATMENT

KEY POINT

Early diagnosis and wide surgical excision are important for successful outcome.

- Other treatments used with some success include cryotherapy, radiofrequency hyperthermia, and radiation therapy (including gold-198, cobalt-60, strontium-90, and radon-222 implants).

Pythiosis

Pythiosis is caused by a fungus-like organism, *Pythium insidiosum,* and is characterized by invasive, ulcerative, proliferative lesions that usually occur on the legs or ventrum.

HISTORY

KEY POINT

Disease occurs only when there is prolonged contact with water in which organisms are living and frequently arises at the site of previous wounds. It is seen in wet tropical to subtropical areas where horses have prolonged access to stagnant water.

CLINICAL FINDINGS

- Large, proliferative, ulcerative lesions are characteristic and are usually associated with a previous wound. There are often discharging sinuses from the surface of the granulomatous tissue, and irregular, gritty, yellow to gray masses ("kunkers") commonly lie within the sinus tracts.
- Most lesions are single, intensely pruritic, and rapidly progressive.
- Lymphangitis or at least limb edema is frequently seen with larger lesions. Internal spread of disease occurs rarely.

DIAGNOSIS

- Cytologic examination of necrotic tissue from lesions may reveal hyphae.

KEY POINT

Biopsy reveals fungal hyphae within areas of necrotic eosinophils and neutrophils, surrounded by granulomatous to pyogranulomatous inflammation.

- For absolute confirmation of pythiosis, fungal culture is performed. The organism grows readily on standard media. Kunkers are a good source of fungi for culture.

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DIFFERENTIAL DIAGNOSIS

- Sarcoid
- Squamous cell carcinoma
- Exuberant granulation tissue
- Habronemiasis
- Bacterial granulomas

TREATMENT

KEY POINT

Surgery to remove all infected tissue is curative and is the treatment of choice. There should be radical trimming of excess tissue down to below the skin surface. Recurrences are common, and repeated surgeries may be required.

- Lesions can enlarge very rapidly; therefore, surgery should be performed immediately to remove infected tissue if pythiosis is suspected, without waiting for laboratory confirmation of infection.
- Immunotherapy has been used as an adjunct to surgery for treating pythiosis. Two vaccines have been developed, both prepared from *Pythium* cultures, and have been used with good success. Painful reactions and sterile abscesses at injection sites are common.
- Systemic amphotericin B has also been used successfully for treatment in conjunction with surgery, but nephrotoxicity and anemia can occur.

Sporotrichosis

This is a fungal infection caused by the dimorphic fungus *Sporothrix schenckii,* which is normally found in the environment in soil and vegetation worldwide. It usually infects horses via wound contamination, especially wounds from thorns or splinters.

CLINICAL FINDINGS

- Skin lesions usually consist of chains of hard subcutaneous nodules that develop along lymphatic tracts, most commonly on the medial aspects of limbs. Nodules enlarge and may ulcerate and discharge a small amount of thick brown-red exudate that forms a surface crust.

DIAGNOSIS

- Organisms may be seen on cytologic examination of exudates or aspirates from lesions, and the organism can be readily cultured to confirm the diagnosis.

KEY POINT

This is a zoonotic disease, and great care should be taken in handling infected animals and samples collected for diagnostic testing if sporotrichosis is possible.

DIFFERENTIAL DIAGNOSIS

- Histoplasmosis
- Mycetomas
- Bacterial granulomas
- Pythiosis
- Ulcerative lymphangitis

TREATMENT

- Sodium iodide is most often recommended; it is given intravenously for 2 to 5 days (20% solution given at a dose rate of 20-40 mg/kg/day), followed by oral and topical iodides.
- Treatment usually is effective.

Ulcerative Lymphangitis

This is a relatively uncommon disease in the horse caused by bacterial infection of the cutaneous lymphatics. *Corynebacterium pseudotuberculosis* is the most common causal bacterium.

HISTORY

- Disease is associated with a history of wounds and poor hygiene.

CLINICAL FINDINGS

KEY POINT

Early in the course of the infection there is limb edema followed by development of multiple, painful, firm to fluctuant nodules that ulcerate and develop draining tracts.

- The discharge is purulent and often creamy to green in color.
- Most cases seem to involve a hindlimb, and edema and swelling often involve the entire hindlimb.

DIAGNOSIS

- Cytologic examination and bacterial culture of exudates and/or fine-needle aspirates from nondraining nodules will allow diagnosis.

DIFFERENTIAL DIAGNOSIS

- Sporotrichosis
- Histoplasmosis

- Mycetomas
- Bacterial granulomas
- Pythiosis

TREATMENT

KEY POINT

Aggressive treatment early in the course of the disease is indicated to prevent fibrosis and disfigurement and to give the best chance of recovery.

- Exercise, hydrotherapy, surgical drainage, and high doses of procaine penicillin (20,000-40,000 IU/kg q12h) given for prolonged periods (up to 6 months) may be effective. Poor local environmental conditions must be improved. Clean bedding must be provided and the feet must be cleaned daily.
- Discharges can be infectious to other animals, including people, and should be disposed of carefully.

NODULAR SKIN DISEASE WITHOUT ULCERATION OR EXUDATION

These diseases are characterized by intact nodules. However, ulceration of nodules can occur with aggressive or chronic disease.

Melanoma

Melanomas are benign or malignant tumors arising from melanocytes or melanoblasts.

HISTORY

- Melanomas are common in adult to aged horses. They may be solitary or multiple, located within the skin or subcutis, and most commonly occur on the perineum, ventral surface of the tail, periocular region, and distal limbs.
- About 75% of tumors are malignant.
- They are common in Arabians and Percherons.

KEY POINT

Melanomas are almost exclusively seen in those horses that turn dappled gray and then white as they age. Horses' original coat color varies, and as they age the hair turns gray and the skin remains pigmented.

CLINICAL FINDINGS

- Melanomas are usually firm and nodular and may be alopecic, ulcerated, and hyperpigmented.
- Three growth patterns have been described for melanomas in horses. Some display slow growth for years without metastasis, some grow slowly for years and then suddenly grow rapidly and metastasize, and some display rapid growth and malignancy from the beginning.

DIAGNOSIS

- Melanomas can be confirmed by biopsy and histopathology. Cytologic features can be highly suggestive.

DIFFERENTIAL DIAGNOSIS

- Other forms of neoplasia—mast cell tumor, fibroma, basal cell tumor
- Nodular necrobiosis

TREATMENT

- Surgical excision is the treatment of choice, especially with solitary lesions. However, most cases are multiple, and excision can be difficult, particularly when tumors are located on the perineum.
- Cryotherapy and oral cimetidine therapy (2.5-4.0 mg/kg q8h for 2 months) have been used with variable results where surgical treatment alone is unsatisfactory or unsuitable.

Papillomatosis (Warts)

Papovaviruses are known to cause warts in a variety of species, including horses.

HISTORY

- Horses less than 3 years old are most frequently affected.

CLINICAL FINDINGS

- Characteristically, papillomas have a verrucous appearance, but occasionally aural plaques occur.
- Lesions are usually multiple and most common around the lips and nose. They may also occur bilaterally on the medial surface of the pinnae, in which case they range in appearance from small, smooth, depigmented papules and plaques to larger, often coalescent hyperkeratotic plaques.

DIAGNOSIS

- The typical appearance of the lesions strongly suggests the diagnosis in most cases; however, biopsies can be taken for confirmation.

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DIFFERENTIAL DIAGNOSIS

- The verrucous lesions are typical and not confused with other skin conditions. The aural plaque lesions may be confused with insect bites, but these are usually on the outer part of the ear.

TREATMENT

- Papillomatosis is a self-limiting disease, and spontaneous regression of lesions occurs with time. Because the lesions are unsightly and occur in young animals that may be for sale, a number of treatments have been used with questionable success to hasten regression. These include topical podophyllin, topical dimethyl sulfoxide, and autogenous vaccines.

KEY POINT

Although lesions are best left untreated, it is important to isolate infected horses so that transmission is less likely.

Nodular Necrobiosis ("Collagenolytic Granuloma," "Equine Eosinophilic Granuloma")

Nodular necrobiosis is probably the most common nodular skin disease of the horse. It is possibly related to a hypersensitivity to insect bites, but the disorder is poorly understood.

HISTORY

- Disease usually appears in summer.

CLINICAL FINDINGS

- Single or multiple nodules of variable size are seen that are firm and round with no ulceration or alopecia. Lesions occur most commonly on the withers and back (particularly where the saddle sits) and can also be seen on the girth area, mane, rump, and face.

KEY POINT

Horses show complete lack of pain or pruritus associated with the lesions.

DIAGNOSIS

- Skin biopsies reveal granuloma formation with eosinophil infiltration surrounding areas of collagen degeneration. Cytologic examination usually reveals many eosinophils, lymphocytes, and histiocytes.

DIFFERENTIAL DIAGNOSIS

- Neoplasia—mast cell tumor, fibroma, melanoma, basal cell tumor
- Habronemiasis
- Amyloidosis
- Foreign-body granuloma
- Epidermoid and dermoid cysts

TREATMENT

- Treatment options include surgical excision, intralesional glucocorticoids, and systemic glucocorticoids. Multiple lesions are often best treated with systemic glucocorticoids—prednisolone can be used at a dose of 1 mg/kg once daily for 10 to 14 days, with gradual reduction in dose over 4 weeks. Single or only a few nodules can be treated with local injections of long-acting glucocorticoids such as methylprednisolone acetate (20 mg per lesion; Treatment No. 74) or triamcinolone acetate (3-5 mg per lesion; Treatment No. 107). Relapses after treatment can occur; however, these can often be successfully retreated.
- Insecticides and repellents, applied daily or twice weekly, have lead to remission in some horses.
- Larger calcified lesions do not respond to steroid treatment and may require surgical excision. However, some care is required, particularly where there are lesions in the dorsal midline, because nonhealing wounds may result.

Axillary Nodular Necrosis

This disease is very similar to nodular necrobiosis, but on histopathologic examination of biopsy samples there is no collagen degeneration seen. One or two nodules appear in the girth area behind the axillae.

Unilateral Papular Dermatosis

This is an uncommon poorly understood disease with some similarities to nodular necrobiosis characterized by multiple papules and nodules limited to one side of the body. Lesions are often on the trunk and occasionally spread to the shoulders, neck, and abdomen. There are similarities to herpes zoster in humans, and thus a viral etiology with peripheral nerve involvement has been proposed.

Nodules and papules are firm and well circum-

scribed, ranging from 5 to 15 mm in diameter, and asymptomatic. Lesions are characterized by eosinophilic folliculitis and furunculosis histologically and will regress spontaneously over weeks to months.

Amyloidosis

This is a rare nodular skin disease of horses, characterized by organized deposits of amyloid within the dermis. Amyloid can also be deposited in the mucosa of the upper respiratory tract. The etiology is unknown.

Multiple papules, nodules, and plaques of varied size occur, most commonly on the head, neck, and pectoral areas. They can appear quite suddenly, in which case they may resemble urticaria, or they can appear gradually. Respiratory signs may occur concurrently. Skin biopsy confirms the diagnosis. No treatment is effective, and the disease usually has a prolonged progressive course.

Urticaria

Urticarial reactions are common in the horse and represent a clinical syndrome that can have a wide variety of causes. Immunologic and nonimmunologic mechanisms can be involved, both of which induce mast cell degranulation within the skin. Drugs are perhaps the most commonly identified cause. Hypersensitivities (insect, food, and atopy) have been associated with urticaria, as have intestinal parasitic infestations and infectious diseases (bacterial, fungal, viral). Other causes, such as physical (heat, cold, exercise) and psychological factors (anxiety or anticipation of racing), also may occur in horses. Urticaria can also result from contact with externally applied products (shampoos, insecticides).

HISTORY

- Sudden onset of skin wheals is characteristic. Episodes may be acute, chronic, or recurrent.

CLINICAL FINDINGS

KEY POINT

Multiple wheals are seen with an urticarial reaction. Affected areas can vary in size from small localized lesions (1-3 cm) to extensive generalized ones involving large areas of skin.

- Wheals pit with digital pressure and are usually soft but occasionally quite firm. Wheals may be associated with subcutaneous edema and serum leakage through the epidermis, but overlying skin is normal.

DIAGNOSIS

- Diagnosis of urticaria is mostly evident from the history and clinical findings; however, biopsy can be used for confirmation if wheals are firmer than expected or lesions are chronic (greater than 2 months' duration). With chronic lesions, marking the outline of several lesions with a waterproof marking pen and rechecking after 24 to 48 hours can be helpful. Urticarial lesions will have regressed, even if more have appeared in different locations, whereas masses will still be present.
- Establishing the cause of the urticaria is more difficult. A thorough history needs to be obtained. All medications must be stopped. Most lesions of acute urticaria disappear within 1 to 2 days.
- With recurrent or chronic urticaria, if there is no history of drug use, no response to withdrawal of drugs or topical products, and no history of related infections, an investigation for allergies is indicated. An insect control trial, elimination diet, and/or intradermal skin testing can be performed.

DIFFERENTIAL DIAGNOSIS

- Bacterial folliculitis
- Erythema multiforme
- Mast cell tumor
- Amyloidosis
- Early dermatophytosis—before development of alopecia

TREATMENT

- Removal/avoidance of suspected causal factors is obviously important if possible. Acute cases can often be treated successfully with glucocorticoids. Dexamethasone (0.1 mg/kg IV or IM) is sometimes reported to be more effective than prednisolone (1 mg/kg TV or IM).
- Idiopathic cases can be treated with antihistamines. Hydroxyzine hydrochloride seems to be particularly effective and is used at a dose rate of 1 to 2 mg/kg twice daily during bouts of disease.

MISCELLANEOUS SKIN DISEASES

Erythema Multiforme

This disease is an uncommon, acute, self-limiting disease of the skin and/or mucous membranes with classic lesions. It is an immunologic reaction

with several possible triggering factors, which include infections, drugs, and neoplasia.

HISTORY AND CLINICAL FINDINGS

- The clinical appearance is variable, but usually lesions are symmetric and often involve the trunk and limbs.
- Lesions seen include macules, papules, urticarial plaques, or vesicobullous lesions. Target lesions may form owing to the peripheral expansion of lesions.
- Individual lesions may persist for several days.
- Pruritus and pain are usually absent.

DIAGNOSIS

- Diagnosis is based on skin biopsy with history and clinical signs. Distinctive histopathologic findings are seen, with keratinocyte death (apoptosis) a feature.

DIFFERENTIAL DIAGNOSIS

- Urticaria
- Amyloidosis
- Mast cell tumor
- Vesicular diseases

TREATMENT

- The disease usually runs a self-limiting course, resolving over 1 to 3 months. Frequently, the disease is asymptomatic, so treatment is not needed in most cases. Investigation for triggering factors is advised, especially with recurrent or severe cases.

Drug Reaction

Cutaneous drug eruptions are hypersensitivity reactions that can occur after drug administration, resulting in a vast array of skin lesions that can mimic those of virtually any other skin disease. Drugs commonly causing cutaneous drug reactions include numerous antibiotics, anti-inflammatory drugs, vaccines, glucocorticoids, tranquilizers, and topical parasiticides.

HISTORY

- There is a history of recent or current drug administration.

CLINICAL FINDINGS

- Clinical findings are highly variable. A wide variety of skin lesions can be seen.

DIAGNOSIS

- Diagnosis can be based only on resolution of symptoms within 10 to 14 days after withdrawal of drug administration. Histopathologic changes in the skin vary as much as the clinical lesions. Recurrence of lesions on rechallenge with the drug is diagnostic; however, this is not without some risk of more severe reaction in the patient. Purposeful rechallenge is generally not advised.

DIFFERENTIAL DIAGNOSIS

- Any skin disease

TREATMENT

- Topical or systemic glucocorticoids can be used, but most drug eruptions are not responsive to this therapy. The reaction abates after withdrawal of the drug, but this may take several weeks.

TRAUMATIC SKIN DISEASE

Skin Wounds

One of the most common reasons that clients present their horses to the veterinarian is trauma resulting in wounds to the skin. The most common wounds are those involving the distal limb and are the result of lacerations due to barbed wire, gates, or fences. Wounds to the distal limb of horses are a particular problem because of the poor wound contraction in this region. Wound contraction is enhanced by myofibroblasts at the skin edges, which result in the wound margins' being moved inward. The minimal wound contraction possible in the distal limb is largely the result of poor skin mobility, in contrast with areas such as the chest and neck, where there is substantial loose skin. Poor healing in these sites can also be related to frequent movement, relatively poor blood supply, and a lower tissue temperature of the distal limb. Delayed wound healing in the distal limb can allow the formation of excessive granulation tissue, which is probably the most common complication of wounds to the distal limbs of horses. It is important to address all wounds to the lower limbs thoroughly and rapidly to encourage early healing and to minimize the risk of forming exuberant granulation tissue.

HISTORY

- It is important to determine the time since the wound occurred to allow decisions to be made

about whether the wound should be sutured or not.

CLINICAL FINDINGS

- The extent of the wound, its depth, and the amount of soft tissue damage must be determined, particularly if it is a puncture wound. With wounds on the distal limb, inspection of the depth of the wound for any damage to the periosteum is important. Periosteal damage can lead to subsequent sequestrum formation with a discharging sinus.

KEY POINT

Penetrating wounds should be evaluated carefully, especially those adjacent to joints. It is important to identify joint involvement and to minimize joint movement during healing.

- It is also important to determine the extent of gross contamination of the wound with foreign matter.

TREATMENT

KEY POINT

The major decisions in wound treatment are how much of the wound should be debrided and should the wound be sutured.

- All obviously necrotic tissue should be debrided. Areas of suspect viability may be observed over the next few days before a decision on debridement is made.

KEY POINT

Wounds need to be thoroughly cleaned. Saline irrigation is mostly thought to be the method of choice. Copious irrigation will be needed for heavily contaminated wounds.

- Povidone-iodine or chlorexidine solutions can be used, but even mild solutions irritate tissue. A large number of simple irrigation devices exist to help remove debris and contamination. High-pressure lavage can be of great value in helping to remove gross contaminants before suturing, but recently its usefulness for reducing bacterial contamination has been questioned.

KEY POINT

The use of local antibiotic solutions, ointments, and powders will cause irritation to wounds and is best avoided.

- In areas such as the chest and neck, even extensive wounds may not require suturing because of the excellent wound contraction. This usually results in a good cosmetic appearance with minimal wound care within a period of 2 to 3 weeks.
- Distal limb wounds are much more of a problem, and poor wound healing and poor cosmetic results of healing are frequent. Bandaging with frequent changes until the wound is healing well is advised in most cases, and casting can be considered for some wounds.

KEY POINT

Tetanus prophylaxis is crucial in any horse that has a wound.

- Antibiotics should be used if there is evidence of infection on bacterial culture and/or cytologic examination and may also be indicated with deep puncture wounds.

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CHAPTER 14

Neurology

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Neurologic diseases are common in horses and present significant diagnostic and therapeutic challenges. Size and temperament of the horse and the environment in which the horse lives frequently limit examination. The neuroanatomic and neuroendocrine pathways involved in dysfunction of the nervous system are extremely complex, and few signs are pathognomonic of a particular neurologic disease. In addition, different diseases can present with very similar signs, whereas certain specific neurologic diseases can be highly variable in their presentation. Despite these difficulties, the clinician need not be overwhelmed when dealing with a horse with suspected neurologic disease. If an ordered and logical approach to the examination is undertaken, an anatomic location of the lesion(s) and usually a diagnosis of the disorder can be obtained. Unfortunately, omission of parts of the examination frequently occurs and may lead to a failure to identify abnormalities, with resultant inaccuracies in prognosis.

NEUROLOGIC EXAMINATION

The goals for the clinician in cases of suspected neurologic disease are to establish

- Whether or not a neurologic problem is present;
- Whether the problem primarily involves the nervous system and to subsequently determine the anatomic location of the problem;
- Whether the problem secondarily involves the nervous system.

Essential to deciding if a neurologic problem exists is obtaining a detailed signalment (i.e., breed, age, sex, and use) and history and to perform a complete physical examination. Physical examination also can aid in detection of primary diseases of the musculoskeletal, cardiovascular, endocrine, or hepatic systems that can mimic neurologic disease (e.g., rhabdomyolysis) or result in secondary neurologic signs (e.g., hepatoencephalopathy). Apart from assessment of vital signs, particular emphasis should be placed on observation of the horse's mental attitude, behavior, posture at rest, and gait when walked. Also important are performing basic ocular tests (e.g., menace response, pupillary light response); evaluating bony and soft tissue symmetry of the head, neck, trunk, and limbs; and performing an external thoracolaryngeal ("slap") test.

KEY POINT

In general, neuronal dysfunction can be caused by direct effects of disease on neurons or by effects of disease on supporting elements or blood vessels.

Horses with primary or secondary neurologic diseases present usually with multiple behavioral, mentation, strength, or gait problems. If there is evidence of these signs, then a more detailed neurologic examination should be conducted. The main purpose of the neurologic examination is to localize the problem to a major anatomic region of the neuromuscular system, either in the brain (i.e., cerebrum, cerebellum, brainstem, and peripheral cranial nerves), spinal cord, peripheral spinal nerves, or muscles.

KEY POINT

If it is not possible to account for all neurologic signs with a single lesion, the presence of multifocal disease or multiple diseases should be considered.

By anatomically localizing a neurologic problem, a list of differential diagnoses of the diseases known to affect that area can be formulated. This may be simplified if options are grouped according to broad categories of disease that include

- Degenerative disorders
- Anomalies (malformations)
- Metabolic diseases
- Nutritional and neoplastic (primary or secondary) disorders
- Inflammatory (primary or immunogenic), infectious, and ischemic diseases
- Traumatic and toxic disorders

Additional diagnostic tests then may be selected to determine a definitive diagnosis, treatment plan, and prognosis.

History

Some important questions to be addressed when presented with the horse with neurologic disease include

- How old is the horse and what is it used for?
- What is the breed and sex of the horse?
- Has the horse been regularly vaccinated and dewormed?
- What season of the year did the problem begin?
- How long have the signs been present?
- Are the signs progressive?
- Were the signs sudden or insidious in onset?
- Are there other neurologic signs?
- Are there other physical signs?
- Is there a history of trauma?
- Is there a history of intercurrent infectious disease?
- Are other horses affected?
- Has the horse been recently imported?
- Has the horse's diet been altered recently?
- Is the quality of feed source adequate?
- Has the horse had access to possible toxic plants?
- Has the horse received any medications recently?
- Has the horse's behavior altered?
- Has the horse's gait altered?
- Is the horse insured?

Examination Procedure

KEY POINT

The procedure for neurologic examination is simplified if initial efforts are directed toward defining the neuroanatomic site of the lesion using a craniocaudal approach. Accurate recording of findings is essential, and repeated examinations frequently are necessary.

Although clinicans should develop a format that achieves an accurate and complete neurologic examination, the method described by Mayhew (1989) is logical and sequential and is broadly outlined below.

Examination begins at the head, evaluating the function of the brain and cranial nerves. Assessment of the spinal cord follows, focusing on the neck, forelimbs, back, hindlimbs, tail, and anus. The peripheral nerves are the final structures to be examined.

EXAMINATION OF THE HEAD

Evaluation of behavior, mentation, head posture and movement, and cranial nerves is performed to determine if there is evidence of brain (i.e., cerebrum, brainstem, cerebellum) or cranial nerve disease. Examination should include observation of the horse at rest in its normal environment and during motion and careful palpation of appropriate structures of the cranium and neck.

Demeanor and Behavior. Information relating to the animal's current and previously observed behavior patterns should be obtained from the owner or handler. This is important because differences exist between individuals, sexes, breeds, and ages. Abnormal behavior (e.g., head pressing, compulsive yawning or wandering, circling, licking objects, aggressiveness, mild signs of depression, seizures, and blindness) often is obvious and reflects primarily cerebral dysfunction. Some common causes include metabolic derangement (e.g., hypocalcemia, hyperkalemia), skull fractures, protozoal, bacterial and viral infections, and heavy metal toxicosis.

Responsiveness. Response to visual stimuli (menace and bright light), touch of the skin, noise, smell, presence of food, and painful stimuli should be undertaken with the aim of assessing the horse's state of awareness. Responsiveness of the horse is related predominantly to brainstem (reticular activating system) function and to a lesser extent to the cerebral cortex. Mental status varies from normal and alert to signs of depression, stupor, semicoma, and coma. Profound loss of awareness usually is related to lesions of the reticular activating system of the brainstem. Fractures of the basisphenoid-basioccipital bones at the base of the skull and viral, bacterial, and protozoal infections commonly affect the brainstem.

Head Position. Head position and coordination should be assessed in horses during normal activity, eating, and at rest. A cerebral lesion may result in the horse circling, with the neck but not the poll deviated to the affected side. In contrast,

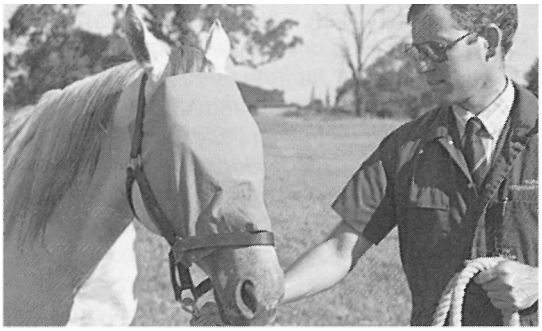


Figure 14-1. Application of a blindfold for assessment of vestibular function.

vestibular lesions frequently result in a head tilt that is characterized by the poll being deviated to the side of the lesion (with the muzzle and neck remaining in the midline), a body lean to the same side, and nystagmus. These signs may be worsened by application of a blindfold (Fig. 14-1). Cerebellar disease with loss of fine coordinated motor control results in jerky movements of the head that is most pronounced when the horse attempts a purposeful movement such as eating or drinking (referred to as *intention tremor).* Also, gait abnormalities without weakness and a reduced or absent menace response are noted. Lesions affecting the cerebellum include basisphenoid-basioccipital fractures and malformations. An abnormal head position also may be due to pain from a fracture or infection (e.g., injection reaction in the neck) and as a result of neck weakness (e.g., in botulism or equine protozoal myeloencephalitis [EPM]).

A summary of the major deficits resulting from lesions in different sites of the brain is included in Table 14-1.

Examination of the Cranial Nerves

Abnormalities found in cranial nerve examination are helpful in localizing a lesion to a specific nerve or to the brainstem. Dysfunction of several cranial nerves concurrently also may suggest lesions at anatomic locations through which one or more of these cranial nerves pass (e.g., caudolateral guttural pouch disease may affect cranial nerves VII, IX, X, XI, and XII). Cranial nerves may be examined either in order (from I to XII) or more conveniently by evaluating

- Eyes (assessing cranial nerves II, III, IV, and VI and cerebellar and sympathetic control)
- Face and nose (assessing cranial nerves I, V, and VII and sympathetic control)
- Ears and balance (assessing cranial nerve VIII and vestibular control)
- Mouth, saliva production, and prehension (assessing cranial nerves V, VII, IX, and XII and basal nuclei)
- Pharynx and larynx (assessing cranial nerves IX, X, and XI)
- Neck musculature (assessing cranial nerve XI)

Unilateral abnormalities in cranial nerve function can be summarized as follows.

TABLE 14-1. Major Findings in Lesions Involving the Brain

Site of the Lesion	Mental Status	Gait Abnormalities	Cranial Nerve Involvement
Cerebrum	Depression, ± coma, ± seizures, ± circling, ± head pressing, ± blindness with normal PLR	Mild weakness	No
Brainstem			Yes
Rostral	Profound depression to semicoma to coma; changes in behavior, polydipsia, polyuria, hirsutism	Variable: none through to ataxia/weakness ± tetraparesis	CN II-IV
Caudal	Semicoma to coma	Hemiparesis to quadriplegia	CN V-XII
Cerebellum	Intention tremor, ± menace reflex	Dysmetria/spasticity no weakness	No (except menace response)

CN, crania] nerve; PLR, pupillary light reflex.

I—Olfactory Nerve. Responsible for sense of smell. Although function of this nerve is difficult to assess, crude assessment can be made by applying a blindfold to the horse and positioning alfalfa, apples, mint, or feces in close proximity to nasal openings.

II—Optic Nerve. Responsible for vision. Assessment of function is made by the menace response, in which a hand or finger is directed toward the eye (without causing air currents) in a threatening gesture to elicit a blink. A normal menace response depends also on a functional facial nerve (cranial nerve VII). At times, depression or excitation results in a negative response to a menace test. Neonatal foals may not respond or may become refractory to repeated menace testing. A further assessment of vision can be made by requiring the horse to walk through an obstacle course (Fig. 14-2) or by watching the horse when placed in a strange environment. The pupillary light response (PLR) also can be used, although this tests the function of both the optic and oculomotor nerve (cranial nerve III) simultaneously. The optic nerve can be damaged by head trauma and pituitary tumors.

III—Oculomotor Nerve. Responsible for motor control of medial and ventral ocular muscles, upper eyelid muscles, and parasympathetic control of pupillary diameter (causing constriction). Dilatation of the pupil is controlled by the dilator muscles innervated by sympathetic fibers from the cranial cervical ganglion. When assessing the oculomotor nerve, the pupils should be checked for size and symmetry and the response to light, direct and consensual.

KEY POINT

If pupillary constriction is absent when a strong light is directed into one eye but a consensual constriction is observed in the opposite eye, the oculomotor nerve of the stimulated eye is malfunctioning. If neither pupil constricts, the stimulated optic nerve is probably nonfunctional. Importantly, PLR does not involve the visual cortex and therefore the reflex can be normal in a horse with blindness associated with cerebral damage.

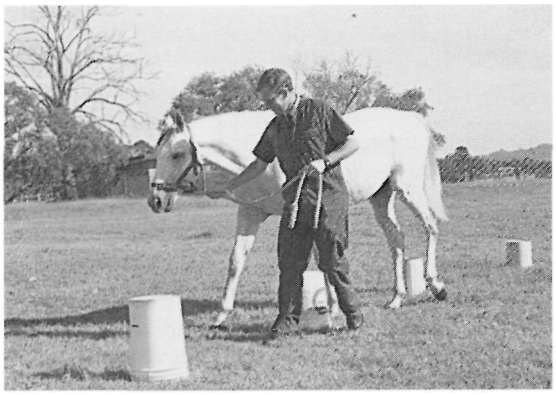


Figure 14-2. Walking through an obstacle course for assessment of vision and proprioceptive function.

Apart from mydriasis (pupillary dilatation), oculomotor dysfunction results in clinical signs of ptosis (drooping of the upper eyelid) and lateral strabismus (deviation of eyeball). The trochlear nerve (cranial nerve IV) and abducens nerve (cranial nerve VI) also are responsible for normal position of the eye due to their innervation of the extraocular muscles. Observing the position of the eyes within the orbits and eye movement when the head is moved tests all three cranial nerves. In general, varying degrees of damage to cranial nerves III, IV, and VI occur together rather than to one single nerve to produce strabismus. Dysfunction can be the result of compressive or inflammatory brainstem lesions or ocular trauma.

KEY POINT

Appaloosa horses with night blindness have a strabismus (star gazing) that is secondary to a bony orbit malformation. This is not indicative of cranial nerve dysfunction.

IV—Trochlear Nerve. Responsible for motor control of dorsal ocular muscles and normal eye position. Lesions of this nerve in which dorsomedial strabismus is observed are rare, but it may be damaged by fractures to the base of the skull.

V—Trigeminal Nerve. Supplies motor innervation to the muscles of mastication and sensory nerve fibers to mucous membranes of the head and most cutaneous structures, including the cornea. This nerve has three branches: mandibular, maxillary, and ophthalmic. Loss of motor function of the mandibular branch results in a dropped jaw and an inability or decreased capacity to chew. The tongue may protrude from the mouth, and drooling of saliva is common. After about 10 to 14 days, there is atrophy of the temporal and masseter muscles. Assessing sensation of the skin and mucous membranes of the head tests sensory function of all three branches. This is done by tapping or pricking the ear, eyelids, cornea, internal nares, and lower lip to produce movement (withdrawal). These movements also require an intact facial nerve. Reduced or no sensation to one side of the face may result in feed impacting in

the rostral cheek pouch. Apart from brainstem lesions, trigeminal nerve dysfunction often is seen with polyneuritis syndromes, EPM, and in cases of yellow star thistle intoxication.

In addition, malfunction of the sympathetic nerve supply to the pupil as it travels to the eye with the ophthalmic branch of the trigeminal nerve may produce *Horner's syndrome.* Signs include pupillary constriction (miosis, with subsequent pupillary asymmetry), ptosis, enophthalmos, and protrusion of the third eyelid. Also noted are nonocular findings such as increased facial temperature, hyperemia of nasal and conjunctival mucosae, and sweating observed on the side with the lesion due to denervation hypersensitivity of the sweat glands to circulating catecholamines. More commonly, signs of Horner's syndrome are observed with damage to the vertebral spine at Tl to T3 or to the brachial plexus and secondary to space-occupying lesions or infections in the neck (affecting the vagosympathetic trunk) or retropharyngeal area (e.g., guttural pouch). Ventral skull fractures and retrobulbar infection or neoplasia also can result in Horner's syndrome.

VI—Abducens Nerve. Responsible for motor control of lateral and retractor ocular muscles. Although rare, lesions of this nerve result in medial strabismus and exophthalmos due to inability to retract globe.

VII—Facial Nerve. Innervates the muscles of facial expression and provides parasympathetic supply to the lacrimal and salivary glands. This nerve is responsible for the reflexes described when testing the trigeminal nerve. This nerve controls blinking (assessed by the menace, palpebral, and corneal reflexes) and movement of the ears, lips, and nostrils. Facial paralysis results in drooping of the ear and lips, ptosis of the upper eyelid on the affected side, and muzzle deviation to the unaffected side. Saliva may drool from the mouth, and food may collect in the cheek on the affected side. Distal lesions involving peripheral components of this nerve affect only the function of the lips and nostrils, whereas with central (i.e., brainstem) or proximal (i.e., middle and inner ear) lesions all these functions usually are compromised. As well, tear and salivary production may be decreased and may result in corneal ulceration or partial dry mouth respectively. In addition, cranial nerve VII also mediates taste from the rostral two thirds of the tongue, but this is difficult to assess. Facial nerve dysfunction is common, resulting from trauma, guttural pouch disease, yellow star thistle intoxication, or polyneuritis. Occasionally, focal cerebral lesions or peripheral nerve irritation may result in facial spasms or grimacing.

VIII—Vestibulocochlear Nerve. The cochlear division of cranial nerve VIII is responsible for hearing. Although unilateral hearing loss is difficult to assess, bilateral otitis media or interna or both may produce complete deafness. The vestibular division of cranial nerve VIII provides the major input to the vestibular nuclei in the brainstem from which connections to the cerebellum and cerebrum occur. Together they control the orientation of the head, body, limbs, and eyes in space (i.e., balance or proprioception). Signs of unilateral vestibular dysfunction include a head tilt and circling toward the side of the lesion. Nystagmus occurs frequently, either with the head in a normal position (spontaneous) or when it is held in various abnormal positions (positional). In *peripheral vestibular disease* (e.g., involving the inner ear), the direction of nystagmus (slow phase) is *toward* the side of the lesion and remains the same (horizontal or arc shaped) regardless of head position. Signs of peripheral vestibular disease usually improve over a period of days to weeks as the horse accommodates to the problem. However, blindfolding often will exacerbate the signs. Care should be exercised when doing this because some horses may rear over backward. In contrast, with *central vestibular lesions* (e.g., involving the caudal brainstem or medulla), the nystagmus (horizontal, vertical, or rotary) is inconsistent and may change as the head posture alters. Limb weakness resulting in ataxia and circling toward the side of the lesion usually is more noticeable with central vestibular dysfunction than with peripheral disease. Signs of depression and involvement of other cranial nerves (e.g., cranial nerve V or VII) also indicate central disease. Bilateral vestibular lesions are characterized by more symmetric signs with wide swaying of the head and sometimes the body, similar to generalized cerebellar disease. Lesions of the vestibulocochlear nerve most commonly occur as a result of middle or inner ear infections, temporohyoid osteoarthropathy, and petrous temporal bone fracture secondary to otitis or head trauma, polyneuritis, and caudal brainstem diseases.

IX—Glossopharyngeal Nerve, X—Vagus Nerve. These nerves provide sensory (cranial nerves IX and X) and motor (cranial nerve IX) fibers to the pharyngeal and laryngeal muscles that control swallowing. As well, the glossopharyngeal nerve relays sensory input from the caudal one third of the tongue and guttural pouch, whereas the vagus nerve supplies parasympathetic motor innervation to major visceral organs, including the heart, lungs, and most of the gastrointestinal tract. Observing normal swallowing of food and water and assessing the swallowing reflex by passage of a nasogastric tube tests the function of these

nerves. If required, endoscopic examination can be used to evaluate laryngeal movements (to detect hemiplegia), esophageal patency, and palatal defects. Other useful tests are the gag reflex (touching pharyngeal wall), cough reflex (touch or compressing the larynx), and the oculocardiac reflex (pressure on the eyeball to elicit bradycardia with normal vagal function). Glossopharyngeal and vagal dysfunction is most often manifest as dysphagia, exercise intolerance, tachycardia, ileus, and laryngeal paralysis leading to secondary inhalation pneumonia and discharge from the nostrils. Lesions commonly are due to guttural pouch disease (along with involvement of cranial nerves VII, XI, and XII), rabies, botulism, lead intoxication, and vitamin E/selenium deficiency.

XI—Accessory Nerve. Provides motor innervation to the muscles of the neck and scapula (i.e., trapezius, omotransversarius, brachiocephalus, and sternocephalicus). Lesions of this nerve are rare and result over time in denervation atrophy, prominent transverse processes of the cervical vertebrae, and an abduction of the dorsal scapula. Abnormal electromyographic activity also is observed when these muscles are tested.

XII—Hypoglossal Nerve. Supplies motor innervation to the muscles of the tongue. Unilateral lesions result in weakness of the tongue with deviation to the unaffected side and eventually atrophy of the muscles of the tongue. Bilateral palsy results in inability to withdraw the tongue into the mouth (in particular when traction is applied to the tongue) and difficulty in prehending food. Painful tongue lesions due to foreign bodies and horses with severe systemic disease also may display tongue hypotonia, which can mimic paralysis. Guttural pouch disease and yellow star thistle intoxication are possible causes of cranial nerve XII dysfunction. In contrast, signs of hypertonicity, abnormal spastic movements of the tongue, and inability to prehend can be observed in basal nuclei disease, also a result of yellow star thistle intoxication.

EXAMINATION OF THE SPINAL CORD

Neurologic diseases of the spinal cord are common. Trauma, osteomyelitis, tumors, and developmental bony malformations cause focal compressive lesions, whereas infectious, degenerative, metabolic, and parasitic diseases cause diffuse lesions within the spinal cord.

KEY POINT

In general, spinal cord disease causes bilateral involvement of the body with clinical signs in body parts caudal to the site of the lesion.

Localization of a lesion to the spinal cord is aided by subdividing the entire spinal cord into six functional segments: upper cervical (C1 to C5), brachial enlargement (C6 to T2), thoracolumbar area (T3 to L3), lumbar enlargement (L4 to S2), sacral segments (S3 to S5), and coccygeal segments (CY1 to CY5). Most spinal cord diseases affect all four limbs to some degree and are the result of cervical compressive lesions (e.g., stenosis, fractures, abscessation) or diffuse spinal cord diseases (e.g., EPM, herpes virus myeloencephalopathy, degenerative myeloencephalopathy, and toxicoses).

KEY POINT

With increasing compression of the spinal cord, functional deficits progress in the following order: loss of proprioception, motor weakness, loss of perception or response to touch, and loss of pain perception.

The localization and differentiation of spinal cord dysfunction in the horse depend heavily on analysis of gait and posture and the determination of the presence of weakness and/or ataxia (i.e., depression or loss of voluntary movement and proprioceptive deficits). Specific manipulative procedures and tests subsequently are required to detect other clinical signs attributable to a spinal cord disorder (i.e., alteration of spinal reflexes, changes in muscle tone, muscle atrophy, and sensory dysfunction).

Proprioception is knowledge of the location in space of the body and limbs. Unconscious proprioception (i.e., muscle stretch perception) is integrated primarily in the cerebellum. Conscious proprioception (i.e., joint perception and position) is mediated by the cerebral cortex, whereas special proprioception (i.e., balance and orientation to gravity) is mediated by the vestibular system, cerebellum, and spinal cord.

It is important to note that many subtle gait deficits due to neurologic dysfunction can easily be mistaken for musculoskeletal problems. Thorough physical examination with analysis of serum biochemical variables should rule out such problems as laminitis, rhabdomyolysis, or degenerative joint disease.

KEY POINT

Interpretation of subtle alterations in gait and locomotion often requires repeat neurologic examinations.

TABLE 14-2. Gait and Posture Abnormalities

Lesion Location	Abnormal Posture	Paresis	Ataxia	Spasticity	Hypermetria
Cerebrum	+ + +	0	0	0	0
Brainstem	+ +	+ +	+ +	+ +	+ +
Vestibular	+ + +	0	+ +	+ +	0
Cerebellum	+ +	0	+ + +	+	+ + +
Spinal cord	+ +	+ +	+ +	+ +	+ +
Peripheral nerve	+ +	+ + +	+	(+ +)*	(+ + +)*
Musculoskeletal	+	+ +	0	+	0

*Usually only with selective sensory fiber involvement.
0, not usually expected; +, mild if present; + +, usually present; + + +, characteristically present.
Adapted from Mayhew, I. G.: Large Animal Neurology: A Handbook for Veterinary Clinicians. Philadelphia, Lea & Febiger, 1989.

Evaluation of Gait and Posture

Gait requires normal function of almost all parts of the nervous system. As well as the spinal cord, lesions of the peripheral nervous system and brain (e.g., cerebrum, brainstem, cerebellum and vestibular system) can cause abnormalities of gait and/or posture (Tables 14-1 and 14-2). However, focal peripheral nerve lesions usually involve a single limb, whereas lesions of the brain frequently are accompanied by multiple behavioral, mentation, head, or cranial nerve deficits.

TABLE 14-3. Criteria for Grading Ataxia, Weakness, Spasticity, and Dysmetria

Grade	Deficit
0	Normal—no deficit
1	Deficit may be detectable at normal gaits. Exacerbated with manipulative procedures (e.g., turning in tight circles, walking up/down a slope ± elevation of head).
2	Deficit obvious at normal gaits. Signs exacerbated with manipulative procedures (e.g., turning in tight circles, walking up/down a slope ± elevation of head).
3	Signs particularly obvious at normal gaits. Horses give the impression they may fall (but do not) and buckle with manipulative procedures (e.g., circling, backing, walking up/down a slope, tail pull, etc.).
4	Profound deficits at normal gait. Horse frequently stumbles and may fall at normal gaits or when manipulative procedures are utilized (e.g., circling, backing, walking up/down a slope, tail pull, etc.).
5	Recumbent horse

Gait deficits are classified as weakness, ataxia, spasticity, and dysmetria and are graded 0 to 5 depending on severity (Table 14-3). Ideally, each limb should be graded to define the severity of involvement and to serve as a numeric base on which the animal's improvement or disease progression can be monitored in repeated examinations. Gait and proprioceptive deficits are assessed with the horse walking, trotting, turning, and suddenly stopping (Fig. 14-3); backing, moving up and down an incline (Fig. 14-4); and after application of a blindfold (see Fig. 14-1). Subtle neurologic deficits in gait and proprioception can be detected by performing *special postural reaction tests.* This includes a series of manipulations that place increasing demand on the neurologic system. Horses are walked through an obstacle course (see Fig. 14-2) and over a step or curb (Fig. 14-5), although this latter test is not always reliable. Also, elevation of the head at normal gaits or while walking up, down, or sideways on an incline may reveal subtle deficits in visual placing, coordination, and strength, especially in the forelimbs (Fig. 14-6). Other tests involve crossing the forelegs (Fig. 14-7) and forcing the horse to adopt a basewide stance. This test is most useful for assessing hindlimb proprioception (Fig. 14—8). Postural reaction techniques for horses test proprioceptive fibers of the peripheral nerves, spinal cord, brainstem, cerebrum, cerebellum, and inner ear.

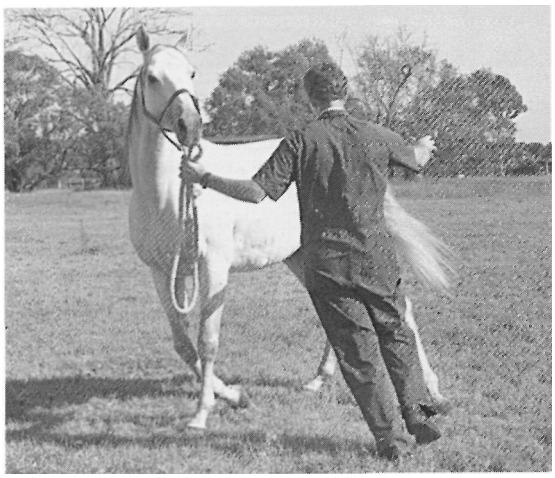


Figure 14-3. Turning the horse in tight circles to assess ataxia and weakness. Particular note should be made of the horse stepping on itself and the degree of circumduction of the outside hindlimb.

KEY POINT

Although postural reactions are good screening tools for the detection of neurologic injury, they are not valuable in precise anatomic localization, particularly of spinal cord lesions. This may require more specific testing that may include proprioceptive reflex testing (e.g., tail pull test, patella reflex in recumbent animals or foals), nociceptive (pain) reflex testing, and use of additional diagnostic aids (see below).

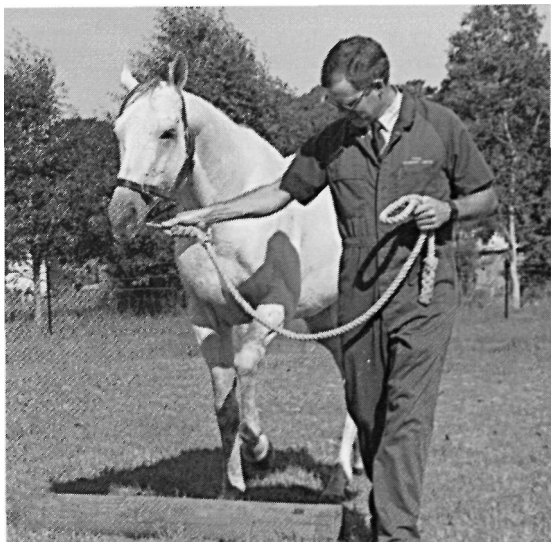


Figure 14-5. Walking over an obstacle to assess proprioceptive function.

Weakness (Paresis). Weakness is a partial impairment of voluntary motor function in a body part, whereas *paralysis (plegia)* is the complete loss of motor function in a body part. Weakness

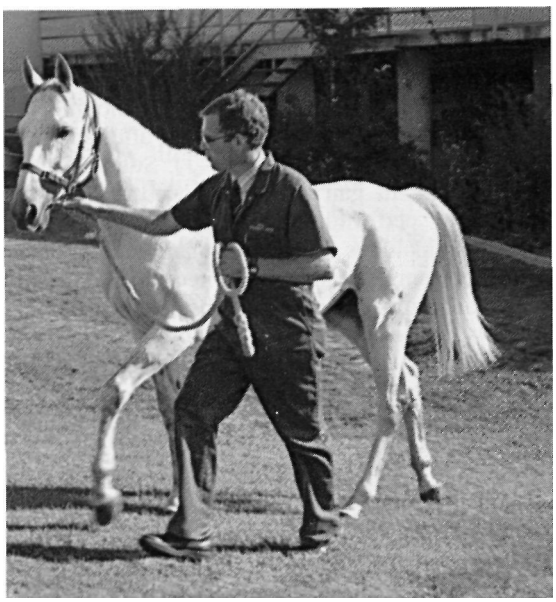


Figure 14-4. Walking up a slope for assessment of the gait. This test is used to exacerbate abnormalities such as spasticity, dysmetria, weakness, and ataxia.

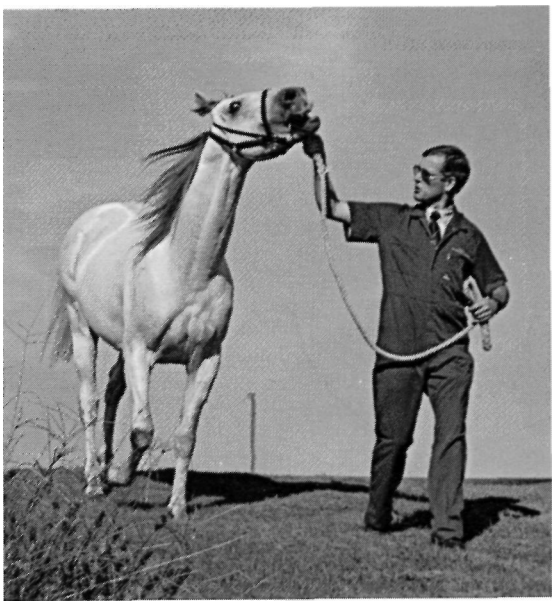


Figure 14-6. Walking the horse down an incline with the head elevated will often exacerbate ataxia, spasticity, and dysmetria.



Figure 14-7. Crossing of the forelimbs to evaluate proprioceptive function. A delayed return of the limb to a normal position may indicate neurologic dysfunction. Care should be taken to avoid the legs touching, because skin sensation may provide false results.

is demonstrated by dragging the feet, stumbling, and possibly increased wear of the toes. When bearing weight, an affected limb will show increased extension of the fetlocks and possibly shaking of the limb, knuckling, and dropping of the body as a whole. These changes are worsened when the horse is required to walk in a tight circle or up/down a slope (particularly with the head elevated; see Fig. 14-6) or is backed up. Weakness in horses occurs (in order of severity) with lesions of the peripheral nerves or myoneural junction, spinal cord, and brainstem. Paresis rarely is a feature of cerebral dysfunction and is absent with lesions involving the cerebellum or vestibular system.

Ataxia (Incoordination). This is an unconscious proprioceptive deficit in which failure of muscle coordination becomes obvious when the horse moves the limbs. Ataxia causes an unstable (swaying) gait and possibly interference of the affected limbs. This results in abnormal foot placement, which becomes worse when the horse is walked up and down a slope. Severely affected horses may step on the opposite foot. The limbs are often circumducted, particularly when the horse is required to turn in tight circles. Pivoting on the affected limbs also may occur during this maneuver or when backing the horse. Ataxia most often is the result of damage to the cerebellum or vestibular system (without paresis) or the spinal cord or brainstem (usually with paresis).

Spasticity (Stiffness). Spasticity describes stiff movement of the limbs with reduced flexion of the joints. Affected horses may have a "tin soldier" gait (also referred to as *hypometria).* This sign often is most obvious in the carpus and hock. Spasticity can be exaggerated when the horse walks up and down an incline with the head elevated. Spasticity is common with cervical spinal cord, brainstem, and vestibular lesions.

Dysmetria. This describes alterations in the

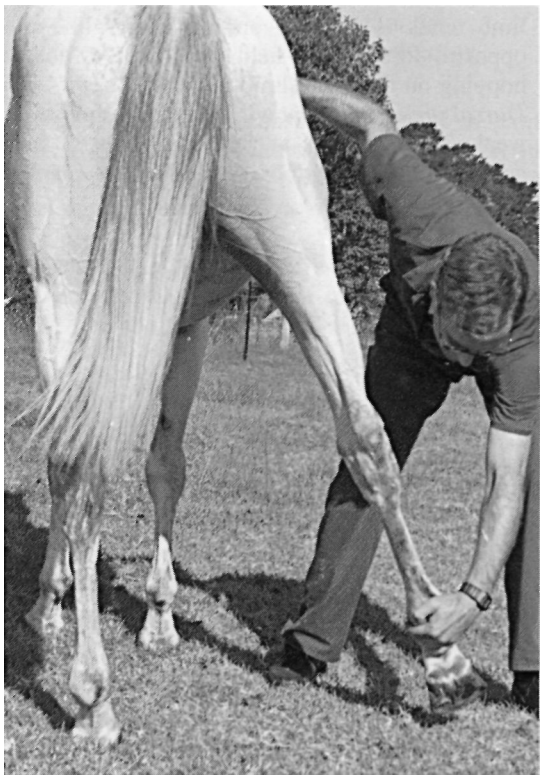


Figure 14-8. Forcing the horse to adopt a base-wide stance in the hindlimbs as an assessment of proprioceptive function. A delayed return of the limb to a normal position may indicate neurologic dysfunction.

range of movement of limbs or joints. It usually occurs as overstepping with excessive joint movement *(hypermetria).* This results in an altered stride length. Hypermetria without weakness (plus an intention tremor) is characteristic of cerebellar disease. Ataxia and weakness is more easily recognized than spasticity and dysmetria.

Other manipulative procedures for evaluation of paresis and ataxia are

- The *sway test,* where the clinician pushes against the horse's shoulders to assess postural reactions (i.e., capacity to resist the force before stepping laterally) (Fig. 14-9). This test is done with the horse standing and then with it walking. This test often demonstrates weakness and/or ataxia, as reflected by a reduced resistance and stumbling when pressure is applied to the shoulder. Similarly, pushing against the pelvis with the horse standing and walking and assessing the resistance and proprioceptive responses help define weakness and ataxia in the pelvic limbs.
- *Thoracic limb hopping* can be performed by lifting each forelimb in turn and the horse made to hop sideways when pushed with the examiner's shoulder. A horse that is weak on a thoracic limb tends to tremble on the limb while the opposite forelimb is held up. Also, difficulty hopping on the weak limb is noted.
- *Dorsal pressure on the withers* may demonstrate weakness if there is an inability to resist with collapse of the forelimbs. Normal horses display arching of the back.



Figure 14-9. The "sway test," pushing on the wither with the horse standing, assesses strength and proprioceptive function because the horse should resist the force.

Specific Manipulative Procedures for Evaluation of Spinal Cord Function

After assessment of the gait, the neck, forelimbs, trunk/back, hindlimbs, and perineum/tail are examined for symmetry, gross skeletal defects, patchy sweating, reflex cutaneous responses to pain, and recognition of any *upper motor neuron* (UMN) or *lower motor neuron* (LMN) signs. Demonstration of abnormalities may assist to precisely pinpoint a problem to a specific spinal cord segment (described previously).

- *Upper motor neurons* arise from cell bodies located in the brain. Their axons descend the spinal cord, terminating at LMNs to initiate voluntary movement. UMN signs are seen in thoracic limbs with lesions of the cervical segment (C1 to C3) and in the pelvic limbs when lesions involve the cervical, brachial enlargement, or thoracolumbar segments (C1 to L3). Signs include paresis to paralysis (tetra- or para-), increased muscle tone (spasticity), ataxia, normal to exaggerated spinal reflexes caudal to the lesion (in recumbent horses), decreased sensation caudal to the lesion, and muscle atrophy due to disuse caudal to the lesion.
- *Lower motor neurons* arise from cell bodies located in spinal cord gray matter. Their axons leave the spinal cord via ventral nerve roots and terminate at skeletal muscle of the head and body through cranial and peripheral nerves. LMN signs are seen in the thoracic limbs when a lesion is between C6 and T1 (brachial enlargement) and in the pelvic limbs when the lesion is between L5 and S2 (lumbar enlargement). Signs include paresis to paralysis (tetra- or para-), decrease in or loss of muscle tone, ataxia, decreased or loss of sensation caudal to the lesion, depressed to absent spinal reflexes (in recumbent horses), and rapid severe denervation muscle atrophy caudal to the lesion.

KEY POINT

It must be remembered that recognition of UMN or LMN signs can localize a lesion not only to specific areas in the spinal cord but also to lesions within the brain or anywhere along the LMN pathway (peripheral axons and muscles). Diffuse or multicentric disease is less definable using UMN or LMN terminology.

- *The neck, forelimbs, trunk, and hindlimbs* should be observed and palpated for evidence of malformation, asymmetry, and muscle atrophy. The neck should be manipulated dorsoventrally and laterally to assess normal range of movement

(Fig. 14-10) and the horse watched while grazing. Reluctance to flex the neck may reflect cervical pain. In addition, observation at rest for rapid oscillatory muscle tremors or coarse jerking of muscle groups (myoclonus) should be performed.

- *The laryngeal adductor response or slap test* can be useful as an aid in assessment of cervical spinal cord disease. The test can be performed by palpating the dorsal and lateral laryngeal musculature over the arytenoid cartilage and feeling the normal "flick" or adduction of the contralateral arytenoid as the dorsal chest wall on one side is slapped during expiration. Observing the larynx through an endoscope while performing the test may be necessary. In horses with cervical spinal cord disease, the adductor response of the contralateral arytenoid cartilage is often (but not consistently) absent.
- In general, well-delineated *dermatomal patterns of sweating* on the neck, shoulder, and trunk occur caudal to lesions involving the descending sympathetic tracts in the spinal cord. However, care must be taken in interpreting patchy sweating that is not well demarcated, in particular in excited or distressed horses. Also, sweating of the face and neck to the level of C2 may be observed in unilateral Horner's syndrome.
- *Skin sensation* should be assessed over the neck, trunk, and limbs. This procedure is undertaken to assess sensory fibers from the skin (dermatomes), muscle or joints, specific spinal cord segments, and motor nerves to cutaneous muscles that elicit a twitch (i.e., reflex arcs). A pen or probe is used to map areas of decreased or increased sensation. Perception of pain also should be evaluated, although separately from segmental reflex action. A two-pinch test is used to assess decreased perception and response to painful stimuli. This is performed by inserting a fold of skin into the jaws of a hemostat and after the horse has settled, a brief sharp squeeze is applied to elicit a pain response.

KEY POINT

Lesions of the spinal cord decrease sensation caudal or distal to the lesion, sometimes increase sensation at the lesion site, and do not affect sensation proximal to the lesion. Therefore, testing should he done in a caudal to cranial and distal to proximal direction to evaluate decreased sensation through increased sensation to normal sensation.

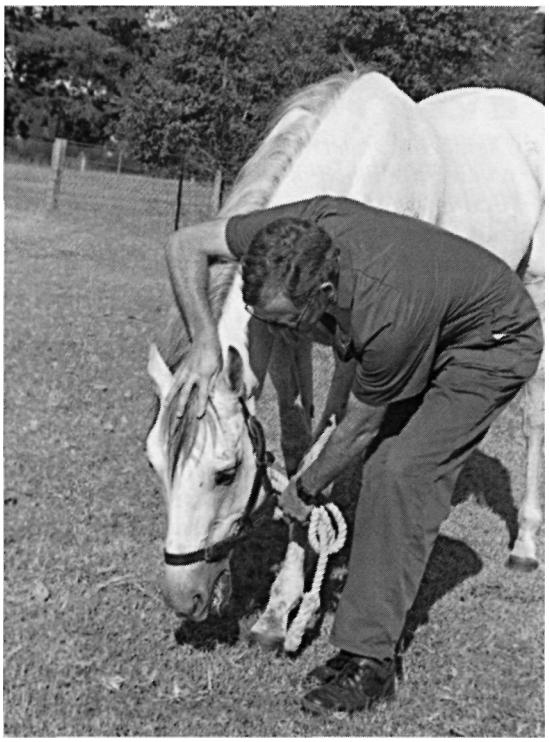


Figure 14-10. Flexion of the neck. The neck should be flexed dorsoventrally and to each side to assess pain.

- *Hyperesthesia* (increased sensitivity or pain) generally reflects irritation, compression, or inflammation of a spinal cord segment, nerve root, or sensory nerve and may be present with little or no motor deficit.
- The *cervicofacial reflex* results in a local skin twitch, flicking of the ear rostrally, blinking of the eyelids, and drawing back of the lips on the side being tested ("smile" reflex). This is observed when the skin behind the ear is pricked and the skin along the side of the neck down to the level of the second cervical vertebra is pricked. The *cervical reflex* with flinching of the cervical musculature and skin occurs when the skin of the lateral neck below C2 is stimulated. Absence of these reflexes and local muscle wasting may reveal an abnormality consistent with a cranial or caudal cervical spinal cord lesion, respectively. The *panniculus reflex* of the thorax and body results in contraction of the cutaneous trunci muscles when the skin is prodded along the body with a pen. This response is absent caudal to the level of a lesion in the spinal cord (between C8 and S1). The panniculus reflex cannot be elicited from stimulation over the sacrum or the neck.
- If a pen tip is run over the thoracolumbar area (from the wither to the sacrum) near the spine,

the normal response is for the horse to extend the thoracolumbar spine. In contrast, running the pen or blunt instrument over the croup should result in flexion of the thoracolumbar spine.

- Skin sensation of the limbs is assessed in a similar manner to the body and is briefly discussed below.
- A *tail-pull test* should be performed by applying a lateral pull to the tail with the horse standing still and when moving (Fig. 14-11). This test initiates an extensor (patellar/quadriceps) reflex. A poor reflex results in weakness suggestive of an LMN lesion at the level of L3 to L4. A hyperactive reflex from a UMN lesion involving cervical and thoracolumbar spinal segments results in difficulty pulling to the side while the horse is standing but be easily pulled while walking.
- *Tail tone and the perineal reflex* **should** be tested. Absence of voluntary muscle tone or weakness with lack of resistance to elevation of the tail is indicative of a lesion of sacrococcygeal cord segments, nerves, or muscles. The perineal reflex is then tested by lightly pricking the perineal skin. A normal response occurs with contraction of the anal sphincter and clamping down of the tail. Absence of this reflex suggests dysfunction of the pudendal nerve or sacral or coccygeal spinal segments.
- *A rectal examination* should be performed. This will assist in identification of space-occupying lesions and fractures or luxation of the pelvis or lumbar, sacral, and coccygeal vertebrae. The rectum should be assessed for tone and for fecal retention and the urinary bladder assessed for size and tone. In recumbent adult horses, urination may not occur, and the bladder may become greatly distended. Catheterization to empty the bladder often is required. This will then reduce the volume of the bladder and thereby allow a better examination of the pelvic structures.
- Depending on the level, focal lesions of the sacral and coccygeal spinal segments may involve the cauda equina and result in varying degrees of decreased sensation and tone and depressed reflexes of the tail, anus, perineum, hips, and caudal thighs (LMN signs). Also, urinary incontinence with a flaccid distended bladder and fecal retention usually is found with lesions involving sacral segments S3 to S5.

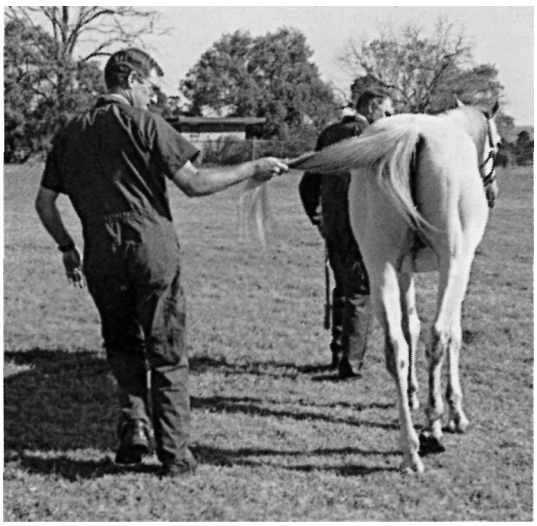


Figure 14-11. The "tail-pull test." With the horse walking, the tail is pulled to the side, and the horse's ability to resist the force and maintain normal foot placement is assessed.

EXAMINATION OF PERIPHERAL NERVES

The peripheral nervous system (PNS) consists of the cranial and spinal nerves that combine to form the peripheral nerves that supply sensory and motor innervation to the entire body. The cranial nerves have been described previously. In general, disease of the PNS is unilateral, limited to one or more nerves within a single limb, and results in *both* sensory and motor dysfunction (i.e., LMN signs) distal to the lesion. In contrast, lesions confined to the spinal cord gray matter or ventral spinal roots produce only motor dysfunction.

KEY POINT

The degree of nerve injury dictates the relative likelihood of regeneration, with restoration of function being most likely with transient interruption of function (e.g., ischemia during recumbency) and least likely with complete severance of the nerve. The rate of regrowth of axons is approximately 1 mm/day or 25 mm (1 inchj/month.

Mononeuropathy in which a specific peripheral nerve or its nerve roots are damaged results from physical injury secondary to compression, laceration, or contusion or from intramuscular injection of drugs. In addition to gait abnormalities (e.g., paresis, abnormal positioning, decreased ability to flex or extend joints) and denervation atrophy, variable degrees of sensory loss are encountered. Generally, specific areas of desensitivity in an affected limb can be pinpointed by using both a sharp probe and the two-pinch technique to a particular spinal nerve or nerves (e.g., radial and/or axillary) that innervate that area. Loss of reflex withdrawal or twitch indicates dysfunction of the nerve, whereas loss of conscious response indicates greater degree of nerve damage.

KEY POINT

If touch is lost but pain is present, it is a good prognostic sign that the nerve fiber is intact and still transmitting sensation.

Anatomic mapping of cutaneous innervation of the thoracic and pelvic limbs has been described. Dysfunction of the radial nerve ("dropped elbow"), musculocutaneous nerve (inability to flex the elbow), and suprascapular nerve ("sweeney") are discussed elsewhere in this chapter.

Polyneuropathy indicates injury or disease to several peripheral nerves such as the brachial plexus, lumbosacral plexus, or the cauda equina. Although rare, physical injury is the most common cause. In contrast, diffuse peripheral neuropathy frequently occurs as a result of metabolic (e.g., hypocalcemia), toxic (e.g., heavy metal intoxication), infectious (e.g., botulism, tick paralysis, EPM), and degenerative (e.g., equine motor neuron disease [EMND]) insults. In addition, some diffuse nervous system conditions such as ryegrass or paspalum staggers, "shivering," stringhalt and "bunny hopping" have not been well defined by particular lesions in the nervous system.

Conclusions after Completion of the Neurologic Examination

KEY POINT

The results of history and the physical and neurologic evaluations should be documented in a logical order during the examination.

In general, neurologic disorders can be suspected after careful history taking and a routine physical examination that incorporates certain basic tests. Signs that are likely to be associated with an abnormality of the nervous system include paresis, ataxia, head tilt, tremor, sensory and proprioceptive deficits, visual deficits, and altered mental status. Confirmation of the presence of neurologic dysfunction is made once the neurologic examination is completed. Concurrently, the major component of the nervous system in which the dysfunction is likely to be located is determined. With this information, decisions can be made regarding the likely differential diagnosis, choice of ancillary diagnostic tests, and treatment strategies. Although practical financial constraints and the necessity to consider the horse's welfare sometimes dictate a poor outlook for some horses suffering from neurologic disorders, in other cases spectacular outcomes are possible.

Neurologic Examination in Foals

Foals have different neurologic responses during the first few weeks of life than those of adult horses, summarized as follows:

- Restraint of young foals may result in a short-lived catatonic (narcolepticlike) state.
- A reduced or absent menace response is common. However, pupillary light reflexes and blinking in response to bright light should be present.
- Jerky head movements are common.
- A wide-based stance often is adopted and may not indicate neurologic dysfunction.
- Dysmetria and apparent incoordination characterize the gait. Some foals in the first few days of life will pace at slow gaits.
- Foals should have a normal suck reflex.
- Responses to the slap test of laryngeal adductor and cervical spinal function is quite variable or absent until about 1 month of age.

Neurologic Examination in the Recumbent Horse

The recumbent horse presents a serious diagnostic and therapeutic challenge. Although recumbent horses should be evaluated as thoroughly as the standing patient, with a complete history (including signalment) and physical examination, aspects of the neurologic examination may be impracticable, and modification is required. However, certain spinal reflexes that cannot be evaluated when the horse is standing can be assessed in the recumbent horse, providing invaluable information. Appropriate laboratory tests such as hematologic and biochemical analysis and cerebrospinal fluid (CSF) analysis also can be performed in recumbent horses to refine the diagnosis and determine the immediate therapeutic needs of the horse.

KEY POINT

Attempts to make the horse stand should be deferred until the horse is stabilized (i.e., attending to oxygen, circulatory, fluid, and drug requirements) and a careful examination is performed in an attempt to rule out fractures.

The neurologic examination in the recumbent horse that is unable to rise should begin initially with assessment of consciousness. Horses with semicoma, signs of severe depression, dementia, blindness, or seizures may have cerebral or brainstem lesions. Recumbent horses that are alert and responsive usually suggest either a spinal cord lesion, peripheral neuropathy, or other disorder unrelated to the neurologic system or secondarily affecting the nervous system. Localization of a lesion to specific spinal cord segments involves assessment of the horse's ability to perform certain head movements and evaluation of spinal reflex activity of the thoracic and pelvic limbs:

- *C1 to C3 lesions:* Horses invariably are unable to lift their heads or have severe pain when raising the head. These horses also display hyperreflexia in all limbs.
- *C3 to C5 lesions:* Horses are able to raise their heads but not their necks and exhibit normal to increased thoracic and pelvic limb reflexes.

KEY POINT

The most common cause of cervical lesions leading to recumbency is cervical vertebral fractures or luxations as a result of trauma.

- *C6 to T2 (brachial enlargement) lesions:* Horses are able to raise their head and neck from the ground but cannot sit sternally. Lesions of the brachial intumescence frequently are caused by trauma and result in hyporeflexic thoracic limbs and normal to hyperreflexic pelvic limbs.
- *T3 to L3 lesions:* Horses usually can sit in sternal recumbency and/or dog sit. Normal thoracic limb reflexes and normal to increased pelvic limb reflexes are observed.
- *L4 to S2 (lumbar enlargement) lesions:* As with thoracolumbar disorders, horses are able to dog sit and have normal thoracic limb reflexes. However, flaccid paralysis and decreased reflexes in the pelvic limbs normally is found.

KEY POINT

Cases of degenerative myelopathy, polyneuritis equi, and herpes virus 1 myeloencephalopathy occasionally present with horses in a dog-sitting posture, whereas pelvic fractures, sacroiliac fractures, and fractures of the femur invariably cause a horse to dog sit. Rarely, horses with severe cervical spinal cord stenosis or subluxation that are too weak to stand might dog sit.

Recumbent horses in which mentation and reflexes are normal should be evaluated for other causes of recumbency unrelated to the nervous system (e.g., myopathy, colic) or secondarily affecting the neurologic system (e.g., metabolic diseases).

Spinal Reflexes

Spinal reflexes can be performed in the recumbent horse using a large rubber mallet or plexor. All reflexes should be classified as normal, increased (hyperreflexic), or decreased (hyporeflexic).

Patella Reflex. This reflex evaluates the femoral nerve (sensory component) and spinal cord segments L4 to L5 (motor component). The hindlimb is held partially flexed and parallel to the ground and the middle patella ligament is tapped. The normal response is sudden extension of the stifle joint and limb.

Cranial Tibial Reflex. This test evaluates the peroneal nerve (sensory component) and spinal cord segments L6 to S1 (motor component). The middle of the long digital extensor muscle (on the cranial lateral aspect of the hindlimb) is tapped. A normal response is flexion of the tarsus.

Hindlimb Flexor Reflex and Nociceptor Response. Depending on where the stimulation occurs, this reflex assesses sensory components of the sciatic, femoral, peroneal, and tibial nerves and spinal cord segments L5 to S3 (motor component). With the hindlimb extended, the skin of the coronary band (sciatic), medial thigh (femoral), dorsal tarsus and metatarsus (peroneal), and plantar metatarsus (tibial) is pinched with forceps. In normal horses, the stimulated limb is withdrawn, although recumbent horses also may have a normal response.

Forelimb Flexor Reflex and Nociceptor Response. This reflex evaluates the sensory components of the median and ulnar nerves and the motor components of the axillary, musculocutaneous, median, and ulna nerves and spinal cord segments C6 to T2. With the forelimb extended, the skin of the distal limb and coronary band is pinched with forceps. A normal response consists of flexion of all joints as the limb is withdrawn.

Biceps Reflex. This reflex assesses the musculocutaneous nerve (sensory component) and spinal cord segments C7 to C8 (motor component). The middle of the biceps and brachialis muscles or the biceps tendon is tapped to elicit contraction of the muscles and elbow flexion.

Triceps Reflex. This reflex tests the radial nerve (sensory component) and spinal cord segments C7 to T1 (motor component). The forelimb is partially flexed and the distal end of the long head of the triceps (at its origin on the olecranon) is tapped. The normal response is sharp contraction of the associated muscle and elbow extension.

KEY POINT

Limb reflexes should be evaluated only on the limbs on the upper side of the laterally recumbent horse. The results of reflex testing on the limbs on the ground may not be accurate.

DIAGNOSTIC AIDS

Cerebrospinal Fluid

Analysis of CSF may be useful in specific cases, especially when neurologic dysfunction is suspected to be the result of inflammatory diseases of viral, protozoal, and bacterial etiology (e.g., EPM, equine herpesvirus 1 disease [EHV 1], bacterial meningitis, and polyneuritis equi). Unfortunately, CSF values may be normal in an animal with severe neurologic deficits, in particular in compressive or degenerative diseases or in diseases involving the PNS. Also, equivocal results often are obtained if CSF is collected too early or late in the course of the disease or if the CSF is collected from a site distant from the lesion. The common sites for collection of CSF are the atlanto-occipital (AO) and lumbosacral (LS) spaces. In general, if a lesion has been localized to an area cranial to C2, CSF collection from the AO space will be more diagnostic, whereas CSF from the lumbosacral site is indicated for lesions caudal to C2.

CSF COLLECTION

Collection from the AO Space

The horse is placed under general anesthesia, and the poll and rostral neck are clipped and prepared as for surgery. In foals and recumbent horses, analgesia and excellent restraint using sedation may be all that is necessary. The head is flexed at a right angle to the axis of the neck. The cranial borders of the atlas are palpated and constitute the lateral landmarks for the puncture site. The eminence of the nuchal crest or the occipital protuberance cranial to the atlas is palpated and used to identify the midline.

KEY POINT

The site for needle insertion is where the line between the cranial borders of the atlas bisects the midline.

With the clinician wearing sterile surgical gloves, an 18-gauge, 9-cm (3.5-inch) spinal needle (Becton-Dickinson, Rutherford, NJ) is inserted. The point of the needle is advanced slowly toward the lower jaw with the bevel directed caudad. There is a progressive increase in resistance to penetration as the needle is inserted. However, when the needle penetrates into the subarachnoid space, there is often a noticeable "give" and no further increase in resistance. The subarachnoid space usually is penetrated at a depth of 5 to 7 cm (2-3 inches) (Fig. 14-12). This approach should avoid penetration of the cerebellum and brainstem. Once the needle is in place, CSF is collected aseptically into a sterile syringe.



Figure 14-12. Line drawing of the atlanto-occipital space showing the site for insertion of an 18-gauge, 9-cm (3.5-inch) spinal needle.

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Collection from the LS Space

This is the most frequently used method for collection of CSF because it can be performed in the conscious standing horse and has fewer risks of complications. In addition, CSF flows caudad, so abnormalities due to spinal cord lesions are more commonly detected in fluid collected from the site. The horse should be restrained in stocks preferably without sedation to avoid an asymmetric stance, which makes collection difficult. Although also demanding, CSF collection in recumbent horses can be made easier if the upper pelvic limb is elevated and both hindlimbs advanced to enlarge the lumbosacral space. Anatomic landmarks used to identify the site for insertion of the needle include a line drawn between the caudal borders of the tuber coxae, the caudal edge of the sixth lumbar spinous process, and the cranial edge of each tuber sacrale. In most horses, firm digital pressure is required to detect the soft tissue depression on the midline just caudad to L6 where the needle is inserted (Figs. 14-13 and 14-14). In obese and recumbent horses, ultrasonography or rectal palpation of the ventral lumbosacral eminence can aid in establishing the proper location of the lumbosacral space. The site is clipped and aseptically prepared. Two to 3 mL of lidocaine (Treatment No. 67) are injected under the skin, and a stab incision is made with a no. 15 scalpel blade. Small amounts of hemorrhage are controlled before insertion of the needle.

KEY POINT

It is most important that the horse stands with weight evenly distributed on both hindfeet when the needle is inserted.

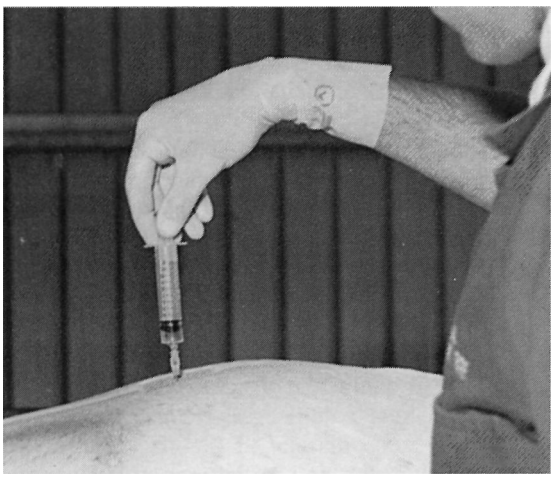


Figure 14-13. Position of an 18-gauge, 15-cm (6-inch) spinal needle just anterior to an imaginary line drawn through the tuber coxae to enable collection of cerebrospinal fluid from the lumbosacral space.

With the clinician wearing sterile gloves and standing beside the horse, an 18-gauge, 15-cm (6-inch) spinal needle (Becton-Dickinson) is inserted (see Fig. 14-13). It is helpful for an assistant to stand behind the horse to ensure that the needle is inserted down the plane of the midline, because small errors in direction at the site of insertion will be greatly magnified at the LS space. In some cases, the clinician may be better able to visualize the plane of insertion if he or she stands on a small platform beside the horse. The needle is inserted to about 11 to 13 cm (4.5-5 inches) in adult horses before penetration of the subarachnoid space occurs. The sensations for the operator are similar to those described for the AO procedure. When the subarachnoid space is penetrated, the horse will often flinch. With correct needle placement, gentle aspiration with a 10-mL syringe will yield CSF. If CSF is not obtained, the spinal needle should be rotated 90 degrees or advanced, provided there is no contact with bone. Manual occlusion of the jugular veins may increase intraspinal pressure and the subsequent yield of CSF.

KEY POINT

Neurologic deficits rarely are caused by penetration of the spinal cord at this level of the caudal CNS.

CSF ANALYSIS

Ideally, CSF pressures should first be measured before withdrawal by attaching a spinal manometer with a three-way stopcock to the spinal needle. A slow or nonexistent elevation in CSF pressure during bilateral jugular occlusion implies obstruction of CSF flow within the cranium or vertebral column secondary to compressive or neoplastic lesions or spinal abscesses. As well, CSF pressure may be increased as a result of systemic changes in blood pressure and in the presence of intracranial tumors, abscesses, hemorrhage, or edema.

After collection, CSF can be subjected to bacteriologic and cytologic examination. In the former case, a sample of the fluid should be placed in a sterile container and forwarded to a bacteriology laboratory. If cell counts and cytologic evaluation cannot be performed within 30 minutes of collection, a portion of the sample should be mixed with an equal volume of 50% alcohol to preserve cellular characteristics. Placing the sample in an EDTA tube (purple top) also may help preserve cells. Samples should be examined for color, clarity, presence of a clot, total and differential nucleated cell counts, and red cell count. Additionally, total protein concentration, albumin concentration, immunoglobulin G (IgG) concentration and the

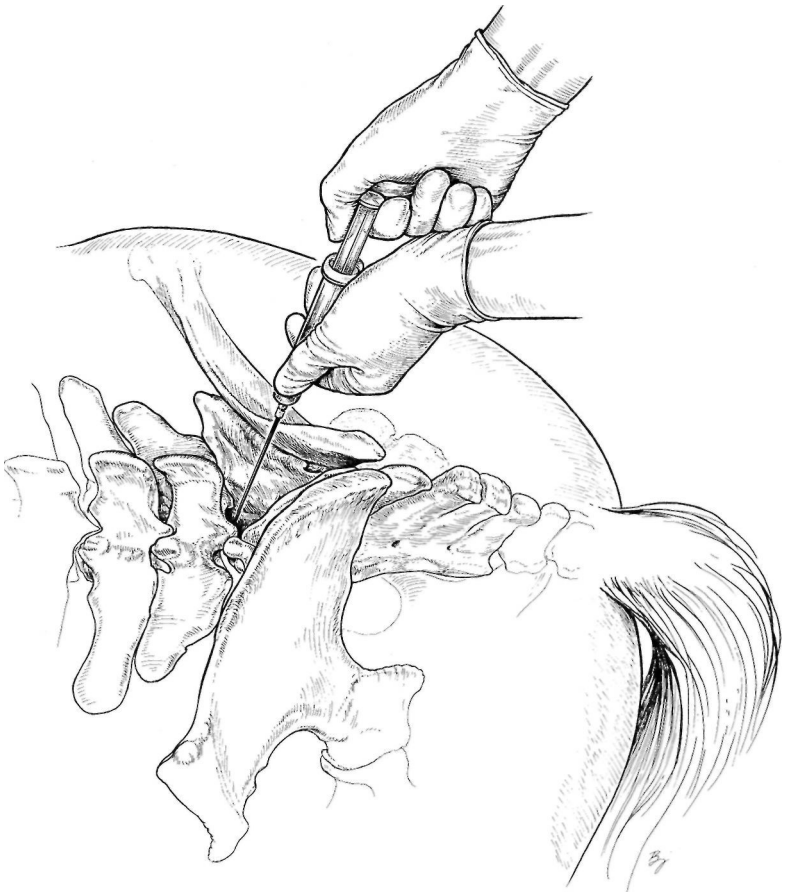


Figure 14-14. Line drawing showing the needle position in the lumbosacral space for collection of cerebrospinal fluid.

derived variables albumin quotient and IgG index, enzyme activity (i.e., creatinine kinase [CK], aspartate aminotransferase [AST], and lactate dehydrogenase [LDH]), glucose, electrolytes, and lactic acid concentration should be determined. Samples of CSF that are contaminated with blood may clot, whereas in cases of bleeding into the CSF, a centrifuged sample will be xanthochromic. Other causes of CSF xanthochromia include increased protein or bilirubin concentrations usually as a result of high serum concentrations or secondary to a damaged blood-brain barrier (BBB). Examples of disorders that may compromise the BBB include trauma, EPM, or EHV 1 myeloencephalopathy. Reference values for the constituents of normal and abnormal CSF are provided in Table 14-4.

Radiology

KEY POINT

Radiology constitutes an extremely valuable tool for defining nervous system disorders, particularly those due to trauma, malformations, or bony infections.

With meticulous technique, positioning, film exposure, and development, definitive identification of most disorders of the skull and cervical vertebral column can be made. Although diagnostically useful radiographs of the head and cervical spine can be obtained in standing horses with small portable x-ray machines, specialized equipment found in referral institutions usually is necessary for optimum results. In particular, radiographic evaluation of the thoracolumbar vertebral column in the adult standing horse is difficult without high-intensity radiographic equipment. Plain radiographs taken of the head can characterize skull fractures; fractures involving the basioccipital, basisphenoid, and petrosal bones; and temporohyoid osteoarthropathy and middle and inner ear infection with involvement of the tympanic bulla. Radiographs of the cervical vertebral column can readily identify AO malformation, displaced fractures, cervical stenotic myelopathy ("wobblers"), and osteomyelitis. In general, radiographs should be

TABLE 14-4. Characteristics of Cerebrospinal Fluid

Variable	Normal Value/Range (AO and LS spaces)	Abnormal Findings
Pressure (mm H_2O)	<500	With intracranial tumors, hemorrhage, or edema No f after jugular occlusion suggests cranial or spinal cord obstruction
Appearance	Clear, colorless, no clot	Some infections may turbidity; fibrinogen due to inflammation may cause clotting Hemorrhage xanthochromia
Specific gravity	1.003-1.005	
Total nucleated cell count (/μL)		
AO space	<1	Bacterial infections: | total number, mostly neutrophils
LS space	<3 Predominantly mononuclear cells	Viral infections: f total number (mainly mononuclear cells, although neutrophils can be with EEE and VEE) Parasitic infections (protozoal, helminth): total number (+ eosinophils) Trauma, large hemorrhage, and neoplasia: variable total number (neutrophils, mononuclear cells)
Red blood cells (/μL)		
AO space	0-558	With blood contamination during collection
LS space	0-167	With CNS trauma (+ xanthochromia)
Total protein (mg/dL)	20-118 (0.2-1.18 g/L) (up to 124 in LS space CSF)	With infectious diseases, trauma, and some toxicoses (e.g., leukoencephalomalacia)
Albumin (mg/dL)	24-56 (0.24-0.56 g/L)	With damage to BBB (e.g., in EHV 1, polyneuritis)
Albumin quotient (CSF albumin:serum albumin)	1.0-2.0	As above
IgG (mg/dL)	3-10 (0.03-0.1 g/L)	With damage to BBB or intrathecal production of IgG in inflammatory diseases
IgG index (CSF IgG:serum IgG/albumin quotient)	0.12-0.27	Intrathecal production due to inflammatory spinal cord diseases (e.g., EPM, bacterial meningitis, viral encephalitides, EMND, polyneuritis, and some tumors)
Enzyme activities		
CK (IU/L)	0-8	CK and AST in EPM, EDM, EHV 1, EMND and other conditions that alter the BBB; LDH in spinal lymphosarcoma
AST (IU/L)	0-16	
LDH (IU/L)	0-8	
Glucose (mg/dL)	35-70% of serum value	Often in bacterial neurologic diseases
Electrolytes		
Sodium (mEq/L)	5-10 < serum values	Alteration in electrolytes may occur after disruption of BBB
Potassium (mEq/L)	2.5-3.5	
Chloride (mEq/L)	5-10 > serum values	
Calcium (mg/dL)	3.3-5.0 (0.03-0.05 g/L)	
Lactic acid (mg/dL)	1.92-2.3 (0.02-0.023 g/L)	(>4.0) in EEE, head trauma, and brain abscess

f, increased; J,, decreased; BBB, blood-brain barrier; CNS, central nervous system; EDM, equine degenerative myloencephalopathy; EEE, eastern equine encephalitis; EHV 1, equine herpesvirus 1; EMND, equine motor neuron disease; EPM, equine protozoal myeloencephalitis; VEE, Venezuelan equine encephalitis.

examined for evidence of proliferation, lysis and abnormal bone shape or bone displacement. Cervical radiographs also should be evaluated for determination of vertebral canal diameter.

Modern high-speed rare-earth screens should be used and a grid generally is required for caudal cervical vertebra and structures caudal to them. If greater definition is required or patient compliance is a problem, sedation or general anesthesia should be considered before taking radiographs. However, general anesthesia may worsen some neurologic problems. Routinely, lateral views only are taken, although dorsoventral projections of the head also can be performed.

KEY POINT

When radiographs do not show abnormalities but nondisplaced fractures of the skull and cervical vertebra or vertebral body osteomyelitis are suspected, horses may be referred for nuclear scintigraphy *as an aid to diagnosis.*

Positive contrast radiography *(myelography)* is widely practiced in larger hospitals and referral centers. Myelographic examination is performed under general anesthesia and is used to detect space-occupying lesions of the vertebral canal (e.g., abscesses, hematomas, or tumors) or to identify and confirm dynamic or static compressive cervical spinal cord lesions. Non-ionic contrast agents such as iohexol (Omnipaque, Winthrop Pharmaceuticals, New York) and iopamidol (Niopam 300, Meriel, New York) are less irritant and have a lower incidence of adverse effects than other agents. After insertion of the spinal needle at the AO site as previously described, 20 to 40 mL of CSF are removed and a similar volume of iohexol (350 mg/mL iodine) is injected over a 4-minute period. The head is elevated at 30 degrees for 5 minutes after injection to facilitate caudal flow of the contrast agent. Flexed, extended, and neutral views of the cervical spine should be obtained. Spinal compression or obstruction is confirmed when there is narrowing of the dorsal and ventral contrast column by >50% at diametrically apposed sites or when a failure of contrast material to pass a specific site is observed. Complications associated with myelography include signs of depression, increased ataxia, seizures, fever, and meningitis. Fatalities have been reported more frequently in horses that are severely ataxic before myelography. It is probably advisable to administer a nonsteroidal anti-inflammatory agent (e.g., phenylbutasone at 4.4 mg/kg PO q12h) to horses before and for 48 hours post-myelogram.

KEY POINT

Although generally only available in tertiary institutions, computed tomography (CT) *can provide additional qualitative information to myelography and radiography regarding the location and severity of spinal cord compression in horses with cervical stenotic myelopathy. Diagnosis of pituitary tumors and brain abscesses in adult horses and hydrocephalus in foals also has been performed using CT.*

Clinical Pathology

Routine *hematology* may provide some indication of diseases with an inflammatory component, as reflected by a leucocytosis and possibly increases in fibrinogen concentrations. In long-standing cases, anemia of chronic disease may be present. In cases where there is suspected trauma and hemorrhage, blood loss may be indicated by a reduction in the packed cell volume and red cell numbers.

Plasma *biochemical analysis* often is useful in assisting the clinician in defining the cause of neurologic dysfunction. Marked elevations in liver enzyme activities and bile acid concentrations are indicative of liver disease, a common cause of encephalopathies in horses. Also, elevations in CK and AST activities in serum generally are indicative of a myopathy. Hypocalcemia, hypoglycemia, or hypokalemia often can be demonstrated in some horses showing signs of muscle tremor, weakness, tetany, and synchronous diaphragmatic flutter. In addition, hyperkalemia (5.0-11.7 mmol/L) during an episode of acute weakness and recumbency in a quarter horse or quarter horse cross may indicate hyperkalemic periodic paralysis (HyPP).

Determination of serum *vitamin E* (ct-tocopherol) concentration may be useful in horses showing ataxia and tetraparesis to identify the early stages of equine degenerative myeloencephalopathy or equine motor neuron disease. Also, decreased whole blood *selenium* concentration and *glutathione peroxidase* activity in weak or recumbent foals or adults may suggest a selenium deficiency myopathy.

Serologic testing using paired serum samples may be useful for confirming the diagnosis of myeloencephalopathy due to EHV 1 and the viral encephalitides (i.e., eastern equine encephalomyelitis [EEE], Western equine encephalomyelitis [WEE], and Venezuelan equine encephalomyelitis [VEE]). However, interpreting serum (and CSF) EHV 1 acute and convalescent antibody titers can

be difficult because of extreme variations between individual horses.

Toxicologic testing in horses is limited to lead determinations in blood and urine.

Electrodiagnostic Tests

Several electrodiagnostic techniques have been used in horses. However, because of expense of equipment, the need for extensive experience for valid interpretation, and the necessity for general anesthesia for most testing, their use is limited to specialty or institutional settings.

Electromyography (EMG) measures the electrical activity of muscle during contraction or at rest from a recording electrode placed in the muscle. *Nerve conduction velocity* (NCV) testing involves stimulating a peripheral nerve with electrical current and recording the electrical activity from other segments of the nerve or from the muscles innervated by the nerve. Diseases of the LMN (e.g., disorders involving the ventral spinal cord cell bodies, ventral root axon, peripheral nerve, myoneural junction, and/or innervated skeletal muscle) can lead to changes in muscle electrical activity or nerve conduction velocity, or both. In horses presented for obscure lameness, muscle asymmetry, muscle atrophy, or ataxia as a result of compressive myelopathies, radial and suprascapular nerve injury, EMND, or EPM, EMG may reveal abnormal electrical potentials, such as fibrillation potentials and positive sharp waves in affected muscles. By combining EMG and NCV testing, the localization, diagnosis, response to treatment, and prognosis of such disorders can be determined. Needle EMGs can be performed in many major extrinsic muscles and on facial, laryngeal, esophageal, pectoral, and external anal sphincter muscles. Needle EMG also may be helpful in evaluating horses presented for signs of primary muscle disease (e.g., muscle tremor, fasciculations, stiffness and weakness) and in early detection of left laryngeal hemiplegia.

KEY POINT

In general, EMG should be performed at least 10 to 14 days after the onset of signs because definitive denervation potentials in muscle occur only after this time.

Electroencephalography is the graphic recording of electrical activity arising from the cerebral cortex. Variations in waveform symmetry, shape, frequency, and amplitude may be helpful to further diagnose, localize, and characterize (i.e., focal or diffuse, inflammatory or degenerative) cerebral lesions in horses with signs of seizures, stupor, and aimless wandering. Response to maintenance treatment for some seizure disorders also can be monitored using electroencephalography. The procedure is best performed with the horse under general anesthesia to avoid artifactual changes caused by eye blinking, head movement, and auditory and visual stimuli. Use of drugs such as acepromazine and xylazine should be avoided. In addition, age specific reference values are available and should be used because electroencephalography of immature horses is significantly different from that of adults.

Auditory brainstem response testing is a graphic recording of electrical activity generated by the eighth cranial nerve and its projections along the brainstem in response to an acoustic stimulus. Qualitative changes in waveforms may be used to distinguish between peripheral and central vestibular disease, to detect hearing deficits, to localize diffuse brainstem disorders, and to detect lesions involving the middle and inner ear.

Diseases of the Brain and Cranial Nerves

Bacterial Meningitis

Equine bacterial meningitis is a rare invariably fatal neurologic disease of the three membranes surrounding the brain and spinal cord.

KEY POINT

Although bacterial meningitis may occur at any age and in any breed, it is most often found in neonatal foals as a sequela to septicemia.

Neonates are apparently more susceptible to meningitis because of an immature immune system and because the BBB may be more permeable to organisms. Older foals and adults develop meningitis as a result of infection introduced by direct trauma to the cranium and as a complication of ascending infection in the cauda equina region after tail docking or anesthetic blocking of the ventral tail. Another means of infection is from extension of an adjacent suppurative lesion (such as in the guttural pouch, sphenopalatine sinus, or pituitary gland) with involvement of intracranial emissary vessels.

HISTORY AND PRESENTING SIGNS

- Neonates in which there has been failure of passive transfer of immunity or signs of septice-

mia or primary infection in other body sites (e.g., lung, gastrointestinal tract and umbilicus)
- Acute onset of signs consistent with diffuse central nervous system dysfunction
- Older horses with signs of intracranial disease and evidence of trauma, exophthalmos, or recent exposure to "strangles"
- Older horses with hindlimb signs and a history of tail blocking or surgery

CLINICAL FINDINGS AND DIAGNOSIS

- Widespread inflammation involving several areas of the central nervous system results usually in progressive, multifocal, or diffuse neurologic signs. Initial neurologic findings are vague and nonspecific and include weakness, signs of depression, and, in neonates, loss of suck reflex.
- Clinical signs progressively worsen with cranial nerve involvement (e.g., head tilt, nystagmus, blindness, drooping lips, eyelids, and ears), head and neck stiffness, star gazing, hyperesthesia, ataxia, recumbency, seizures, and eventually coma.

KEY POINT

Concurrent omphalophlebitis, polyarticular septic arthritis, diarrhea, or pneumonia may increase the likelihood of bacterial meningitis in foals. However, fever is not a consistent finding of meningitis in neonates.

- In adults, bacterial meningitis has been reported secondary to shedding of *Streptococcus equi* from abscesses within the brain, *Klebsiella* septicemia from metritis, *Actinomyces* pneumonia, and secondary to *Bacteroides* infection extending from a guttural pouch and basilar empyema.
- In general, signs progress rapidly in foals and may be fatal within hours of the onset of neurologic signs. In contrast, clinical signs in adults progress over a few days.
- Hematologic examination may reveal changes consistent with those occurring in inflammatory disease, including leukopenia with the presence of band and toxic neutrophils and hyperfibrinogenemia. In foals, failure of passive transfer of immunoglobulins is common, as reflected by low serum IgG concentrations (<4 g/L [<400 mg/dL]). Blood culture should be undertaken in valuable neonates suspected of septicemia and bacterial meningitis.
- Biochemical analysis also should be performed to investigate the possibility of hepatic disease or hypoglycemia. As well, antibody titers may be useful to rule out viral encephalitides.

KEY POINT

Increases in the number of nucleated cells (neutrophils and mononuclear cells), the concentration of protein, and possibly the presence of bacteria in the CSF confirm the diagnosis of meningitis.

- In addition, glucose concentration of the CSF often is low, but this should be compared with blood glucose concentrations, which in septicemic neonates may be lower than normal. Note that normally CSF glucose concentrations are 35% to 70% of plasma values. In foals, CSF and blood submitted for culture frequently reveal bacteria such as *Escherichia coli, Klebsiella* spp., *Salmonella* spp., *Actinobacillus equuli*, and *Streptococcus* spp.
- Ideally, CSF should be obtained early in the course of the disease, preferably taken from the site closest to the lesion.

DIFFERENTIAL DIAGNOSIS

- Trauma
- Septicemia
- Neonatal maladjustment syndrome
- Hypoglycemia or hypocalcemia
- Congenital malformations (e.g., hydrocephalus)
- Brain abscess
- Neoplasia
- Hepatoencephalopathy
- Viral, protozoal, or parasitic encephalitides
- Rabies
- Idiopathic seizure syndrome
- Other toxicities

TREATMENT

- Treatment is directed toward controlling bacterial infection, inflammation, and, if present, seizures. Early diagnosis and treatment are essential for a successful outcome, preferably before fulminant neurologic signs are present.
- Although the healthy BBB often is impermeable to drugs, infection of the meninges increases penetration of most antimicrobial agents into the CSF. In foals with concurrent septicemia, initial antibiotic therapy may involve Na or K penicillin G (15-20 mg/kg IV or IM q6-12h; Treatment Nos. 85 and 86) and gentamicin (2-3 mg/kg IV q8-12h or 6 mg/kg IV q24h; Treatment No. 56) or amikacin (4-8 mg/kg IV or IM q8-12h; Treatment No. 4). However, aminoglycosides develop low CSF concentrations and may be less useful in cases of neonatal meningitis. Cefotaxime sodium (40 mg/kg IV q6-8h;

Treatment No. 16) often is effective but expensive, whereas ceftiofur sodium (2-4 mg/kg IV [not IM] q12h; Treatment No. 18) also may be useful.

- In adults, trimethoprim-sulfadiazine (30 mg/kg IV or PO q12h; Treatment No. 108) or the above antibiotics can be used. Antibiotic selection is subsequently tailored on the basis of culture and sensitivity results. Therapy should be continued for 14 days after sterilization of CSF (or absence of clinical signs) for gram-positive organisms and 21 days for gram-negative organisms.
- Reduction of inflammation, edema, and intracranial pressure can be achieved using mannitol (0.25 mg/kg slowly IV as a 20% solution q4-6h for 24 hours; Treatment No. 68) or dimethyl sulfoxide (DMSO) (1 g/kg slowly IV as a 10-20% solution q12-24h; Treatment No. 34). Although corticosteroids generally are considered contraindicated in infections of the central nervous system, exceptions include critically ill patients in shock or those with severe deterioration of neurologic signs. Prednisolone sodium succinate (1-2 mg/kg IV once; Treatment No. 93) and dexamethasone (0.1 mg/kg IV q24h; Treatment Nos. 29 and 30) can be used. Mannitol may be prohibitively expensive in adults, whereas the anti-inflammatory and antiedema effects of DMSO are reported to be more effective than mannitol and dexamethasone in reducing intracranial pressure.
- If seizures are a problem, treatment with diazepam (0.2-0.5 mg/kg IV as required; Treatment No. 32) often is effective. Longer term control can be achieved with phenobarbital (20 mg/kg IV over 30 minutes as a loading dose followed by 5-10 mg/kg slowly IV as required or 0.5-1.0 mg/kg for foals and 0.5-2.0 mg/kg for adults PO q12h or to effect; Treatment No. 88). To prevent phenobarbital toxicity, the dose regimen should be adjusted for the individual and serum levels kept below 40 p-g/mL.
- Nutritional and intravenous fluid support of foals is imperative to maintain normal hydration, acid-base status, and blood glucose concentration. Adults can be fed a slurry containing feed pellets through a nasogastric tube.
- Recumbent patients should be turned frequently and should be adequately padded to prevent pressure sores. Well-fitting slings also can be used. Concurrent problems such as self-induced trauma, large colon impaction, pneumonia, diarrhea, gastric ulcers, corneal ulcers, septic arthritis, and other complications of septicemia in foals must be addressed appropriately.

Brain Abscess

Abscesses in the cerebral hemispheres usually occur in horses as a sequel to "strangles" (infection with *S. equi* var. *equi),* although other streptococci occasionally are isolated.

HISTORY AND PRESENTING SIGNS

- Usually young horses less than 5 years old
- Previous strangles infection or a history of strangles in the herd
- Fluctuating behavioral changes, gait abnormalities, and blindness

CLINICAL FINDINGS AND DIAGNOSIS

- There may be fever, inappetence, and evidence of strangles abscesses elsewhere.
- Compulsive circling, head pressing, blindness, deviation of the head to the side, and ataxia are common features of brain abscesses. Additional cranial nerve deficits frequently are seen as a result of increased intracranial pressure and brainstem compression.
- Antemortem diagnosis is difficult and is based on the history, clinical signs of cerebral disease, and results of ancillary tests. There may be a leukocytosis (with neutrophilia), hyperfibrinogenemia, and hyperglobulinemia. CSF often has increased protein and total nucleated cell count (neutrophilia and monocytosis). On rare occasions, bacteria may be identified.
- Early diagnosis and localization of cerebral abscesses is possible if CT is available.
- Postmortem examination reveals a pyogenic abscess in the cerebrum or elsewhere in the brain.

DIFFERENTIAL DIAGNOSIS

- Viral encephalitides
- Hepatoencephalopathy
- Trauma
- Equine herpesvirus myeloencephalitis
- Equine protozoal myeloencephalitis
- Bacterial meningitis
- Mycotoxic encephalomalacia
- Verminous encephalitis
- Parasitic thromboembolism
- Neuritis of the corda equina

TREATMENT

KEY POINT

Treatment is difficult and often unrewarding because of the rapidly lethal effects of

abscessation and increased intracranial pressure.

- Successful surgical drainage of a focal cerebral abscess has been described in a horse using CT to define the site of drainage.
- Antimicrobial, anti-inflammatory, and anticonvulsive medications as described for bacterial meningitis are essential. However, long-term therapy with procaine penicillin (20,000-50,000 IU/kg [15-20 mg/kg] IM q12h; Treatment No. 84) or trimethoprim-sulfadiazine (20-30 mg/kg PO q12h; Treatment No. 108) alone for 4 to 6 weeks is mostly unsuccessful if drainage is not performed.

Cerebellar Abiotrophy

Cerebellar abiotrophy is a heritable neurologic disorder in Arabian (pure and crossbred) and Oldenberg horses and Gotland ponies.

HISTORY AND PRESENTING SIGNS

- Arabian, Oldenberg, or Gotland foals of either sex, between 1 and 6 months old
- Abnormal gait and head tremor during eating or drinking
- Generally signs progressively worsen

CLINICAL FINDINGS AND DIAGNOSIS

KEY POINT

Affected foals have a base-wide stance and gait abnormalities characterized by ataxia, dysmetria or spasticity, and a tendency to pace.

- There is no evidence of weakness. An intention tremor occurs, and although most other cranial nerves appear normal, the menace response usually is absent. Signs progress over weeks to months.
- CSF protein concentration may be elevated (>2.0 g/L or 200 mg/dL). CK activity of the CSF also may be increased above 20 IU/L. Other clinical pathology findings are unremarkable.
- Presumptive diagnosis is based on the history, breed, and clinical signs, in particular the presence of an intention tremor. Definitive diagnosis is made on histologic examination of the cerebellum postmortem.

DIFFERENTIAL DIAGNOSIS

- Trauma
- Acquired cerebellar diseases (e.g., herpesvirus myeloencephalopathy)
- Septicemia
- Congenital malformations
- Spinal cord diseases

TREATMENT

- Although there is no successful therapy for cerebellar abiotrophy, some mildly affected foals can function normally as adults if there is sufficient neurologic compensation. However, parents of affected foals should not be bred.

Cranial Nerve Palsies

Cranial nerve (CN) palsies are not uncommon in horses. Lesions can involve specific single or multiple peripheral cranial nerves or affect the central nuclei of these nerves as a result of focal, multifocal, or diffuse brainstem disease. In diffuse brainstem disease, additional signs associated with brainstem, reticular activating system, or cerebral dysfunction also may be noted (e.g., signs of depression, ataxia, and paresis).

KEY POINT

Cranial nerve dysfunction usually results from trauma to the particular nerve or from an inflammatory process secondary to a primary infectious process.

Examination for cranial nerve dysfunction is described in detail in the neurologic examination section at the beginning of this chapter. In this section, only the most commonly affected cranial nerves will be discussed.

HISTORY AND PRESENTING SIGNS

Trigeminal Nerve (Cranial Nerve V)

- Evidence of trauma to the jaws
- Dropped jaw and dropping of food and water from the mouth
- Inability of prehension of food often observed

Facial Nerve (Cranial Nerve VII)

- Evidence of cranial trauma or a recumbent horse thumping its face on the ground
- General anesthesia
- Halter that is too tight with the horse pulling back
- Altered facial expression

Vestibulocochlear Nerve (Cranial Nerve VIII)

- Evidence of cranial trauma, often with profuse aural hemorrhage

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- Rarely, drainage of exudate from the external ear may be observed
- Acute onset of vestibular signs (e.g., head tilt, circling, nystagmus, and ataxia)

CLINICAL FINDINGS AND DIAGNOSIS

Trigeminal Nerve

- Sudden onset of a dropped jaw may occur with or without fracture of the mandible or stylohyoid bone or both. Temporomandibular luxation also can occur in association with fractures of the mandible.

KEY POINT

Neurologic diseases resulting in a dropped jaw, with no other neurologic signs, rarely are encountered in horses. Trigeminal nerve palsy more frequently occurs with other cranial nerve palsies associated with multifocal disorders affecting the brainstem.

- After 10 to 14 days, masseter, digastricus, and temporalis muscle atrophy can be marked. Enophthalmus and drooping of the eyelids may be seen. Lesions can be unilateral or bilateral.
- With trigeminal nerve (sensory branch) involvement, degrees of hypoalgesia also occur. Facial hyperesthesia and facial irritation may result from trigeminal nerve root inflammation (secondary to early, diffuse bacterial meningitis) or idiopathic trigeminal neuritis.
- Diagnosis is based on the physical findings. Diagnosis of fractures is aided by radiography, while diffuse or multifocal neurologic diseases require a thorough neurologic examination and laboratory investigation.

Facial Nerve

- There is drooping of the lips and ear on the side of the lesion and paralysis of the muscles of facial expression, with the muzzle being pulled away from the affected side.
- Ptosis is common, and the ear droops. If there is only damage to the buccal branches of the nerve, as frequently occurs following damage during general anesthesia or as a result of application of a halter that is too tight, ptosis, and ear droop may not be present.
- Facial muscle twitching may be observed prior to paralysis in the early stages of irritative inflammatory lesions.
- Exposure keratitis is a common sequela to facial nerve palsy, resulting from ptosis and reduced tear production.
- With persistence of the palsy (>6 months), there is fibrosis of affected muscles, making return to normal function less likely.
- With bilateral lesions, dysphagia is common.

KEY POINT

Involvement of facial nuclei with diseases affecting the brainstem can produce a selective, partial, facial paresis.

Vestibulocochlear Nerve

KEY POINT

The vestibulocochlear nerve, only one component of the vestibular system, *runs from the receptor organ within the inner ear and terminates in the vestibular nuclei located in the brainstem. Further connections to the spinal cord, cranial nerves that control eye position, cerebellum, autonomic nervous system, reticular formation, and cerebral cortex allows the horse to coordinate eye, limb, and trunk movements with changes in head position.*

- Predominant signs of acute unilateral vestibular system dysfunction include head tilt, circling or staggering toward the side of the lesion, asymmetric ataxia, and nystagmus.
- With *peripheral vestibular disease* (e.g., involving inner ear, most commonly as a result of otitis media/interna or traumatic skull fracture), the nystagmus usually is horizontal or arc-shaped. The fast phase of the nystagmus is directed away from the side with the lesion and will not change with changing head position. Also, while ataxia often is severe, postural reactions and muscular strength are normal. Application of a blindfold frequently exacerbates the signs.
- Facial nerve paralysis may occur in association with peripheral vestibular disease owing to its proximity to the vestibulocochlear nerve within the petrous temporal bone.
- Otitis media/interna can induce bony proliferation of the tympanic bulla and proximal stylohyoid bones that result in fusion of the temporohyoid joint. With progression, acute stress fracture of the petrous temporal bone, and signs of peripheral vestibular disease may occur.
- In *central vestibular disease* (e.g., involving vestibular nuclei and related tracts, most commonly as a result of inflammatory disease or space-occupying mass), the nystagmus can be horizontal, vertical, or rotatory, which may change with changing head position. Although

the fast phase of the nystagmus usually is directed away from the lesion, this is not a consistent finding. In addition, central vestibular disorders frequently cause generalized proprioceptive deficits, mild paresis, signs of depression, and other cranial nerve signs, in particular palsy of the trigeminal and abducens nerves.
- Central or peripheral vestibular disease also may produce strabismus.
- Although rare, horses affected with bilateral peripheral vestibular disease exhibit symmetric ataxia with wide swaying motions of the head and trunk and do not demonstrate other typical signs of vestibular dysfunction.

KEY POINT

Certain cerebellomedullary lesions can produce paradoxical vestibular signs with a head tilt and ataxia opposite the lesion.

- Differentiation between peripheral and central vestibular disease largely is based on the history, physical, and neurologic findings.
- Radiographic examination of the skull should be performed. Ventrodorsal, lateral, and lateral oblique views taken with the horse under general anaesthesia are essential to establish a diagnosis of osteoarthropathy (e.g., proliferation, sclerosis, joint fusion, and fractures) of the temporohyoid joint. Petrous temporal bone fractures often are difficult to identify radiographically, whereas basisphenoid fractures usually are easily recognized.
- If available, bone scintigraphy may allow for an immediate identification of early lesions of the petrous temporal bone in cases in which radiography is inconclusive.
- Endoscopic visualization of the affected temporohyoid joint within the guttural pouch also may be useful.
- Because of accumulated debris within the long, narrow, external auditory canal, otoscopy frequently is difficult in the conscious horse. Adequate visualization of the tympanic membrane usually requires general anesthesia and complete cleaning of the canal. Additionally, tympanocentesis (see "Headshakers") can be performed via the otoscopic earpiece as an aid in the diagnosis of otitis media/interna.
- Analysis of CSF can be helpful in the diagnosis of inflammatory or traumatic diseases and to rule out certain infectious causes (e.g., EPM).
- Idiopathic vestibular syndrome is diagnosed by ruling out other possible causes of vestibular dysfunction.

DIFFERENTIAL DIAGNOSIS

Trigeminal Nerve

Motor Branch

- EPM
- Botulism
- Nigropallidal encephalomalacia
- Neuritis of the cauda equina
- Masseter muscle myopathy (nutritional myodegeneration)

Sensory Branch

- Trauma
- Rabies
- Brain abscess
- Verminous encephalitis
- EPM
- Leukoencephalomalacia
- Neoplasia

Facial Nerve

- Peripheral facial nerve trauma
- Cranial trauma
- Otitis media/interna
- Osteoarthrosis and fractures of the temporohyoid region
- EPM
- Verminous myelitis
- Neuritis of the cauda equina
- Brain abscess
- Viral encephalitides
- Neoplasia

Vestibulocochlear Nerve

- Cranial trauma (e.g., with fracture of the petrous temporal bone or basioccipital and basisphenoid bones)
- Otitis media/interna
- Temporohyoid osteoarthropathy
- EPM
- Verminous myelitis
- Neuritis of the cauda equina
- Viral encephalitides
- Hepatoencephalopathy
- Lightning strike
- Neoplasia
- Spontaneous idiopathic vestibular syndrome

TREATMENT

Trigeminal Nerve

- There is no specific therapy for trigeminal palsy. Treatment for multifocal neurologic diseases that secondarily affect cranial nerve V is warranted (see p. 558). Anti-inflammatory therapy using flunixin meglumine (1.1 mg/kg IV q12-24h; Treatment No. 52) and DMSO (1.0 g/kg

slowly IV as a 10-20% solution ql2-24h; Treatment No. 34) may be beneficial.

Facial Nerve

- If the nerve has been severed in a peripheral location, there is some potential therapeutic benefit obtained from rejoining the severed ends of the nerve. A proportion of the damaged fibers may regrow to their respective muscles of facial expression in 6 to 12 months, although complete return of function is rare.
- In most cases peripheral nerve injury results from contusion and stretching without breakage in the skin or severance of the nerve. As a result, anti-inflammatory therapy such as flunixin meglumine (0.6-1.1 mg/kg IV q12-24h for up to 5 days) and/or DMSO (1.0 g/kg slowly IV as a 10-20% solution q24h) may provide benefit. If the insult is short-lived and there is loss of function only, recovery within 14 days can occur. Prognosis for return of normal function when axons are lost as a result of injury generally is poorer, although partial return to function may occur within 6 to 12 months. Palsy due to central lesions (e.g., equine protozoal myeloencephalitis) has a less favorable prognosis.

Vestibular Nerve

- Therapy for cranial trauma or skull fracture following temporohyoid osteoarthropathy involves reduction in intracranial pressure and cerebral edema using anti-inflammatory agents (e.g., dexamethasone and flunixin meglumine), osmotic (e.g., DMSO and mannitol) and renal (e.g., furosemide) diuretics, and hypertonic saline solutions (see Cranial Trauma). Glucocorticosteroids should be used with caution if an infectious process or disruption of the BBB is suspected.
- Osteoarthropathy of the temperohyoid region may respond to administration of antibiotics such as trimethoprim-sulfadiazine (15-30 mg/kg of the combination PO q12h; Treatment No. 107) for at least 30 days. Nonsteroidal anti-inflammatory therapy using phenylbutazone (2.2-4.4 mg/kg PO q12h; Treatment No. 88) also can alleviate inflammation.
- In cases with concurrent facial nerve paralysis, corneal ulceration secondary to decreased tear production may necessitate treatment with topical artificial tears.
- Idiopathic vestibular syndrome tends to resolve spontaneously irrespective of treatment.

KEY POINT

Signs of peripheral vestibular disease (and to a lesser degree signs of central vestibular disease) may rapidly improve 2 to 3 weeks after onset owing to visual and central compensation. However, residual vestibular or facial nerve signs may persist.

Cranial Trauma (Head Trauma)

Cranial trauma occurs in horses as a consequence of kicks, collision with an immovable object, penetrating wounds, or rearing over backward. These incidents can result in fractures either of the frontal and parietal bones of the calvarium or the basisphenoid, basioccipital, and temporal bones. The latter bones may be fractured when horses fall over backward and strike the poll.

KEY POINT

Although the degree of neurologic dysfunction depends on type and extent of injury, initially this may not be readily determined. This is because of adrenaline or autonomic domination, which for approximately 30 to 120 minutes overrides normal sensory perception and reflex activity and makes neurologic evaluation virtually meaningless.

Therefore, recovery from severe neurologic deficits (e.g., concussion leading to coma) may occur. However, it also is possible that progression and deterioration of mild clinical signs may occur (e.g., as a consequence of ongoing hemorrhage and edema within a closed calvarium), with the prognosis subsequently becoming grave.

Regardless of the cause of the initial trauma, subsequent damage to the brain involves a rapid and overlapping cycle of events, including membrane and microvascular disruption, reduced cerebral blood flow, tissue hypoxia, cerebral edema and hemorrhage, and increased intracranial pressure. High intracranial pressure within the rigid and closed calvarium perpetuates this cycle with further nervous tissue compression and destruction. If persistent or severe enough, herniation of the brain laterally or caudally can occur.

Spinal cord trauma often is due to many of the same causative and pathophysiologic mechanisms as cranial trauma and is discussed elsewhere in this section.

HISTORY AND PRESENTING SIGNS

- All ages are affected
- History of trauma (e.g., rearing over backward, being kicked, hit by a car, fall in a race)
- Neurologic signs will depend on the site and severity of the lesion and vary from compulsive

wandering, seizures, and blindness to weakness, head tilt, circling, hemorrhage from the nostrils and ears to recumbency and loss of consciousness
- Evidence of skin contusions, skull fracture, and other fractures involving mandibular, maxillary, orbital, periorbital, and long bones

CLINICAL FINDINGS AND DIAGNOSIS

- Clinical signs depend on the site of the injury and degree of concussion, contusion, laceration, and hemorrhage.
- Penetrating wounds, fracture fragments, or indications of blunt trauma may be evident. A general physical examination in addition to a neurologic examination is essential to identify concurrent cardiorespiratory and musculoskeletal injuries.

KEY POINT

Many horses with cerebral or brainstem damage initially are presented recumbent in a state of semicoma or coma. Generally, if there is nonpenetrating cranial trauma, most semicomatose recumbent horses or those that regain consciousness will be able to stand in time (minutes to hours). A full neurologic examination at this point will provide more accurate information about the severity and location of neurologic dysfunction.

- *Cerebral dysfunction*, from frontal bone trauma and fracture, commonly results in changes in behavior (e.g., signs of depression, stupor, and seizures) and gait (e.g., compulsive wandering in circles toward the side of the affected cerebral hemisphere and weakness). Blindness with normal PLR and decreased facial sensation on the side opposite the lesion frequently is noted. Development of asymmetric pupils or slowly responsive mydriatic pupils and more profound signs of depression indicates increasing intracranial pressure and edema with midbrain involvement. Loss of consciousness and development of mydriatic unresponsive pupils indicates that the cerebrum has herniated caudally.
- *Brainstem dysfunction* (e.g., from compression and hemorrhage) usually is the result of basisphenoid fractures that occur at the suture between the basisphenoid and basioccipital bones. The basilar artery and venous sinuses often are lacerated by basisphenoid fractures, producing massive hemorrhage into the calvarium, guttural pouch, or middle and inner ear. Along with hemorrhage into the middle and inner ear cavities, petrous temporal bone fracture also may destroy the middle and inner ear. These latter injuries result in *peripheral or central vestibular dysfunction* and are the most commonly encountered neurologic syndromes after cranial trauma.
- Neurologic signs associated with brainstem and vestibular dysfunction include signs of depression, tetraparesis and ataxia, cranial nerve deficits, and signs of vestibular dysfunction (e.g., head tilt and circling toward the side of the lesion, nystagmus with the fast phase directed away from the lesion). Facial nerve palsy characterized by ipsilateral ear droop, ptosis, flaccidity of the lower lip, and muzzle deviation to the contralateral side also may occur.
- Hemorrhage or leakage of CSF from the ear suggests petrous temporal bone fracture. Nasal hemorrhage also may result from fracture of the cribriform plate or from basisphenoid fractures with subsequent hemorrhage into the guttural pouch.
- Severe brainstem injuries can be associated with erratic breathing and dilated and unresponsive pupils, indicating a grave prognosis.

KEY POINT

Bilateral blindness with mydriatic unresponsive pupils also may result from shearing damage or deceleration/acceleration forces to the optic nerve or optic chiasm after a traumatic poll or frontal injury. Although the prognosis for recovery of vision is poor, other neurologic signs may not be observed.

- Diagnosis usually is based on history, circumstantial evidence, and clinical signs. Radiographs are helpful to identify fractures, although nondistracted fractures (e.g., those of the basisphenoid or basioccipital bones) and fractures of the petrous temporal bone may be extremely difficult to detect on radiographs.
- Although alterations in CSF consistent with cranial trauma include xanthochromia, high red cell count, high protein concentration, and increased creatine kinase activity, CSF analysis provides minimal definitive diagnostic or prognostic information.

KEY POINT

Collection of CSF from the AO space is contraindicated if increased intracranial pressure is suspected because of the risk of caudal brain herniation. However, samples collected from the LS space, although safer, may be normal because the sample is not obtained from a site close to the lesion.

- If available, CT also can provide additional important information in horses with head trauma.

DIFFERENTIAL DIAGNOSIS

- Viral encephalitides
- Hepatoencephalopathy
- Verminous encephalitis
- Equine herpesvirus myeloencephalopathy
- Bacterial meningitis/brain abscess
- Hypocalcemia
- Tetanus
- Botulism
- Septicemia (foals)
- Benign idiopathic seizure syndrome (Arabian foals)
- Congenital malformations (foals)

TREATMENT

- In mild cases, stall rest and restriction of movement may result in full recovery. Mostly, aggressive medical therapy is necessary.
- Immediate care of horses with cranial trauma consists of maintaining a patent airway, circulatory support, controlling hemorrhage, and stabilizing any fractures. Sedation of a struggling or violent horse often is required to facilitate examination and treatment. However, use of phenothiazine tranquilizers (e.g., acepromazine) should be avoided because of increased risk of seizures and potential for elevation in intracranial pressure. Xylazine (0.1 mg/kg IV) can be administered safely to horses preferably with the head maintained in an elevated posture above the level of the heart.

KEY POINT

Reduction in intracranial pressure and cerebral edema can be attempted using anti-inflammatory agents, osmotic and renal diuretics, and hypertonic saline solutions. Treatment within 8 hours of injury to reduce edema formation results in a more favorable outcome than later intervention.

- Dexamethasone (0.1-0.2 mg/kg IV q6-8h for 1-4 days; Treatment Nos. 29 and 30) is the initial drug of choice in all horses with cranial (and spinal cord) trauma to control inflammation and to prevent lipid peroxidation and neural edema. Clinical improvement may be observed within 4 to 8 hours. Horses should be monitored closely because of increased risk for development of steroid-induced laminitis.
- Administration of nonsteroidal anti-inflammatory drugs such as flunixin meglumine (1.1 mg/kg IV q24h; Treatment No. 52) may assist in reducing pain associated with fractures, but these drugs have limited efficacy in reducing central nervous system edema.
- Slow intravenous infusion of DMSO (1 g/kg in 0.9% sodium chloride as a 10-20% solution, q12-24h for 3 days; Treatment No. 34) may provide valuable anti-inflammatory, free radical scavenging, and hyperosmotic properties. However, recent research has questioned the purported positive effects of DMSO in animals with neurologic disease.
- If the horse is initially in a coma and recumbent or there is no response to the above therapy or deterioration in signs occurs over the first 4 to 6 hours, more aggressive therapy using osmotic diuretic agents is indicated to reduce intracranial pressure and edema.
- Mannitol (0.25-1 g/kg as a 20% solution IV; Treatment No. 68) may be administered over 20 to 30 minutes and repeated every 4 to 6 hours for 24 hours if there is neurologic improvement after its initial use.
- Although renal diuretics such as furosemide used alone probably are not as effective as glucocorticosteroids and mannitol, furosemide (1 mg/kg bolus IV once followed by 0.5 mg/kg/h for 4 hours; Treatment No. 54) used in conjunction with mannitol can increase the duration of intracranial pressure reduction provided by mannitol.
- In addition, hypertonic saline (4-6 mL/kg of 5-7% sodium chloride solution IV over 15 minutes) given to horses in shock subsequent to head trauma significantly lowers intracranial pressure and cerebral water content and provides adequate cardiovascular support. Glycerol (0.5-2.0 mg/kg IV q6-12h for 24 hours) also may be effective.

KEY POINT

Although use of osmotic substances is warranted in horses with grave or worsening neurologic signs, contraindications to their use include suspected ongoing intracranial hemorrhage, dehydration, hypotension, renal failure, hyperkalemic periodic paralysis, hypernatremia, and hypothermia. It is important that horses receiving osmotic agents should be adequately hydrated or be placed on concurrent intravenous fluid therapy.

- *Anticonvulsant therapy* includes diazepam (5 mg IV in foals, 25-100 mg IV in adults; Treatment No. 32) (Table 14-5) repeated as necessary

TABLE 14-5. Doses and Routes of Administration of Drugs for the Control of Seizures in Horses and Foals

Drug	Dose for Acute Control of Seizure Activity	Maintenance Dose
Diazepam	0.05-0.2 mg/kg IV as required	Not indicated
Xylazine	0.5-2.0 mg/kg IV as required	Not indicated
Pentobarbital	5-20 mg/kg calculated dose; administer to effect	Not indicated
Chloral hydrate	25-100 mg/kg IV *{Note:* Irritant perivascularly)	Not indicated
Phenobarbital	5-20 mg/kg in 25 mL saline slowly IV as a loading dose	4-10 mg/kg PO q8-12h
Phenytoin	1-5 mg/kg IV or PO q4h for up to 24 h	1-5 mg/kg PO q12h

to control intermittent generalized seizures or phenobarbital (5-10 mg/kg IV; Treatment No. 88) (see Table 14—4) for uncontrollable generalized seizures unresponsive to diazepam. Barbiturates also may protect against ischemic damage by reducing cerebral metabolism, retarding lipid peroxidation, and decreasing intracranial pressure. For prolonged control of generalized seizures, phenobarbital (10 mg/kg ql2h PO) is effective.

Adequate *nursing care,* nutritional support, and maintenance of fluid and electrolyte balance are important. Recumbent horses also require padding at pressure points and frequent positional change with the head and neck elevated above the body to reduce intracranial pressure.

Other factors that may impede successful treatment include infections (e.g., aspiration pneumonia, bacterial meningitis), bladder paralysis, and cystitis. Broad-spectrum antimicrobial drugs (e.g., third-generation cephalosporins, such as ceftiofur) are indicated in horses suspected of basisphenoid, petrous temporal bone, and/or open frontal bone fractures.

In horses with open cranial or depressed frontal bone fractures or if progression of neurologic signs occurs despite medical therapy, emergency *craniotomy* to allow decompression and removal of bone fragments may be necessary. In these cases, referral of the horse to a specialist facility for appropriate surgery should be considered; although in many instances, euthanasia is the only alternative.

KEY POINT

With frequent repeated neurologic examination to monitor progression of neurologic signs, intensive nursing care, judicious medical therapy, and time, some horses recover uneventfully despite profound clinical manifestations soon after the trauma. In general, response to therapy in 6 to 8 hours indicates a favorable prognosis for life.

- In other cases, neurologic signs progress or significant complications arise, resulting in death or the necessity for euthanasia. Some horses improve initially, but residual deficits remain. For example, permanent blindness can occur as a result of damage to the optic nerve roots.

"Headshakers"

Headshaking or head tossing is a frustrating and poorly understood condition in which a horse exhibits sudden, excessive, and occasionally violent rotatory, horizontal, or vertical head movements during exercise or at rest in the absence of external stimuli. In severe cases, horses become so distracted that they may be impossible to ride.

KEY POINT

Although recognition of headshaking is relatively easy, determining the underlying cause of the behavior frequently is difficult and unrewarding. The vast majority probably occur as a result of an imbalance of the autonomic and sensory innervation to the nasal passages resulting in sensations akin to those felt at the onset of sneezing.

Recently, headshaking related to seasonal exposure to sunlight (photic headshaking) and similar to photic sneezing in humans has been described.

HISTORY AND PRESENTING SIGNS

- No breed or sex predilection
- Mostly observed in adult horses when exercising
- Sometimes occurs seasonally (i.e., spring and early summer) or only in sunlight
- Intermittent ear rubbing

- Resentment to placing of a bit and manipulation of head
- Difficulty eating or chewing with dropping of food

CLINICAL FINDINGS AND DIAGNOSIS

- Side-to-side or up-and-down head motions or intermittent jerky or rotational movements are noted either during ridden exercise, lunging, or at rest. In many horses, sneezing, snorting, and nose rubbing often preceded by lip and nasal twitching accompany headshaking.
- Other clinical signs are related to a specific cause (see differential diagnosis), although this may not be readily determined in many cases.

KEY POINT

Examination of horses with headshaking should include a complete physical, ophthalmic, otoscopic, neurologic, and dental evaluation. As well, endoscopy of the nasal passages, ethmoid region, pharynx and guttural pouches, and radiography of the skull and cervical spine should be performed. Unfortunately, even if traumatic, inflammatory, or degenerative processes are identified, proving a causal relationship to the headshaking often is difficult.

- Particular attention to examination of the ear canal may reveal presence of exudate, parasites, or inflammation. Also, digital pressure applied to the base of each ear may aid in detection of sensitivity or pain suggestive of inflammation or temporohyoid osteoarthropathy.
- Direct endoscopic examination of the temporohyoid joint within the guttural pouch can be a sensitive early procedure to detect osseous changes at the joint associated with chronic otitis media or interna.
- Endoscopy of the upper airway also may reveal left laryngeal neuropathy, which has been reported to be associated with headshaking.
- Ventrodorsal, lateral, and lateral oblique radiographs of the skull also may be diagnostic in cases of headshaking that are associated with otitis media/interna and bony changes of the petrous temporal and stylohyoid bones.
- Radiographs are best performed under general anesthesia with care being taken to mark left and right sides. Abnormal findings include increased density of the tympanic bullae and bulbous enlargement around the temporohyoid joint and proximal part of the stylohyoid bone.
- Tympanocentesis under general anesthesia also should be considered if radiographs are inconclusive. After thorough cleaning of the external auditory meatus (preferably before anesthesia), a 6-inch spinal needle preloaded with a 3-mL syringe containing 0.75 mL sterile water is introduced carefully through the tympanic membrane via a long presterilized otoscopic ear piece. After the sterile water is injected, fluid is aspirated 10 to 15 seconds later and submitted for bacterial culture and cytologic analysis. A normal middle ear aspirate should be clear, acellular, and should contain no protein or pathologic bacteria or fungi.
- Intradermal skin testing and serum radioallergosorbent testing (RAST) or enzyme-linked immunosorbent assay (ELISA) testing have been used to define environmental allergies in horses and may be of benefit to determine a possible allergic role in horses with headshaking. However, the results must be interpreted carefully in view of the many limitations of these tests.
- If no obvious physical causes of headshaking are identified and if horses shake their heads only when exposed to sunlight, evaluation for photic headshaking should be undertaken. Horses are blindfolded or placed in a darkened environment to determine if the behavior ceases. Bilateral infraorbital nerve blocks also may alleviate the condition if it is associated with irritation to the nares, lip, or cheek. Anesthetizing the infraorbital nerve within the infraorbital canal desensitizes the upper lip, cheek, nostril, teeth, alveoli, and gums to the level of the second premolar.

DIFFERENTIAL DIAGNOSIS

- Ill-fitting bit and tack
- Otitis media/interna
- Temporohyoid osteoarthropathy
- Ear mite *(Psoroptes* spp.) infestation
- Harvest mite *(Trombicula autumnalis)* larval infestation
- Oral diseases (e.g., maxillary osteomas, malocclusion, dental periapical osteitis)
- Ocular diseases (e.g., detached melanotic iris cysts)
- Guttural pouch mycosis
- Allergic vasomotor rhinitis
- Ethmoidal hematoma
- Other nasal, cheek, pharyngeal, or guttural pouch disease
- AO bursitis
- Photic headshaking secondary to trigeminal nerve irritation

TREATMENT

- Any underlying medical abnormalities (e.g., mites, guttural pouch mycosis, ethmoid hematoma, oral disease) should be treated appropriately. Muzzle and ear nets to decrease irritation from small airborne foreign bodies may be useful. Also, nebulized pharmacologic agents (e.g., xylometazoline or betamethasone) and hyposensitization may be used in cases of allergic rhinitis.
- In horses in which headshaking is associated with otitis media/interna with or without temporohyoid osteoarthropathy, broad-spectrum antibiotics (e.g., penicillin and aminoglycoside combinations, potentiated sulfonamides, or second- or third-generation cephalosporins) should be used for up to 1 month along with nonsteroidal anti-inflammatory drugs to reduce pain. However, natural fusion of the arthritic temporohyoid appears to offer the best means for alleviation of pain and headshaking.
- Horses that are suspected of being photic headshakers usually can be successfully managed using cyproheptidine (0.3-0.6 mg/kg q12h PO) with relief from headshaking observed within 24 hours. Ideally, therapy should continue until the season during which headshaking occurs is over.
- Altering the photic biorhythym with melatonin (12 mg q24h PO) also has been successful in horses with photic headshaking.
- Although bilateral infraorbital neurectomy can eliminate headshaking in some cases, this procedure should be reserved as a salvage procedure in horses that fail to repond to cyproheptidine but consistently improve after bilateral infraorbital nerve blocks.

Hepatoencephalopathy

Hepatoencephalopathy is a disorder of cerebral function resulting in dementia, head pressing, and altered behavior in horses as a consequence of hepatic dysfunction. The signs, causes, and management of hepatoencephalopathy are discussed in the section describing liver diseases in Chapter 7.

Leukoencephalomalacia (Mycotoxic Encephalomalacia, "Moldy Corn" Poisoning, "Blind Staggers")

Equine leukoencephalomalacia (LEM) is a multifocal neurologic disorder resulting from ingestion of cereal grains (in particular corn) contaminated by mycotoxins produced by *Fusarium* spp. field molds. The disease has been reported to occur as a result of ingestion of both fumonisin B_1 toxin produced by *Fusarium moniliforme* and fumonisin B_2 toxin produced by *Fusarium proliferatum.* In horses, the fumonisins cause disruption of endothelial cell membranes, principally those of the neurologic and hepatic systems. LEM is recognized throughout the world and frequently occurs as sporadic outbreaks in particular during late autumn to early spring.

HISTORY AND PRESENTING SIGNS

- Usually there is a history of repeated ingestion of corn over several weeks rather than a single exposure
- Invasion by *Fusarium* occurs mostly when corn has been stressed during the growing season by heat, drought, excessive rain, or some combination of these factors
- Often 25% to 100% of horses in a herd are affected
- Sudden death is common
- Signs of depression, inappetence, and separation from the herd followed by other behavioral changes
- Swelling of the lips and muzzle
- Signs of colic

CLINICAL FINDINGS AND DIAGNOSIS

- Ingestion of contaminated corn may result in mutifocal neurologic and/or hepatic disease.

KEY POINT

The course of the disease appears to depend on the concentration of fumonisin in the feed, the amount of feed consumed, and the tolerance of the individual horse to the mycotoxin.

- However, the relationship between dose and severity of clinical disease has not been established and not all corn contains toxic concentrations of fumonisins.
- In addition to clinical findings noted above, neurologic signs progress to asymmetric cranial nerve deficits, mania, head pressing, and circling. Ataxia and weakness followed by recumbency, coma, or seizures also can occur.
- Individual epizootics characterized by high prevalence of blindness, colic associated with duodenitis/proximal jejunitis, or pharyngeal paralysis have been reported.
- Clinical signs indicating hepatic involvement include icterus, petechiae of the oral mucous membranes, and swelling of the lips or muzzle.

- Although horses with signs of neurologic and/or hepatic dysfunction frequently die within hours, most horses succumb to the disease over several days. If an animal survives, it will usually have some permanent cerebral deficits.

KEY POINT

The activity of liver-specific enzymes in the plasma normally is elevated in horses with the neurologic and hepatotoxic forms of the disease.

- Other nonspecific clinicopathologic abnormalities include prerenal induced elevations in serum creatinine concentrations and an increase in hemoglobin and packed cell volume (PCV). In some cases there is a decrease in PCV. Neutrophilic leukocytosis or mild lymphopenic leukopenia also may occur.
- Analysis of the CSF provides variable results depending on the extent and site of the lesion. In most cases, nonspecific increases in the number of neutrophils and total protein concentrations occur sometimes accompanied by xanthochromia. Increase in myelin basic protein (>14 ng/mL) also may support a diagnosis of LEM.

KEY POINT

Antemortem diagnosis of LEM is based on history, clinical signs, laboratory data (altered CSF in particular), and the presence of large numbers of spores of¥. moniliforme *in corn-based feed samples. Definitive diagnosis requires isolation of fumonisin toxins (>5 ppm) from suspected feed samples and demonstration of a characteristic liquefactive necrosis of the cerebral hemispheres, brainstem, cerebellum, and spinal cord at postmortem.*

DIFFERENTIAL DIAGNOSIS

- Trauma
- Viral encephalitides (e.g., EEE, WEE, VEE, rabies)
- Equine herpesvirus myeloencephalopathy
- Equine protozoal myeloencephalitis
- Hepatoencephalopathy (i.e., secondary to pyrrolidine alkaloid or other toxicoxis, infection, or neoplasia of the liver)
- Theiler's disease
- Bacterial meningitis/brain abscess
- Verminous encephalitis
- Botulism
- Neuritis of the cauda equina

TREATMENT

- There is no specific treatment for LEM. Although supportive therapy may be helpful, most animals die or are euthanized.
- Provision of shelter, water, and palatable nutritious feeds is important. Suspected contaminated feed sources should be removed and animals removed to an uncontaminated pasture.
- Aggressive management of inflammation and edema may aid recovery in acute cases. Flunixin meglumine (1.1 mg/kg IV q24h; Treatment No. 52) or phenylbutazone (4.4 mg/kg PO q24h; Treatment No. 89) can be used for their anti-inflammatory effects. Empirically, dexamethasone (0.1-0.2 mg/kg IV q24h for 3-4 days; Treatment Nos. 29 and 30) also is of value. Mannitol (0.25 mg/kg IV as a 20% solution q4-6h for 1 day; Treatment No. 68) may help decrease brain edema, whereas DMSO (1 g/kg slowly IV as a 10-20% solution q12-24h for several days; Treatment No. 34) also may be useful in decreasing clinical signs.
- Other treatments include thiamine (5 g IV ql2h), gastrointestinal protectants such as activated charcoal, and laxatives to aid elimination of toxic substances and balanced intravenous fluids.
- The risk of toxicity is higher if broken, shriveled, or discolored corn is fed to horses. As well, corn screenings should never be fed.

Narcolepsy (Fainting Disease)

Narcolepsy is a rare central nervous system disorder characterized by repeated and uncontrolled episodes of sudden onset of skeletal muscle weakness and atonia (often referred to as cataplexy) and abnormal sleep-like activity.

KEY POINT

A biochemical abnormality of the sleep-wake centers of the brainstem, which involve decreased concentrations of serotonin, dopamine, and norepinephrine, may be responsible for the condition.

HISTORY AND PRESENTING SIGNS

- Onset of signs usually before 1 year of age but can occur in adults
- History of muscle weakness or collapse
- Signs may be initiated when the horse is stimulated (e.g., feeding, return to herd mates, stroking of the head and neck, and various treatment procedures)

- May be familial in some Suffolk and Shetland pony foals

CLINICAL FINDINGS AND DIAGNOSIS

- Clinical manifestations can be variable, with episodes being infrequent (weeks apart) or relatively constant (every 5 to 10 minutes).
- During episodes, there can be mild muscle weakness through to collapse with loss of spinal reflexes and sleep characterized by rapid eye movement. Respiratory and cardiac function is normal during the attack.
- Adult horses often buckle at the knees and stumble and if forced to walk, may be ataxic.
- Horses can be aroused from the attack with varying degrees of difficulty. Most recover or rise quietly. Signs last from seconds to minutes.

KEY POINT

Between episodes, affected horses are normal.

- Diagnosis is based on the history, clinical manifestations, and absence of underlying systemic or metabolic abnormality. Hematologic, biochemical, and CSF analysis generally are normal.
- The anticholinesterase drug, physostigmine salicylate (0.06-0.08 mg/kg slowly IV; Antilirium, Forest, Maryland Heights, MO) can be administered to horses suspected of narcolepsy to induce an attack in less than 15 minutes. Untoward affects, including colic, bradycardia, and bronchospasm may be observed following administration of physostigmine.
- In severely affected horses, a decrease in the severity of signs minutes after administration of atropine sulfate (0.04 to 0.08 mg/kg IV; Treatment No. 11) may support the diagnosis. Atropine also can prevent recurrence of attacks for 3.5 to 30 hours.

DIFFERENTIAL DIAGNOSIS

- Cranial trauma
- Hyperkalemic periodic paralysis
- Exertional rhabdomyolysis
- Cardiovascular causes of syncope
- Seizures
- Hypocalcemia
- Restraint of neonates leading to a narcoleptic-like state
- Botulism (foals)
- Hypoglycemia (foals)
- Septicemia (foals)
- Anaphylaxis
- Snake envenomation

TREATMENT

Treatment of narcolepsy often is unrewarding. The antidepressant drug, imipramine (Tofranil, Geigy Pharmaceuticals, Summit, NJ) at a dose rate of 0.55 mg/kg IV or 1.5 mg/kg PO q8h blocks the uptake of seratonin and norepinephrine and reduces REM sleep and may be of value in some narcoleptic horses.

KEY POINT

The prognosis for this disorder varies enormously among individual cases. Some foals have several episodes and then appear to recover spontaneously. In Shetland and Suffolk ponies and in adults of other breeds, signs may persist for life.

Nigropallidal Encephalomalacia ("Yellow Star Thistle Poisoning")

Nigropallidal encephalomalacia is a fatal disease of horses caused by toxins in yellow star thistle (yellow burr) or Russian knapweed. These plants grow predominantly in western United States and Australia and when ingested result in toxic necrosis specifically of the substantia nigra and globus pallidus of the basal nuclei.

HISTORY AND PRESENTING SIGNS

- All ages, breeds, and both sexes affected
- Access to pasture or hay containing *Centaurea solstitialis* (yellow star thistle or yellow burr) or *C. repens* (Russian knapweed) for at least several weeks. Horses tend to develop a desire to selectively eat these plants
- Sudden onset of difficulty with prehension, eating, and chewing and subsequent weight loss

CLINICAL FINDINGS AND DIAGNOSIS

- Affected horses have an acute onset of dysfunction of the muscles of mastication characterized by rigidity and fasciculation. Horses cannot move their mouths normally or chew food. Some horses also show behavioral abnormalities accompanied by aimless wandering and ataxia.
- We have seen several cases with pronounced facial and lip edema.
- Swallowing reflexes are normal and passage of a stomach tube can be achieved.
- Clinical pathology tests are unremarkable, though there may be evidence of dehydration.
- The diagnosis is based on signalment, history, and neurologic findings.

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DIFFERENTIAL DIAGNOSIS

- Botulism
- Guttural pouch mycosis
- Viral encephalitides
- Hepatoencephalopathy
- Bacterial meningitis/brain abscess
- Verminous encephalitis
- Equine protozoal myeloencephalitis
- Masseter myopathy (nutritional myodegeneration)
- Neuritis of the cauda equina
- Trigeminal nerve palsy
- Bilateral facial nerve or hypoglossal nerve paralysis

TREATMENT

There is no specific treatment for this disorder. Affected horses can be maintained with feeding through a nasogastric tube but usually die from starvation.

Parasitic Thromboembolism

Parasitic thromboembolism of the central nervous system occurs as a result of thromboembolic showers originating from thromboarteritis lesions induced by *Strongylus vulgaris* larvae.

KEY POINT

Fourth or fifth stage larvae also may directly cause inflammation and necrosis during aberrant migration through the brain and spinal cord. Larvae probably emerge from verminous thrombi attached to the endocardium, aorta, carotid, or vertebral arteries and are transported to the ipsilateral cerebrum.

HISTORY AND PRESENTING SIGNS

- Any age, sex, or breed, though young horses (<3 years old) are more frequently affected
- Acute onset of behavioral changes, blindness, signs of depression, seizures, and possibly recumbency

CLINICAL FINDINGS AND DIAGNOSIS

- Signs are often asymmetrical. In horses that are standing, blindness, aimless wandering, pacing, and circling to the affected side can occur. Belligerent behavior or depression also may be features. Mild ataxia is common. Death can ensue.

KEY POINT

Severity of signs varies with number and size of the parasites and with location and extent of lesions.

- In many cases, signs do not progress and may improve over time.
- Diagnosis is difficult and often speculative. A minimum database should include a complete blood count, biochemical profile, urinalysis, and CSF analysis to eliminate other possible diagnostic differentials.
- In parasitic thromboembolism secondary to *S. vulgaris*, hematologic and biochemical findings usually are nonspecific while CSF xanthochromia, mildly increased protein, and total nucleated cell counts with or without eosinophilia can be expected.

DIFFERENTIAL DIAGNOSIS

- Cranial trauma
- Hepatoencephalopathy
- Viral encephalitides
- Rabies
- Bacterial meningitis/brain abscess
- Verminous encephalitis
- Leukoencephalomalacia
- Equine herpesvirus myeloencephalopathy
- Equine protozoal myeloencephalitis
- Neuritis of the cauda equina
- Neoplasia
- Equine degenerative myeloencephalopathy

TREATMENT

- Since a definitive diagnosis rarely is made, treatment is empirical and supportive.
- In acute neurologic disease, anti-inflammatory therapy is advised. Good choices include flunixin meglumine (1.1 mg/kg IV q24h; Treatment No. 52) or phenylbutazone (4.4 mg/kg PO q24h; Treatment No. 88). Dexamethasone (0.1-0.2 mg/kg IV q24h for 3-4 days; Treatment Nos. 29 and 30) also can provide benefit. However, if equine protozoal myeloencephalitis is strongly suspected, glucocorticosteroids *should not* be administered. Intravenous administration of mannitol (0.25 mg/kg IV as a 20% solution q4-6h for 1 day; Treatment No. 68) and/or DMSO (1.0 g/kg slowly IV as a 10-20% solution q12-24h for 1 to 3 days; Treatment No. 34) may be valuable because increased intracranial pressure is likely.
- If it is assumed that migrating parasites are still present, administration of fenbendazole (50 mg/kg PO q24h for 3 days) is indicated. Although

ivermectin (0.2 mg/kg PO once; Treatment No. 62) has good efficacy against *S. vulgaris*, it is not recommended in treating parasite migration because the drug may take up to 2 weeks to kill larvae.
- Seizures may be controlled using diazepam (25 to 100 mg IV, repeated as necessary). (See also next section.)
- Good supportive care is important, including provision of shelter, water, and palatable, nutritious feeds. Thiamine, vitamin E, and selenium also may be administered to support nervous tissue function.
- The prognosis must always be guarded. Cases with mild deficits may survive or even return to normal function. In more severely affected or recumbent patients, euthanasia should be considered.

Seizures (Fits, Convulsions)

Seizures are due to abnormal electrical discharges in the cerebrum that result in involuntary alterations of consciousness, autonomic functions, and motor activity of the entire body or any body part. In adult horses, seizures more frequently occur in conjunction with other signs of brain disease.

KEY POINT

Severe damage must occur to the forebrain to precipitate seizures in adult horses. In contrast, foals have a lower seizure threshold and are more susceptible to conditions causing seizures.

Seizure activity is manifested clinically in two forms:

- A *partial seizure* involves a discrete area of the cerebral cortex and results in localized signs such as asymmetric facial or limb twitching, excessive chewing, compulsive running, or self-mutilation. Cervical myelography, anesthesia, cranial trauma, and certain metabolic (e.g., uremia) and toxic (e.g., metaldehyde) causes commonly result in partial seizures, some of which may develop into generalized seizures.
- A *generalized seizure* involves the entire cerebral cortex and usually begins with signs of anxiety and restlessness ("aura") lasting minutes to hours, followed by recumbency, loss of consciousness, and generalized, symmetric clonic-tonic muscle contractions. Horses also may demonstrate strabismus, mydriasis, ptyalism, jaw clamping, thrashing of limbs, opisthotonus, and urinary and fecal incontinence. The seizure phase may last from 5 to 60 seconds, and after the seizure, horses may show signs of depression and temporary blindness for minutes to days. Generalized seizures rarely occur in rapid succession *(status epilepticus).*

KEY POINT

Generalized seizures are the most common form of seizures observed in adult horses and foals.

A variety of congenital, metabolic, infectious, toxic, traumatic, vascular, or neoplastic factors may cause seizures. The most common causes of seizures in foals under 2 weeks of age are neonatal sepsis, CNS hypoxia, and ischemia (neonatal maladjustment syndrome), trauma, and bacterial meningitis. The most common causes of seizures in foals less than 1 year of age include trauma, benign epilepsy in Arabian foals, toxicities (e.g., organophosphates [OP]), and viral encephalitides. The most common causes of seizures in adult horses older than 1 year are cranial trauma, hepatoencephalopathy, toxicities (e.g., OP, moldy corn, rye grass, heavy metals), metabolic disorders (hypocalcemia, hyperlipemia), EPM, and viral encephalitides.

KEY POINT

Diagnosis of the cause of the seizure is based on signalment (in particular, age and breed), a thorough history and neurologic examination, and ancillary diagnostic tests (e.g., CSF analysis, serology, radiography, toxicology, and if available electrodiagnostics and computed tomography). Importantly, other conditions that can mimic seizures such as colic, exertional myopathy, long bone fractures, vestibular disease, hyperkalemic periodic paralysis, syncope, and narcolepsy should be ruled out.

As most causes of seizures are presented elsewhere in this chapter, a few selected disorders are described below:

Benign Epilepsy of Arabian Foals

A seizure disorder in foals, particularly Arabians, that is present in the first year of life. The clinical manifestations normally abate by the time the foal reaches adulthood.

Inadvertent Intracarotid Injection

Inadvertent administration of substances into the carotid artery when attempting intravenous injection is a common cause of a single convulsive episode.

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Organophosphate Toxicity

Exposure of horses to toxic doses of these anticholinesterase preparations commonly results in seizure activity. Toxicity is quite common because these agents are used widely for medicinal purposes in horses and have a low therapeutic index. Anticholinesterase compounds also are used commonly for agricultural purposes, posing the risk of unintended exposure of horses to toxic amounts of the preparations.

HISTORY AND PRESENTING SIGNS

Benign Epilepsy of Arabian Foals

- Foals less than 12 months old
- Intermittent violent convulsions
- Unexplained head injuries

Inadvertent Intracarotid Injection

- Attempt to administer drugs into the jugular vein
- The horse moved or jumped during injection
- Behavior change followed by blindness, recumbency, and convulsions

Organophosphate Toxicity

- All ages
- Recent exposure to these agents topically, via the gastrointestinal tract, or possibly from agricultural sources
- Evidence of weakness, tremors, colic, increased salivation

CLINICAL FINDINGS AND DIAGNOSIS

Benign Epilepsy of Arabian Foals

- When seizure activity is not present, foals are normal on neurologic examination. Some foals will have evidence of trauma as a result of the seizures, including head and eye injuries and contusions of the gums and skin.
- During convulsions, foals become unaware of their environment, recumbent, and convulse, sometimes quite violently. Subsequent to the episode, affected foals are depressed and may show signs of cerebral dysfunction similar to those occurring with neonatal maladjustment syndrome. Signs include wandering aimlessly, failure to drink, inability to recognize the dam, depression, head pressing, and blindness.
- There are no specific clinicopathologic findings associated with this disease.

KEY POINT

Diagnosis is based on the age and breed of the foal and history or evidence of signs and by ruling out other causes of seizure activity.

Inadvertent Intracarotid Injection

- Injection of substances into the carotid artery may result in a range of effects, depending on the volume and type of agent administered.
- On many occasions, no change in behavior or a short period of apparent agitation may ensue.
- In more severe cases, there is a brief period of apparent anxiety, possibly some muscle twitching, particularly on the face, followed by recumbency and convulsive activity. Horses often paddle and appear quite violent. After a variable period (seconds to minutes), signs become less intense. Many horses regain their feet, but remain blind and show signs of depression and apparent disorientation that may last for days. In some cases, permanent, irreversible effects including death occur.
- In most circumstances, water-based sedatives (e.g., xylazine) are inadvertently administered into the carotid artery. These agents tend to be associated with the fewest residual effects. In some cases, damage to the recurrent laryngeal nerve may result in subsequent laryngeal hemiplegia.
- Diagnosis is based on the knowledge of an injection into the carotid artery or on the dramatic results following an injection into what was thought to be the jugular vein.

Organophosphate Toxicity

- Clinical manifestations include nystagmus, tremor, salivation, patchy sweating, dyspnea, colic, diarrhea, muscle weakness, ataxia, and occasionally seizures.
- Diagnosis is based on a history of exposure to one of the chemicals in this group, the clinical signs, and results of laboratory analysis. Determination of acetylcholinesterase activity in red blood cells or plasma helps support the diagnosis.

DIFFERENTIAL DIAGNOSIS

Benign Epilepsy of Arabian Foals

- Trauma
- Neonatal maladjustment syndrome
- Septicemia
- Bacterial meningitis/brain abscess
- Viral encephalitides
- Hepatoencephalopathy
- Equine herpesvirus myeloencephalopathy
- Severe hyponatremia (neonates)
- Hyperkalemic periodic paralysis (older foals)
- Heat stroke

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Inadvertent Intracarotid Injection

- Trauma
- Anaphylaxis
- Hyperkalemic periodic paralysis

Organophosphate Toxicity

- Trauma
- Viral encephalitides
- Hepatoencephalopathy
- Verminous encephalitis
- Equine herpesvirus myeloencephalopathy
- Hyperkalemic periodic paralysis
- Bacterial meningitis/brain abscess
- Colic
- Respiratory disease

TREATMENT

Doses of drugs commonly used for the treatment of seizures are provided in Table 14-5. Diazepam and/or phenobarbital sodium is the best initial treatment for seizures in adult horses and foals. Glucocorticosteroids usually in combination with DMSO may also provide benefit.

Apart from initial control of an acute generalized seizure episode, anticonvulsant therapy is indicated when several multiple seizures occur over 1 to 3 days, if one seizure occurs every 2 months, or when clusters of seizures occur more than 3 to 4 times per year. Maintenance therapy should only be instituted after careful owner counseling. An affected horse is unsafe to ride until it has been seizure-free for 6 months without medication. If used, maintenance therapy should be decreased slowly to determine if continued treatment is necessary.

KEY POINT

The phenothiazine-derivative tranquilizers (e.g., acepromazine) are contraindicated because they reduce the seizure threshold or may worsen current seizures. Because ketamine can increase cerebral blood flow and intracranial pressure, which may worsen seizures, this drug also is contraindicated. Although xylazine may increase intracranial pressure, it may be necessary to administer xylazine in an emergency until a more appropriate anticonvulsant becomes available.

Benign Epilepsy of Arabian Foals

- Many foals with mild signs and infrequent convulsive episodes probably do not require specific therapy. In those that are more severely affected, maintenance anticonvulsant therapy is indicated.

KEY POINT

We have found phenobarbital to be useful for this purpose (20 mg/kg IV as a loading dose followed by maintenance doses of 4-10 mg/kg PO q12h; Treatment No. 87).

- Therapy is continued for 4 to 12 weeks, and then the foal is weaned from the drug over 10 to 14 days. If signs recur, reinstitution of therapy is indicated.
- Symptomatic therapy for head injuries and corneal ulcers is indicated.
- In the majority of foals, convulsive activity abates by the time they are 12 months old.

Inadvertent Intracarotid Injection

- In general, no treatment is necessary. If the seizure activity is prolonged, administration of diazepam (0.05-0.5 mg/kg IV as required; Treatment No. 32) is useful.
- In horses with residual effects, treatment with flunixin meglumine (1.1 mg/kg IV q24h; Treatment No. 52) and DMSO (1.0 g/kg slowly IV as a 10-20% solution q12-24h for 1-3 days; Treatment No. 34) may be of benefit.

Organophosphate Toxicity

- If seizures are present, treatment with diazepam (0.05-0.5 mg/kg IV as required; Treatment No. 32) is indicated. Atropine (0.05-0.5 mg/kg IV or SC every 60 minutes 0.01-0.1 mg/kg until salivation is decreased and mydriasis occurs; Treatment No. 11) is helpful in controlling symptoms. **Note: Large doses of atropine will cause ileus.** Administration of pralidoxime chloride (2-PAM; 20 mg/kg initial dose IV followed by similar or lesser doses q4-6h as required; Protopam Chloride, Wyeth-Ayerst, Philadelphia, PA) also is useful.
- Intravenous fluids to assist in promoting diuresis and removal of free drug from the circulation can be of value (see Chapter 18). Other supportive therapy may be necessary.
- If the drug has been absorbed from the skin, washing the horse with shampoo and water is necessary. Similarly, if the route of absorption is the gastrointestinal tract, administration of dioctyl sodium sulfosuccinate (10-20 mg/kg PO as a 5% solution in 4-8 L water q48h; Treatment No. 35) helps promote expulsion of the toxic agent from the tract.

Diseases of the Spinal Cord

Cervical Stenotic Myelopathy ("Wobbler" Syndrome, Cervical Vertebral Malformation)

Cervical stenotic myelopathy (CSM) results from abnormal growth and/or articulation of the cervi-

cal vertebral bodies, resulting in vertebral canal narrowing and focal compression of the spinal cord. This compression can be constant regardless of cervical position, referred to as *static compression*, and occurs predominantly in the caudal cervical region, C5-6 and C6-7. Otherwise, spinal cord compression may be intermittent only occurring when there is movement, particularly flexion or extension of the neck. This is referred to as *dynamic compression* or instability and most commonly affects intervertebral sites C3-4 and C4-5.

Recent studies suggest that in general, there is no difference in site distribution of lesions between horses less than or equal to 2 years of age and those greater than 2 years of age.

KEY POINT

Although osteochondrosis often occurs in the vertebral column (and at other body sites) of affected horses, no causal relationship between osteochondrosis and CSM has been established. However, both conditions are developmental orthopedic diseases caused by multiple factors including overnutrition, mineral imbalances, rapid growth, trauma, a genetic predisposition, and abnormal biomechanical forces.

HISTORY AND PRESENTING SIGNS

- Young horses between 1 and 3 years of age usually are affected.
- Predominantly Thoroughbreds and quarter horses, although members of most breeds have been affected.
- Males more frequently affected than females.
- Horses usually rapidly growing and are well grown for their age.
- Dietary rations may be low in copper, high in zinc, and contain excessive quantities of carbohydrate.
- History may include poor performance, stumbling, falling down, or an obscure lameness.

CLINICAL FINDINGS AND DIAGNOSIS

- Ataxia and weakness are the most common clinical findings. Signs usually occur acutely, often associated with a fall that exacerbates mild spinal cord compression. In general, the hindlimbs are more severely affected (at least one grade worse) than in the forelimbs.
- Neurologic signs most commonly are symmetric but can be more severe on one side if compression is greater on that side.
- Stumbling, toe dragging, and delayed proprioceptive positioning are accentuated by manipulative procedures (e.g., circling, walking up and down a slope, with and without the head being elevated, backing, and the "sway" and "tail-pull" reactions).
- Progression of signs often is variable, with some horses appearing to worsen and then stabilize, whereas others improve, followed by recurrence of signs.
- In general, there are no obvious abnormalities detectable in the cervical vertebral column on physical examination. Rarely, there are localized alterations in pain perception, muscle atrophy of cervical musculature, sweating, and hyporeflexia of cervical reflexes adjacent to the affected cervical vertebrae. These signs more commonly are noted in horses over 4 years of age and probably occur as a result of peripheral nerve compression by proliferative articular processes as nerve roots exit the vertebral canal at C5 to C7.

KEY POINT

Young horses with CSM also may acquire concurrent signs of developmental orthopedic disease of the appendicular skeleton such as joint effusion secondary to osteochondrosis, physeal enlargement of long bones, and flexural limb deformities.

- Diagnosis is based on clinical signs and results of CSF analysis and radiographs.
- Plain lateral radiographs of the cervical vertebrae often demonstrate characteristic malformations, including (1) "ski-jump" enlargement of the caudal epiphysis of the vertebral body, (2) abnormal ossification of the articular processes, (3) malalignment between adjacent vertebrae, (4) caudal extension of the dorsal laminae, and (5) degenerative joint disease of the articular processes. However, these subjective radiographic findings only support the diagnosis of CSM and do not reliably distinguish between CSM-affected and unaffected horses.

KEY POINT

Objective assessment of vertebral canal diameter using the sagittal ratio technique is *more accurate than subjective radiographic evaluation of bony malformation for identification of horses with CSM.*

- Using precise lateral radiographs of the cervical vertebrae, sagittal ratio is calculated for each cervical vertebra by dividing the *minimum sagit-*

tal diameter (MSD) by the width of the vertebral body. The MSD is obtained by measuring the narrowest sagittal diameter of the vertebral canal. The vertebral body width is measured perpendicular to the vertebral canal at the widest point of the cranial aspect of the vertebral body.

- If the sagittal ratio is below 0.501 when measured at C4 to C6 or less than 0.521 at C7, then the likelihood of the presence of CSM in a horse with spinal ataxia is high and myelographic examination should be performed to confirm the diagnosis. If the sagittal ratio is above 0.56 at C4 to C6 and greater than 0.58 at C7, myelographic examination should be avoided until diagnostic tests focused on alternative causes of spinal ataxia are performed.
- The sagittal ratio technique has a sensitivity and specificity of ~89% for vertebral sites C4 to C7 in horses more than 320 kg in weight.
- For Thoroughbred foals up to 1 year of age (<320 kg) suspected of CSM, a semiquantitative scoring system for assessment of cervical radiographs also has been developed. Incorporating the five characteristic malformations found on cervical radiographs described above with determination of vertebral canal stenosis, a score is designated for each abnormality. Stenosis of the veterbral canal is evaluated by dividing the intervertebral MSD (smallest sagittal canal diameter of the cranial aspect of a vertebra) and intravertebral MSD (smallest sagittal canal diameter of the caudal aspect of an adjacent vertebra) by the length of the corresponding vertebral body. A total score of 12 or higher constitutes a radiographic diagnosis of CSM.

KEY POINT

Horses with CSM have generalized stenosis of the vertebral canal between C3 and C5, regardless of the site of spinal cord compression. Compression can occur from stenosis of the vertebral canal at vertebral sites that lack bony malformation. Therefore, confirmation of the diagnosis can only be made by the demonstration of a 50% or greater reduction in the sagittal diameter of opposing dorsal and ventral dye columns by positive contrast myelography.

- This procedure is relatively complex, requiring general anesthesia, and it provides some risk to the patient. In our experience, these cases are best handled by skilled veterinarians in well-equipped referral facilities. Indications for myelographic examination include identification of the exact site(s) of spinal cord compression before surgical intervention, insurance purposes, and confirmation of a definitive diagnosis of CSM at the owner's request.
- Cytologic analysis and protein concentration of CSF generally is within reference limits, although mild xanthochromia or mild elevations in protein concentration may be found. Evaluation of CSF for antibody to herpesvirus myeloencephalitis and immunoblot analysis for EPM also should be determined.

DIFFERENTIAL DIAGNOSIS

- Spinal cord trauma
- Vertebral fracture or luxation
- Equine degenerative myeloencephalopathy
- Equine herpesvirus myeloencephalopathy
- Equine protozoal myeloencephalitis
- AO malformation
- Neuritis of the cauda equina
- Vertebral osteomyelitis
- Verminous myelitis
- Rabies
- Other congenital abnormality

TREATMENT

- Transient improvement in clinical signs of acute cases of CSM can be observed with conservative medical management designed to decrease pain and inflammation. Corticosteroids such as dexamethasone (0.1-0.2 mg/kg IV q12h for 1-2 days; Treatment Nos. 29 and 30) or prednisolone (1-2 mg/kg PO q12h for several days; Treatment No. 93) commonly are prescribed to reduce inflammation. Phenylbutazone (Treatment No. 89) or flunixin meglumine (Treatment No. 52) also may be used for their anti-inflammatory effects. Slow intravenous administration of DMSO (1 g/kg of a 10-20% solution q12-24h for 1-2 days; Treatment No. 34) also appears to be of value in cases with recent acute onset of signs.
- Foals less than 1 year of age with clinical signs of CSM or foals with radiographic evidence only of CSM may be successfully managed with specific dietary management and stall confinement. In general, diets should provide restricted protein and energy (65-75% of National Research Council [NRC] recommendations), balanced quantities of vitamins and minerals with extra vitamin A and E and selenium, and low-quality roughage (6-9% crude protein).
- Although some older horses with CSM respond to prolonged rest (2-12 months), most will not return to neurologic normality with rest or anti-inflammatory therapy alone, and surgical treat-

ment directed at preventing further spinal cord compression and damage should be considered.

KEY POINT

Surgery to stabilize the vertebral column by fusion of two or more cervical vertebrae and/or decompression of the spinal cord by subtotal dorsal laminectomy is performed in a number of surgical referral facilities.

- Although interpretation of the results of these procedures tends to be controversial, reported rates of neurologic improvement range from 44% to 90%, with 12% to 62% of horses returning to athletic function. The shorter the duration of clinical signs before surgical intervention, the better the prognosis and likelihood of a return to function.
- Euthanasia should be considered in horses that are severely affected and whose clinical signs make them a danger to themselves or to their handlers.

Occipitoatlantoaxial Malformations

Congenital malformations involving the occipital bones, atlas, and axis in horses are relatively rare, but when they occur, they may result in signs of brainstem or spinal cord compression. Because of this, clinical signs have varied from none to a foal being dead at birth. The most common neurologic deficits involve ataxia and weakness affecting all limbs.

HISTORY AND PRESENTING SIGNS

- Young horses (<6 months old)
- Most common signs relate to progressive spinal cord ataxia and tetraparesis
- Occurs in all breeds occasionally
- Familial predisposition for occipitoatlantoaxial malformation in Arabian horses

CLINICAL FINDINGS AND DIAGNOSIS

KEY POINT

Progressive ataxia and weakness usually affecting all limbs are the most common manifestations of occipitoatlantoaxial malformation and may be present at birth or become evident within the first few weeks to months of life.

- In some cases, spinal cord compression is sufficiently severe that foals are found dead or recumbent and unable to rise.
- Palpation of the AO articulation in affected foals may reveal swelling, malarticulation, and a reduction in the amount of movement of that joint. In Arabian foals with occipitoatlantoaxial malformation, the transverse processes are often small and abnormally shaped. Crepitus or a "clicking" sound may be demonstrable when the head and neck are manipulated.
- Diagnosis is based on presenting signs and clinical findings. Radiographs are often helpful in establishing the diagnosis. In Arabian foals with occipitoatlantoaxial malformation, there is AO fusion, hypoplasia of the atlas and dens, malformation of the axis, and modification of the atlantoaxial joint.

DIFFERENTIAL DIAGNOSIS

- Trauma
- Cervical stenotic myelopathy
- Equine herpesvirus myeloencephalopathy
- Bacterial meningitis
- Viral encephalitides
- Rabies
- Other congenital abnormalities

TREATMENT

- Mild cases may require no therapy, although progression of the signs is likely as the foal grows.
- Surgical stabilization and/or decompression of the affected site is reported in some cases, particularly those occurring in non-Arabian breeds. However, under most circumstances, treatment is conservative, and if the signs are severe, euthanasia should be recommended.

KEY POINT

Because the disease has a genetic basis in Arabian horses, parents of known affected animals should not be bred to each other or to related animals.

Spinal Cord Trauma

Trauma to the spinal cord of the horse probably is the most frequently encountered acute neurologic problem in equine practice.

KEY POINT

Mechanisms for spinal cord damage are similar to those that affect the brain after trauma and involve a dynamic process of hemorrhage, inflammation, edema, and

hypoxic ischemia. Although neurologic signs usually are noted immediately after injury, signs may occur weeks to months later as a result of slowly progressive pathologic damage to the spinal cord caused by instability, arthritis, or callus formation.

The most common sites for spinal cord injury from vertebral fractures and luxations are the occipitoatlantoaxial region, the caudal cervical region, and the caudal thoracic and lumbar spine. Adult horses are more likely to develop fractures in the caudal cervical region (C5-7) or the thoracolumbar spine as a result of high-energy low-velocity injuries. In contrast, foals and young horses tend to suffer fractures of epiphyseal separations in the cranial cervical and caudal thoracic areas. Subluxations and luxations of the occipitoatlantoaxial joints and fracture of the dens also are common. These may occur when young horses are tied up and pull back on the lead rope against a fixed object (hyperextension) or by somersaulting (hyperflexion).

HISTORY AND PRESENTING SIGNS

- Horses of all ages and any breed, particularly those that are easily frightened or in foals that are being handled.
- History of a fall, collision with a stationary object, or penetrating injury.
- Acute onset of reluctance to move, incoordination, weakness, or recumbency.

CLINICAL FINDINGS AND DIAGNOSIS

KEY POINT

Variable signs of ataxia, paresis (tetra-, para-, or hemi-), and dysmetria are common depending on the degree and site of neuronal insult. With increasing compression of the spinal cord, loss of proprioception and motor weakness will occur before loss of perception or response to touch and loss of pain perception.

- If the lesion is in the cervical region, all limbs will be affected (i.e., tetraparesis to tetraplegia and ataxia). Horses with cervical fractures usually are in pain and resist neck manipulation. Foals with atlantoaxial subluxation frequently have a stiff neck with audible crepitation when manipulated and head deviation.
- Only the hindlimbs are affected (i.e., paraparesis to paraplegia [dog-sitting] and ataxia) if the lesion is between T1 and S2. The first three thoracic vertebrae are most likely to be involved, followed by fractures of T12.
- Sacral fractures (usually S2) result in minimal abnormalities in pelvic limb gait, but urinary and fecal incontinence, and loss of tail and anal function usually occur.
- In some cases deficits are quite symmetric, whereas in others they are asymmetric.
- Neurologic deficits are usually exacerbated in response to manipulative tests, including circling, walking up and down a slope, and backing (see neurologic examination). If the lesion is severe, local gray matter and sympathetic trunks can be damaged, leading to patchy sweating and localized loss of pain sensation. If the signs persist, muscle atrophy may result and may be evident by 10 to 14 days postinjury.
- Recumbent horses require particular attention to detail when performing the neurologic examination if the site of the lesion is to be correctly identified (see previous section on neurologic examination of the recumbent horse).

KEY POINT

Interpretation of neurologic findings in recumbent horses, immediately and up to 2 hours after a fall, can be difficult due to profound sympathetic or autonomic tone.

- In addition to history and careful neurologic examination, which localizes deficits to a particular spinal cord segment, diagnosis of trauma can be aided by CSF analyses and radiography.
- Analysis of CSF may reveal few abnormalities, even in cases where there is a severe traumatic myelopathy. There may be evidence of hemorrhage into the subarachnoid space, reflected by xanthochromia and an increase in red blood cells in the CSF. Hemorrhage is followed by an increase in the number of nucleated cells (neutrophils and macrophages) and mild increases in total protein concentrations.
- Radiographic examination may identify cervical, sacral, and thoracic fractures but not lumbar vertebral fractures. However, it must be remembered that radiographs only provide images of tissues in two dimensions and fractures without displacement may show no abnormalities. To optimize the likelihood of demonstrating lesions, lateral and dorsoventral or ventrodorsal views should be taken whenever possible.

KEY POINT

Nuclear scintigraphy also may enable identification of nondisplaced fractures, whereas CT, if available, can be helpful in

identifying the nature and extent of vertebral and spinal cord lesions.

DIFFERENTIAL DIAGNOSIS

- Cervical stenotic myelopathy
- Equine protozoal myeloencephalitis
- Equine herpesvirus myeloencephalopathy
- Viral encephalitides
- Hepatoencephalopathy
- Vertebral osteomyelitis
- Bacterial meningitis
- Rabies
- Brain abscess
- Congenital abnormalities

TREATMENT

- Principles of therapy are similar to those described for cranial trauma with aggressive attention to reducing hemorrhage, edema, and inflammation. If necessary, sedation with diazepam (0.05-0.1 mg/kg IV) or xylazine (0.1 mg/kg IV) to effect may assist in restraint.

KEY POINT

In mild cases, stall rest, restriction of movement, and judicious anti-inflammatory therapy (e.g., glucocorticosteroid and nonsteroidal anti-inflammatory drugs) may be all that is necessary.

- In addition to inactivity, medical therapy involves administration of glucocorticosteroids such as dexamethasone (0.1-0.2 mg/kg IV q6-8h for 1 or 2 days; Treatment Nos. 29 and 30). If there is no response to this therapy or deterioration in signs occurs over the first 4 to 6 hours, more aggressive therapy using the osmotic diuretic agent mannitol (0.25-1 g/kg as a 20% solution IV administered over 20-30 minutes; Treatment No. 68) is valuable to reduce swelling in the central nervous system. If there is improvement in signs, therapy with mannitol can be continued every 6 to 12 hours for up to 48 hours. Care should be exercised to ensure that the horse does not become dehydrated. An alternate or additional treatment to mannitol involves slow intravenous infusion of DMSO (1 g/kg in 0.9% sodium chloride as a 10-20% solution, ql2-24h for 3 days; Treatment No. 34).
- Nonsteroidal ant-inflammatory agents such as flunixin meglumine or phenylbutazone may be useful to decrease inflammation and pain, although low doses are recommended to ensure the horse remains inactive and continues to "splint" the damaged region.
- Although surgical stabilization of vertebral fractures rarely is attempted in adults, several surgical approaches are available for correction of atlantoaxial subluxation and unstable cervical fractures in foals. Consideration also should be given to referral to a specialist facility for appropriate surgical decompression and stabilization in horses that respond poorly to medical treatment.
- Rehabilitation also is assisted with early attention to nutritional support and controlled exercise. In recumbent horses, feeding via stomach tube may be necessary, whereas passive flexion and extension of all limbs, frequent turning, massage, and hydrotherapy help combat necrosis and muscle atrophy.

KEY POINT

Repeated neurologic examinations and response to therapy are the best guide to prognosis. Horses that show rapid neurologic improvement have a good prognosis, although it may take weeks to months for complete recovery.

- Recumbent horses, horses suffering from fractures or luxations, or horses that have lost deep pain sensation have a poor-to-grave prognosis.

Tetanus ("Lockjaw")

Tetanus is caused by an exotoxin liberated from the bacterium *Clostridium tetani*. The bacterium is a common inhabitant of the intestinal tract of animals, and spores from this organism are capable of persisting in the soil for many years.

KEY POINT

Horses are the most susceptible domestic animal species to the tetanus exotoxin.

Horses frequently are exposed to the infective form of the bacteria due to their predilection for acquiring wounds and hoof injuries. Spores are deposited into a deep, penetrating wound in which devitalized tissue provides a suitable (anaerobic) environment for growth of the organism and release of potent exotoxin. The most important of the three components of the tetanus exotoxin (tetanospasmin, tetanolysin, and nonspasmogenic toxin) is tetanospasmin, which prevents the release of the inhibitory neurotransmitters glycine and γ-aminobutyric acid (GABA) at inhibitory interneurons in the spinal cord and brain. Also, tetano-

spasmin has additional actions on the neuromuscular junction and autonomic ganglia. Consequently, clinical signs are characterized by potentiation of normal sensory stimuli, resulting in generalized, constant muscular spasticity, hyperesthesia, and convulsions leading to respiratory arrest and death.

KEY POINT

Exotoxin enters the axons of the nearest motor nerves and migrates by retrograde transport to inhibitory interneurones of the central nervous system. Toxin also may be carried hematogenously from wounds to nerves at distant sites, in particular the head. Therefore, the disease can present as a localized progressive illness (e.g., signs of tetanus starting in a limb or in the head) or as a more generalized syndrome.

HISTORY AND PRESENTING SIGNS

- No gender age or breed predisposition
- Frequently, there is *no* history of vaccination
- History of puncture wounds (e.g., lower limb laceration, subsolar abscess of the foot) up to 3 weeks prior to onset of clinical signs
- Stiff gait and spasms of muscles particularly of head and neck

CLINICAL FINDINGS AND DIAGNOSIS

- The incubation period ranges from 3 days to 3 weeks although clinical signs usually become apparent within 7 to 14 days following entry of the organism.

KEY POINT

The most consistently observed initial signs are hyperesthesia with a general increase in muscle stiffness and spasm and prolapse of the third eyelid.

- The masseter muscle is most commonly affected with progression to muscles of the neck, trunk, and limbs. Horses show restriction of jaw movements, erect carriage of the ears, flared nostrils, elevated head, and an unsteady, stiff straddling gait.
- The tail is often held out stiffly, particularly when the horse is required to back or turn.
- Tachycardia, tachypnea, sweating, and fever can be concurrent with severe muscle rigidity.
- The horse is reluctant to eat from the ground, and saliva may drool from the mouth. Dysphagia is common.
- Many of the clinical signs are exaggerated by external stimuli. As the disease progresses, mastication is prevented by tetany, hence the name "lockjaw."
- Colic, constipation, and urine retention are common because horses cannot posture to void feces or urine. Eventually, the tetany becomes sufficiently severe that the horse assumes a "sawhorse" posture and has great difficulty walking. Some horses may fall and their limbs remain in tetany.
- Terminally, opisthotonus is marked, and there may be convulsions. Death occurs due to asphyxiation as a result of tetany of the respiratory muscles.

KEY POINT

The course of clinical disease usually is 5 to 10 days, although signs may persist for weeks in some horses.

Diagnosis is based on the wound history and clinical signs. Horses that have not been vaccinated and have characteristic clinical signs can be assumed to have tetanus. Unfortunately, location of the site of infection is extremely difficult, isolation of the organism almost impossible, and there is no readily available diagnostic test that detects the presence of toxin.

DIFFERENTIAL DIAGNOSIS

- Laminitis
- Exertional rhabdomyolysis
- Pleuropneumonia
- Hypocalcemia
- Heat stroke

TREATMENT

KEY POINT

The aims of therapy include (1) elimination of causative organisms, (2) neutralization of residual toxin, (3) control of muscle spasms, and (4) provide nutritional and ventilatory support.

- Elimination of causative bacteria involves local wound treatment (e.g., debridement, drainage, exposure to the atmosphere, infiltration with antitoxin) and administration of penicillin. Sodium or potassium penicillin (20,000 IU IV q6h; Treatment Nos. 84 and 85) or procaine penicillin

(15,000-20,000 IU/kg or 15-20 mg/kg IM q12h; Treatment No. 83) for at least 7 days is indicated. Tetracycline may be substituted in penicillin-sensitive horses.

- Methods for neutralization of residual toxin include vaccination with tetanus toxoid and administration of tetanus antitoxin (TAT). In the latter case, antitoxin (100 to 5000 U/kg IV, IM, or SC once, followed by 5 U/kg q24h for 5 days; Treatment No. 115) will help neutralize any circulating toxin outside the nervous system. However, it will have no effect on toxin already bound within the central nervous system because parenterally administered antisera does not cross the BBB.

KEY POINT

Acute hepatic necrosis (serum hepatitis or Theiler's disease) has been observed in horses 1 to 3 months after administration of TAT. Nevertheless, TAT is indicated in horses with tetanus and in unvaccinated horses with tetanus-prone wounds.

- Although controversial, intrathecal administration of antitoxin may improve recovery rates if given early in the course of the disease before severe muscle rigidity, spasms, and recumbency. TAT instilled directly into the central nerve system evades the BBB and binds unbound toxin. The horse should be placed under general anesthesia. Ketamine should *not* be used as the induction agent. A spinal needle is inserted into the AO space (see Fig. 14-12), and 50 mL of CSF is withdrawn with an equal amount of antitoxin being replaced. We have had quite variable response with this form of therapy. Some horses have shown rapid stabilization (not reversal) of signs following treatment, whereas others have not responded or have developed seizures, persistent recumbency, or laminitis following injection.
- Administration of tranquilizers, sedatives, general anesthesia, or combinations of these accomplish control of neuromuscular derangements. Acepromazine (0.05 mg/kg IV or IM q12h; Treatment No. 1) is effective in controlling mild muscle spasms. For severe seizures and muscle rigidity, diazepam (0.05-0.5 mg/kg IV as necessary) alone or in combination with xylazine (0.5-1.0 mg/kg IV or IM) can be very effective. Methocarbamol, glycerol guaiacolate, chloral hydrate, or pentobarbital sodium rarely are used to achieve muscle relaxation.
- It is imperative to keep the horse in a dark stall and as free from external stimuli as possible.
- Attention to hydration and electrolyte status is important. Administration of fluids via a small nasogastric tube is possible in many cases, although those horses with pharyngeal dysfunction will require intravenous fluids. Manual evacuation of feces and urinary catheterization may be required.
- In cases of respiratory muscle paralysis, continuous ventilatory support and intensive monitoring is required.

KEY POINT

Lack of previous prophylactic vaccination, short incubation time, and rapid progression of signs with recumbency within 24 to 48 hours of onset indicates poor prognosis in horses with tetanus.

- Prognosis for horses with tetanus must always be considered guarded and grave if treatment is not offered. Complications of tetanus include laminitis, aspiration pneumonia, and pleuropneumonia.
- Prevention of tetanus is readily achieved by the administration of tetanus toxoid (see Appendices 4 and 5). A primary dose is given, a second dose 1 month later, and annual boosters thereafter. Pregnant mares should be vaccinated with tetanus toxoid 1 to 3 months prior to parturition to ensure adequate colostral antitetanus antibodies. Foals should receive the toxoid at 3, 4, and 6 months of age, then annually thereafter.
- This vaccine is one of the most effective equine vaccines available, and its use in all domestic horses should be strongly encouraged. Recovery from tetanus *does not* result in life-long immunity and survivors require further yearly immunization.

Vertebral Osteomyelitis

Vertebral osteomyelitis is a rare but life-threatening condition most common in foals and young horses due to hematogenous spread of organisms to the vertebral bodies. In foals, *Salmonella* spp., *Streptococcus* spp., *Staphylococcus* spp., *E. coli*, and *Actinobacillus* spp., and *Rhodococcus. equi* are the most common causative agents whereas in adult horses, the condition usually is the result of infections with *Mycobacterium* spp. or *Brucella* spp.

HISTORY AND PRESENTING SIGNS

- Typically young horses (<6 months old)
- Possible evidence or history of septicemia (e.g., fever, lethargy, and signs of depression)

- Relatively rapid onset of progressively worsening stiffness and incoordination
- Localized neck pain or reluctance to move the neck or flex the back
- Often found recumbent

CLINICAL FINDINGS AND DIAGNOSIS

- Ataxia, tetraparesis or paraparesis, spasticity, and dysmetria indicative of spinal cord compression often are observed suddenly.
- Manipulative procedures such as circling will often result in worsening of the signs. There may be a reluctance to move the neck or flex the back due to pain.

KEY POINT

Diagnosis of vertebral osteomyelitis is based on a combination of the history, clinical signs, and results of ancillary diagnostic tests (e.g., clinical pathology, radiology, and CSF analysis).

- Identification of an inflammatory response on routine hematologic examination (e.g., leukocytosis, hyperfibrinogenemia, anemia of chronic inflammatory disease) also can reflect osteomyelitis.
- Blood culture may be helpful to demonstrate hematogenous spread of bacteria.
- Radiographic signs consist of proliferative new bone formation, demineralization, sclerosis, soft tissue swelling ventral to the vertebral column, and compression fractures.

KEY POINT

Radiographic changes in the vertebral body may not be evident until 2—8 weeks after the onset of clinical signs. If available, nuclear scintigraphy or computed tomography or both can be used to assist localization of lesions in acute cases of vertebral osteomyelitis.

- Results from analysis of the CSF can be variable. Findings frequently are nonspecific with evidence of xanthochromia, increased total nucleated cell count, particularly macrophages, and increased total protein. On rare occasions there is evidence of meningitis, reflected by marked increases in CSF nucleated cell counts, especially neutrophils and total protein.

DIFFERENTIAL DIAGNOSIS

- Trauma
- Septicemia
- Cervical stenotic myelopathy
- Equine herpesvirus myeloencephalopathy
- Occipitoatlantoaxial malformations
- Bacterial meningitis
- Viral encephalitides
- Rabies
- Other congenital abnormalities

TREATMENT

- If an accurate, early diagnosis of vertebral osteomyelitis is made, the disorder may be treatable. Surgical curettage of affected tissue and long-term administration of appropriate antimicrobial agents can be successful in some cases. However, prognosis for vertebral osteomyelitis generally is poor.
- Anti-inflammatory therapy to reduce and limit spinal cord inflammation and necrosis may be valuable early in the disease. Nonsteroidal anti-inflammatory drugs such as flunixin meglumine (1.1 mg/kg IV q24h; Treatment No. 52) or phenylbutazone (2.2-4.4 mg/kg PO q24h; Treatment No. 88) will provide some pain relief. Slow intravenous infusion of DMSO (1 g/kg in 0.9% sodium chloride as a 10-20% solution, ql2-24h for 3 days; Treatment No. 34) may provide valuable anti-inflammatory and free radical scavenging properties.

DISEASES OF THE PERIPHERAL NERVES AND MUSCLES

Botulism ("Shaker Foal" Syndrome, "Forage Poisoning")

Equine botulism, a disease characterized by progressive flaccid paralysis, is caused by potent exotoxins liberated from the anaerobic bacterium *Clostridium botulinum.* This organism most commonly is found as spores, which are widely but unevenly distributed in soils and aquatic environments worldwide.

There are eight immunologically distinct types of *C. botulinum* toxin. In horses, only toxin types A, B, C, and D have been associated with disease. Greater than 85% of cases with botulism in North America are affected by type B toxin, whereas most of the reported outbreaks or cases of disease in Australia and Europe involve type C.

KEY POINT

The incidence of different types of botulism varies greatly between areas with different soil types. Type B strains are encountered most frequently in slightly acidic soil of high

organic content, whereas types C and D are found more commonly in warmer environments.

Botulinum toxin is a neurotoxin that binds irreversibly to presynaptic nerve terminals at peripheral neuromuscular junctions. This blockade prevents the release of acetylcholine and results in flaccid paralysis. Once toxin is bound to neuromuscular junctions, improved muscular function is achieved only by regeneration of new end plates, which may take 4 to 10 days. Three types of botulism occur:

- *Forage poisoning* involves ingestion of preformed toxin present in feedstuffs (e.g., contaminated big bale hay, lucerne or oaten chaff, and occasionally grain) and is most common in adult horses as a result of *C. botulinum* type B or C. Presence of type B toxin in feed usually is caused by direct proliferation of *C. botulinum* and production of toxin in decaying vegetable matter. In contrast, type C and D botulism in horses typically is associated with contamination of feed materials by carcasses of rodents, foxes, rabbits, birds, and other carrion.
- *Toxicoinfectious botulism* or *shaker foal syndrome* usually is attributed to type B toxin produced within the immature gastrointestinal tract of foals after ingestion of spores.
- *Wound botulism* has only been associated with *C. botulinum* type B. The toxin is absorbed into the blood stream after the organism infects a wound (e.g., injection sites, castration, or leg wound or umbilical remnant in foals) and subsequently is disseminated throughout the body including nervous tissues. This is an extremely rare form of the disease and will not be discussed. However, signs are similar to those described for "forage poisoning."

HISTORY AND PRESENTING SIGNS

Forage Poisoning (Ingestion of Preformed Toxin)

- Adult horses
- History of eating high moisture or protein rich feed during the previous 1 to 10 days (in particular improperly ensiled silage and haylage, hay stored in plastic bags, lucerne and oaten chaff, baled hay contaminated with dead or decaying animals)
- Decreased exercise tolerance with paresis
- Slowness to eat and dropping food from mouth
- Signs of colic or depression
- Recumbency

Toxicoinfectious Botulism (Shaker Foal Syndrome)

- Foals 2 weeks to 8 months old
- Progressive paralysis
- Dysphagia
- Recumbency

CLINICAL FINDINGS AND DIAGNOSIS

The overall clinical picture in equine botulism is the same regardless of pathogenesis. The appearance and severity of signs is variable and is dependent on the amount (and to a degree, type) of toxin ingested. Horses that have ingested large amounts of toxin frequently are found dead.

Forage Poisoning (Ingestion of Preformed Toxin)

KEY POINT

Characteristic signs include progressive, symmetric muscular weakness (without ataxia), resulting in a hesitant, stumbling gait; progression to recumbency; respiratory compromise; and death within days of eating contaminated feedstuffs.

- Muscle fasciculations are a common feature. These may be initially observed in the triceps muscles progressing to involve larger muscle groups. Stress, frequently associated with transportation or movement can exacerbate signs.
- Muscle fasciculations and an inability to lift the head are more marked in cases of type C botulism compared with other types of botulism. Persistently low head carriage may cause severe edema of the head and upper airways, resulting in inspiratory stridor.
- Dysphagia and slower eating time (in particular with type C botulism), reduced tongue tone and slow tongue retraction (especially with type B botulism) are characteristic early signs of botulism, typically observed before onset of obvious muscle weakness. In more advanced cases, the tongue often will hang out, and saliva will dribble from the mouth.
- Sluggish pupillary light response is detected usually within 6 to 18 hours after toxin is ingested, followed by prominent mydriasis lasting for several days.
- Urinary and fecal retention is common, and ileus also is noted usually in recumbent horses. In horses affected by type C botulism, an exaggerated expiratory effort with a prolonged abdominal lift may be observed.

KEY POINT

A presumptive diagnosis of equine botulism can be made based on history and clinical signs. Definitive diagnosis can be very difficult, expensive, and frequently unrewarding.

- Hematologic and serum biochemical values in early cases of botulism usually are within reference limits.
- Presumptive laboratory tests for diagnosis of equine botulism include:
  - o Demonstration of preformed toxin in the feed of a suspected case using an enzyme-linked immunosorbent assay (ELISA).
  - o Demonstration of *C. botulinum* spores in gastrointestinal tract (GIT) contents, feces, or feed using selective media (34% of adult horses with botulism will shed spores in the feces).
  - o Detection of a serum antibody response to *C. botulinum* in the recovered equine patient using the ELISA.
- Definitive diagnosis of botulism requires detection of preformed toxin in plasma, serum, GIT contents, or other body tissues (e.g., liver) of supected cases using the mouse bioassay, an ELISA, or polymerase chain reaction (PCR) test.

Toxicoinfectious Botulism (Shaker Foal Syndrome)

KEY POINT

Affected foals have progressive onset of paresis resulting in a staggering gait and muscle tremor.

- These signs progress to recumbency, inability to rise, and eventually death. Dysphagia is common, with milk often seen dribbling from the mouth and/or nose. Secondary aspiration pneumonia is common. Dilated pupils and paralyzed eyelids also are notable features.
- The principles of diagnosis are similar to that described for Forage Poisoning. Diagnosis is usually presumptive and based on the history and clinical findings. Definitive diagnosis requires identification of preformed toxin in serum, plasma, GIT contents, or other body tissues from dead foals. Such attempts are often unrewarding.

KEY POINT

Identification of C. botulinum *spores in the feces also may be rewarding, since approximately 70% of shaker foals shed spores in the feces. There is almost no shedding of spores in normal, uninfected foals.*

DIFFERENTIAL DIAGNOSIS

Forage Poisoning (Ingestion of Preformed Toxin)

- Equine protozoal encephalomyelitis
- Esophageal obstruction
- Trauma
- Bacterial meningitis
- Leukoencephalomalacia
- Nigropallidal encephalomalacia ("yellow star thistle poisoning")
- Equine herpesvirus-1 myeloencephalopathy
- Viral encephalitides
- Nutritional myonecrosis (masseter myopathy)
- Guttural pouch diseases
- Hypocalcemia
- Hyperkalemic periodic paralysis
- Equine motor neuron disease
- Snake envenomation

Toxicoinfectious Botulism (Shaker Foal Syndrome)

- Septicemia
- Trauma
- Tick paralysis *(Ixodes holocyclus)*
- Nutritional myonecrosis
- Esophageal obstruction
- Hypoglycemia

TREATMENT

- The principles of therapy are similar for all forms of botulism and include antitoxin therapy, rest, meticulous nursing, and avoidance of stressors.

KEY POINT

Use of polyvalent botulinum antitoxin (currently only available in the United States), although expensive, markedly reduces mortality. The recommended dose is 200 mL (or 30,000 IU) for a foal and 500 mL (or 70,000 IU) for an adult IV or IM.

- Antitoxin will adsorb free toxin but not that which is already bound to presynaptic nerve receptors. Therefore, antitoxin administration is most useful in horses who are still standing and who are experiencing a slow progression of signs.
- Plasma harvested from horses previously immunized against type B, C, and D toxoids and administered to affected horses early in the

course of the disease may provide high antibody concentrations. Empirical observations in cases in which 500 mL of this plasma has been administered intravenously in suspected (and later confirmed) cases of botulism indicate a distinct beneficial effect.

- Restriction of movement and avoidance of transportation and extremes of temperature is essential to minimize exacerbation of signs.
- Alfalfa gruel (1.5 to 2 kg twice daily), supplemented with adequate amounts of water, may be used to maintain dysphagic horses. As well, we have found a commercially available liquid diet (Osmolite HN, Ross Laboratories, Columbus, OH) to be a valuable calorie source for horses with dysphagia. A useful regimen is to feed 8 mL/kg then 16, 24, and finally 32 mL/kg total daily dose on successive days. The dose is divided and given every 8 hours. The high dose constitutes maintenance calorie requirements for an adult horse. *Note:* Additional water is required for maintenance (total daily fluid intake of 50-75 mL/kg).
- Foals often require administration of mare's milk (200 mL/kg divided into 6 to 10 feedings) or feed gruels and electrolyte-carbohydrate mixtures via nasogastric tube. Parenteral nutrition for foals with botulism has been described, but it is expensive and time-consuming and normally is only reserved for valuable animals. Ranitidine (Treatment No. 100) or sucralfate (Treatment No. 102) or both can be used to prevent gastric ulceration, which occurs frequently in shaker foals.
- Recumbent foals or horses should be frequently turned and fed and if possible maintained in a sternal position. Twice daily catheterization of the bladder is indicated in horses with urinary retention.
- Administration of mineral oil may help relieve constipation and impaction in horses with ileus. Oral magnesium cathartics, neostigmine and 4-aminopyridine should be avoided because they may potentiate neuromuscular weakness.
- In cases of wound botulism, potassium or sodium penicillin (22,000-44,000 IU/kg IV q6h; Treatment Nos. 85 and 86) should be administered in conjunction with surgical debridement.

KEY POINT

Importantly, antibiotics do not eliminate C. botulinum *from the GI tract of shaker foals and those that potentiate neuromuscular weakness (aminoglycosides, tetracyclines, and procaine penicillin) should be avoided.*

- Therefore, potentiated sulfonamides may be used to treat complications such as aspiration pneumonia.
- Mildly affected horses may recover over 1 to 2 weeks, although return of full strength may take more than a month. Rapid progression of clinical signs with recumbency is associated with greater amounts of bound toxin and a grave prognosis.
- In North America, prevention of toxicoinfectious botulism in endemic areas has been undertaken using a *C. botulinum* type B toxoid (Bot Tox-B, ELISA Technology Co, Lexington, KY). Three doses of vaccine, administered at 1-month intervals (or 2-week intervals in outbreak situations) are recommended to immunize horses successfully.
- Annual revaccination of mares 4 to 6 weeks before foaling is highly recommended in endemic areas.
- A toxoid for *C. botulinum* type B is available in Australia for use in mares and foals against shaker foal syndrome. However, currently there is no multivalent vaccine or type C toxoid available for use in Australia or North America.
- Feed contaminated with botulinum toxin should be removed from the diet of other susceptible horses.

Equine Motor Neuron Disease

EMND is a rare neurodegenerative disease of adult horses causing weight loss, weakness, and muscle atrophy. Reports of the disease have so far been confined to the northern hemisphere, particularly northeastern United States and Canada.

KEY POINT

EMND affects the LMNs primarily in the ventral horns of the spinal cord and in selected brainstem nuclei. The most severely affected areas are in the brachial and lumbar intumescences and cranial nerves V, VII, and XII.

In general, the disease is sporadic, affecting only one horse housed in a boarding stable or farm in which there has been limited or no access to pasture for at least a year.

HISTORY AND PRESENTING SIGNS

- Horses of both sexes between 2 and 25 years old
- Although many breeds have been affected, there is higher prevalence in quarter horses and Thoroughbreds kept in boarding stables
- Increased risk when horses are fed a ration high in grain (e.g., pelleted feed) with poor quality grass or cereal hay and no access to pasture

- Coarse trembling of limb muscles or fine muscle fasciculations with increased tendency to lie down
- Weight loss despite normal or increased appetite
- Loss of muscle mass in upper hind and forelimbs and neck
- Apparent weakness when walking and shifting weight in rear legs when standing
- Black dental tartar, in particular of the incisors

CLINICAL FINDINGS AND DIAGNOSIS

- The most consistent early findings in acute cases of EMND are muscle trembling, unexplained weight loss, frequent episodes of sternal recumbency, and standing with all four limbs close together. Severe trembling can become more pronounced if horses are forced to stand in one location for an extended period.
- Common additional findings include constant shifting of weight in the rear legs, abnormally low head carriage, and elevation of the tail head. Ataxia is not observed with EMND, although horses may have a short-strided gait caused by the weakness.
- Subclinical cases may be prevalent because greater than 30% of motor neurons need to become dysfunctional before clinical signs are apparent.
- As the disease progresses, affected horses develop marked symmetric muscle atrophy primarily of the proximal limb muscles (i.e., quadriceps, triceps, and gluteals) and neck. This results from death of motor neurons accompanied by degeneration of ventral roots and peripheral nerves.
- Although lesions occur in the motor nuclei of some cranial nerves, detectable cranial nerve deficits are rare. However, abnormal pigment deposition frequently may be observed in the nontapetal area of the retina on ophthalmoscopic examination.
- Characteristically, the appetite is normal to ravenous with coprophagia noted in 50% of cases. Tail and anal tone are normal.
- In most cases, clinical signs appear to stabilize over 2 to 8 weeks, although a few affected horses rapidly progress to persistent recumbency and respiratory distress necessitating euthanasia. Horses that stabilize either remain in a state where the clinical signs are arrested for many years or signs may slowly progress over ~1 year with continued weakness, emaciation, and development of bizarre gait abnormalities such as stringhalt. Horses with the arrested chronic form of the disease may regain some muscle mass and strength but manifest reduced performance. However, relapse of trembling and weakness invariably occurs, which generally is fatal.

KEY POINT

There is strong evidence that EMND results from neuronal degeneration secondary to oxidative stress created by a deficiency in the antioxidant α-tocopherol (vitamin E). Greater than 90% of horses with the acute form of the disease have abnormally low concentrations of a-tocopherol in plasma (<1.0 μg/mL), nervous tissue, and muscle. Red blood cell superoxide dismutase activity also is abnormally low, whereas serum selenium concentration is normal.

- Moderate elevations in the serum concentrations of the muscle enzymes CK and AST commonly are found in acute cases of EMND. However, these enzymes may return to normal in "arrested" cases.
- CSF analysis reveals increased protein concentration (mostly due to elevations in IgG) and elevated CK activities in approximately 40% of cases.
- Glucose absorption tests but not xylose absorption tests also are abnormal in almost 30% of horses with EMND.
- Electromyographic findings in acute cases (i.e., positive sharp waves or fibrillation responses in affected muscles) are indicative of denervation.
- Biopsy of the sacrocaudalis dorsalis medialis (tail head) muscle usually provides a definitive diagnosis of EMND. Although more difficult, biopsy of the ventral branch of the spinal accessory nerve also can be used in more chronic cases.

DIFFERENTIAL DIAGNOSIS

- Equine protozoal myeloencephalitis
- Polyneuritis equi
- Rhabdomyolysis
- Laminitis
- Botulism
- Organophosphate toxicity
- Myositis/myopathy
- Lead poisoning
- Colic

TREATMENT

- No treatment has proved successful, and in general prognosis is poor for return to function and guarded for life.

- Current recommendations for therapy in horses with EMND is to administer vitamin E either by providing good quality grass or alfalfa hay or by supplementing 5000 to 7000 IU/day of vitamin E orally.
- In acute cases, prednisolone (0.5-1.0 mg/kg q24h PO) may reduce oxidative damage to nervous tissue and improve clinical signs.

Hyperkalemic Periodic Paralysis

KEY POINT

HyPP is an autosomal dominant inherited disorder of horses, genetically and pathologically identical to HyPP in humans. Confirmed cases of HyPP have been restricted to one family of horses, all descendants of the Quarterhorse sire Impressive.

In horses with HyPP, one (heterozygotes) or both (homozygotes) of the genes that determine the function of the sodium ion channel within skeletal muscle have been altered by a point mutation. Failure of normal sodium channel activity causes persistent depolarization of the muscle cell membrane and a temporary loss of muscle function. Excessive flux of sodium into cells and outward flux of potassium results in hyperkalemia.

HyPP has predominantly been identified in quarter horses, other crossbred quarter horses, American Paint horses, and Appaloosas of North America and Australia. However, export of these breeds may result in the identification of HyPP-affected horses worldwide.

HISTORY AND PRESENTING SIGNS

- Mainly observed in young (<4 years old) well-muscled horses
- Males and females equally affected
- High potassium diets (e.g., those based on alfalfa hay) often fed
- Intermittent episodes of muscle fasciculations and trembling over face and body, weakness, sweating, and recumbency, lasting a few minutes to a few hours
- Episodes often occur after exercise and horses appear normal between episodes
- Sudden death may be the only finding in some horses

CLINICAL FINDINGS AND DIAGNOSIS

KEY POINT

Most affected horses are heterozygotes, display few or no clinical signs, and may have a more muscular appearance and show reduced exercise tolerance. Horses that are homozygotes have attacks more frequently and show greater range and severity of signs.

- Stressful situations such as strenuous exercise, transport, anesthesia, sudden changes in diet, or exposure to cold weather may precipitate clinical manifestations of the disorder in heterozygotes and homozygotes. However, onset of signs usually is unpredictable.
- Except for most cases that are homozygotes, clinical signs seldom become apparent before weaning and may become less severe with increasing age.
- Typically attacks begin with facial muscle spasm, yawning, and fine continuous muscle fasciculations prominent over the shoulders, flanks, and neck area. Advanced signs include tachypnea, sweating, intermittent prolapse of the third eyelid, and weakness that may progress to dog sitting or recumbency.
- Recumbent horses appear to have flaccid paralysis, although normal muscle tone is present. Stimulation and attempts to move may worsen muscular fasciculations. Affected horses remain bright and alert during episodes and respond normally to noise and painful stimuli.
- In some cases, inspiratory stertor and drooling occur as a result of paralysis or spasm of the arytenoids, cartilages, or pharynx. Other less common signs include colic, arrhythmias, and bradycardia that are likely to be associated with substantial increases in serum/plasma potassium concentrations. Rarely, horses die during these episodes.
- Attacks in homozygote horses can range in frequency from daily to a few times a year. Inspiratory stertor, dyspnea, and tongue protrusion associated with dysfunction of the muscles of the tongue, pharynx, and larynx more commonly is observed in cases that are homozygote than in those that are heterozygote.
- Frequently, early signs may be missed, and although attacks can stop at any time, most episodes last for 15 to 60 minutes.

KEY POINT

Diagnosis of HyPP usually is based on history, clinical signs, measurement of elevated serum potassium concentration during an attack, and genetic testing for the HyPP gene.

- A positive gene probe test for the HyPP gene using blood collected into EDTA confirms sus-

ceptibility of an individual and whether the horse is heterozygous or homozygous.
- The classical finding of normal serum or plasma potassium concentrations between episodes (3-5 mmol/L or mEq/L) that rise to 7 to 12 mmol/L (mEq/L) during episodic weakness generally is found. However, in some horses, serum or plasma potassium concentration during an episode is within normal reference limits.

KEY POINT

Blood samples for potassium determination should be kept out of direct sunlight and serum or plasma separated within a few hours of collection to avoid spurious elevations in potassium concentrations secondary to erythrocyte leakage.

- Reduction in clinical signs parallels diminution of serum or plasma potassium concentration, which may be the result of renal excretion and/or local temperature elevation of contracting muscle reducing inward flux of cellular sodium and outward flux of potassium.
- Reduced serum or plasma sodium and calcium concentrations and increases in total plasma protein concentration and PCV also occur during clinical episodes in many horses. In addition, serum or plasma CK activity and acid-base balance is unaltered in response to these periods of episodic weakness.

KEY POINT

Since the introduction of genetic testing, electromyography and potassium chloride (KCl) challenge testing are ***no longer relied on*** *to establish a diagnosis. Although rare, risks of fatality associated with KCl challenge testing should be avoided.*

- Electromyography can demonstrate characteristic electrical abnormalities including prolonged insertional activity with trains of doublets, triplets, or high-frequency repetitive discharges. These changes are present between attacks in approximately 90% of affected horses.
- The KCl challenge test has been used to provoke clinical signs of the disorder and high serum potassium concentrations in affected animals. Potassium chloride initially is administered at 100 mg/kg with 1 to 2 L of water by nasogastric tube to horses *after an overnight fast.* The horse is kept off feed and closely monitored over the next 6 hours. If no signs of HyPP are observed, the horse is allowed to rest for a day and the test repeated at incremental doses of 25 mg/kg/day until signs are noted or a dosage of 150 mg/kg (weanlings) or 200 mg/kg (adults) has been administered without signs. Normal horses show no detrimental effects and have only mild increases in serum potassium concentration after challenge.
- Caution should be exercised in using the provocation test, particularly at high dose rates and in horses with cardiac, renal, or impaired adrenocortical function because it may produce life-threatening hyperkalemia in a small percentage of affected horses. However, in most cases, clinical signs can be rapidly reversed by administration of treatment (see below).

DIFFERENTIAL DIAGNOSIS

- Exertional rhabdomyolysis
- Colic
- Tetanus
- Botulism
- Seizures
- Myotonia
- Pheochromocytoma
- Narcolepsy

TREATMENT

- Mild attacks may be managed by hand walking or stall rest with the horses being observed.
- During severe acute episodes, *slow* administration of intravenous calcium gluconate (0.2-0.4 mL/kg [100-200 mL] of a 23% solution diluted in 1-2 L of 5% dextrose) usually results in rapid remission of signs. Calcium acts to antagonize the effects of potassium at the cellular level.
- An alternative treatment is to use an intravenous mixture of 0.9% saline at 0.5 to 1 mL/kg, 1.3% sodium bicarbonate at 0.5 to 1 mL/kg, and 50% dextrose at 0.25 to 0.5 mL/kg. Potassium-free and bicarbonate or glucose-rich fluids encourage renal excretion of potassium and drive potassium intracellularly. Dextrose (4-6 mL/kg IV of a 5% solution) combined with 5% sodium bicarbonate (1 mmol/kg or mEq/kg IV) or calcium gluconate also can be used.
- Other potential treatments that promote intracellular movement of potassium include epinephrine and insulin.

KEY POINT

Horses with severe upper respiratory tract obstruction may require a temporary tracheostomy.

- Long-term management should be directed at decreasing potassium intake in the diet, avoiding

stressful events and sudden environmental changes and increasing renal excretion of potassium.
- Removal of ingredients high in potassium (e.g., alfalfa [lucerne] and some grass hays, canola oil, soybean oil, molasses, and some protein and vitamin supplements); replacing them with oats, timothy or bermuda grass hay, grains, and beet pulp; and ensuring adequate calcium and phosphorus intake are helpful to control attacks. Proprietary low-potassium feeds are available.
- Feeding regularly, two to four times daily, and allowing the horse free access to salt also is helpful in many cases. If possible, the horse should be kept at pasture and allowed to exercise. Rapid changes in diet should be avoided.
- Treatment with potassium-wasting diuretics, acetazolamide (2-4 mg/kg PO q12h; Diamox, Lederle, Pearl River, NY) and hydrochlorothiazide (0.5-1.0 mg/kg PO q12h; Hydrochlorothiazide USP, Rugby, Rockville Center) has been utilized with some success to reduce frequency of attacks in horses that do not respond to dietary management.

KEY POINT

Horses cannot be legally shown when receiving these medications.

- Given the genetic basis of the disease and the availability of a reliable test for the HyPP genotype, owners should be encouraged to ensure that horses from suspected matings and known affected horses are tested and if positive, breeding of these horses should be discouraged.

Hypocalcemia (Lactation Tetany, Transit Tetany, Idiopathic Hypocalcemia, Eclampsia, Hypocalcemic Tetany)

Hypocalcemia is rare in horses and occurs most frequently in lactating mares, in mares in midgestation, in mares within 2 weeks of parturition, or in mares 1 to 2 days after weaning. Hypocalcemic tetany also has been reported in foals and adult horses following GI tract disorders (e.g., pancreatic or hepatic inflammation); renal disease; prolonged transport with food deprivation; following endurance events, particularly during hot weather; or secondary to certain toxicoses such as cantharidin, oxalate, or cadmium.

Total calcium concentration in serum is approximately 50% ionized or active, 40% protein bound (mostly to albumin), and 10% complexed with various anions. Diet, albumin, acid-base balance, and endogenous hormone concentrations (i.e., parathyroid hormone, calcitonin, vitamin D, and corticosteroids) influence total calcium concentration. However, the severity of clinical signs in horses with hypocalcemia is related to deficits in the serum ionized (active) calcium concentration, which may not be reflected in the measured total calcium concentration.

HISTORY AND PRESENTING SIGNS

- Lactating mares (in particular around the tenth day postpartum) and 1 to 2 days following weaning are the times of greatest prevalence
- Following transport, strenuous exercise, and excessive sweating
- Stiff gait, muscle fasciculations, signs of depression or anxiety, increased respiratory excursions with flared nostrils, and sweating may be observed
- In severe cases, recumbency or convulsions may be noted

CLINICAL FINDINGS AND DIAGNOSIS

- Clinical manifestations are variable, depending on the degree of hypocalcemia. Increased muscle tone, a stiff stilted gait, hindlimb ataxia, muscle fasciculations (especially temporal, masseter, and triceps muscles), trismus, dysphagia, salivation, anxiety, profuse sweating, tachycardia, fever, cardiac arrhythmias, synchronous diaphragmatic flutter, convulsions, coma, and death can occur. Signs may progress over 24 to 48 hours, especially in lactating mares.
- In general, increased excitability or anxiousness is observed when serum total calcium concentrations remain at 2 to 2.5 mmol/L (8-10 mg/dL). Concentrations of 1.2 to 2 mmol/L (5-8 mg/dL) usually produce tetanic spasms and incoordination, whereas concentrations less than 1 mmol/L (4 mg/dL) usually result in recumbency and stupor.
- A diagnosis of hypocalcemia should be suspected on the basis of the history and clinical signs, which often are quite characteristic. Although definitive diagnosis depends on laboratory demonstration of hypocalcemia, this finding alone is infrequently recognized and laboratory data usually reflects a primary disease process.
- In addition, hypomagnesemia or hypermagnesemia and hyperphosphatemia or hypophosphatemia have been associated with hypocalcemia in horses although their role in the syndrome is unclear.

DIFFERENTIAL DIAGNOSIS

- Tetanus
- Exertional myopathy
- Laminitis

- Colic
- Seizures
- Botulism
- Viral encephalitides
- Hepatoencephalopathy
- Equine herpesvirus myeloencephalopathy
- Equine protozoal myeloencephalitis
- Hyperkalemic periodic paralysis
- Neuritis of the cauda equina
- Bacterial meningitis/brain abscess
- Verminous encephalitis

TREATMENT

- Mildly affected horses may recover without specific treatment.

KEY POINT

Because hypocalcemia can be life threatening, therapy should be undertaken when significant hypocalcemia (<2 mmol/L or 8 mg/dL) is demonstrated in affected horses.

- Treatment involves slow intravenous administration of calcium solutions such as 20% calcium borogluconate or solutions recommended for treatment of parturient paresis in cattle. Administration of these solutions IV at 250 to 500 mL/500 kg diluted 1:4 with isotonic saline or dextrose and given over 15 to 30 minutes often results in full recovery, although in some cases this may take several days.
- Calcium preparations should be infused slowly, with close monitoring of the cardiovascular response. Dilution of the calcium solutions in saline or dextrose allows for more rapid administration and decreases the chance of cardiotoxicity. Normally, there is an increase in the intensity of the heart sounds. However, alterations in heart rate or rhythm indicate the need to suspend the infusion. If there is no response to an initial infusion, a second dose may be given 15 to 30 minutes later. Most horses respond to this form of therapy, although relapses can occur requiring repeat treatments.

KEY POINT

Long-term feeding of mineral additives containing calcium or leguminous roughage periparturiently or during training may predispose a horse to hypocalcemic tetany once calcium depletion occurs from any of the aforementioned causes.

- Prevention of hypocalcemic tetany involves feeding according to workload, current milk production, and avoiding stressors.

Myotonia

Equine myotonia is a disorder characterized by sustained, involuntary contraction of skeletal muscle after stimulation or voluntary movement. Some forms of myotonia in horses have a genetic basis, particularly in certain families of Quarterhorses. The cause of the disorder is not known, but an alteration in ion conductance across muscle membranes is implicated.

HISTORY AND PRESENTING SIGNS

- Young horses (<6 months old)
- Quarterhorses more commonly affected
- Affected animals may appear to be "double muscled," particularly in the hindlimbs
- Reports or evidence of a stiff, stilted gait

CLINICAL FINDINGS AND DIAGNOSIS

- Affected animals commonly display mild hindlimb stiffness. Gait abnormalities are most pronounced when exercise begins, although often diminish as exercise continues.

KEY POINT

Myotonia primarily affects the extensor muscles of the limbs. The muscles appear firm and tense on palpation, while bilateral bulging of the thigh and rump muscles may give the impression that the horse is overdeveloped or "double muscled. "

- Stimulation of affected muscles, especially by percussion, induces a prolonged, localized muscle contraction evident as a firm raised lump referred to as "dimpling." Affected muscles may remain contracted for up to a minute or more, with subsequent slow relaxation.
- Most horses with myotonia do not demonstrate progression of clinical signs beyond 6 to 12 months of age. However, in some cases, signs may be severe, progressive, and possibly involve a variety of organ systems, similar to myotonia dystrophica in humans.
- A tentative diagnosis of myotonia can often be made on the basis of age and breed; a stiff gait, particularly at the onset of exercise; muscle bulging; and prolonged, localized contraction after muscle percussion.

KEY POINT

Definitive diagnosis is based on electromyographic examination. Usually this is performed only in university or specialist referral clinics. Affected muscle manifests

pathognomonic crescendo-decrescendo, high frequency repetitive electrical bursts with a characteristic "dive bomber" or, more correctly, "revving motorcycle" type of sound.

- Histologic findings on muscle biopsy specimens include marked variation in size and shape of muscle fibers, acute degeneration or atrophy of fibers, and increases in connective tissue and ring bands.

TREATMENT

- Considering the uncertainties regarding the pathophysiologic basis of myotonia, recommendations for effective therapy currently are not possible.
- The prognosis for horses with myotonia is often variable, depending on the severity of clinical signs. Mildly affected animals may have some amelioration of clinical signs with age. Other more severely affected horses may have a progression of signs associated with fibrosis and pseudohypertrophy, to the point where the animal is no longer able to move without great pain and apparent difficulty. Euthanasia of such animals is warranted.

KEY POINT

Although conclusive evidence regarding the genetic basis of this disorder is still not available, owners of affected horses should be cautioned as to the possible heritability of this disease.

Peripheral Neuropathies

Peripheral nerve palsies are common in horses and can result from direct trauma to the nerve or as a result of local inflammatory changes involving the nerve. In most, but not all, cases, they often lead to specific gait abnormalities in horses, as well as loss of skin sensation, muscle weakness, and atrophy of muscles in the areas supplied by the affected nerves.

HISTORY AND PRESENTING SIGNS

Suprascapular Nerve ("Sweeny")

- Trauma to the front of the shoulder
- Forelimb lameness
- Atrophy of the shoulder muscles ("sweeny")

Radial Nerve

- Most common after general anesthesia or in combination with injury to the brachial plexus
- Occurs in association with fractures of the humerus

Avulsion of the Brachial Plexus

- Trauma to the shoulder/brachial region

Sciatic Nerve

- Mostly found in foals
- Inappropriate placement of intramuscular injections

Peroneal Nerve

- Common after general anesthesia in adults
- Trauma to the lateral stifle

CLINICAL FINDINGS AND DIAGNOSIS

Suprascapular Nerve

- Initially after the injury there is a lateral deviation or "subluxation" of the shoulder when the horse bears weight on the limb. This is thought to be due to damage to other supporting structures in addition to the suprascapular nerve. Over the next 2 to 4 weeks, atrophy of the supraspinatus and infraspinatus muscles occurs.
- Diagnosis is based on the history and clinical signs. Some referral clinics perform electromyographic studies, which assist in diagnosis.

Radial Nerve

- Affected horses are unable to extend the elbow, making weight bearing almost impossible. The dorsum of the foot may face the ground. Horses have a characteristic gait when attempting to walk, because they use their body to propel the leg forward.
- Diagnosis is based on history and physical signs.

Avulsion of the Brachial Plexus

- This injury usually results from compression of the brachial plexus resulting in an inability to bear weight, dropping of the elbow, and great difficulty in advancing the limb when walking. Sensory loss up to the elbow is usual.

Sciatic Nerve

- Affected foals cannot flex and advance the hindlimb. If the lower limb is manually extended, affected foals can bear weight. However, under most circumstances, foals have the dorsum of the foot on the ground. They can bear some weight when in this posture.

Peroneal Nerve

- There is an inability to flex the hock and extend the foot. The fetlock is flexed and may touch

and is dragged along the ground when attempting to walk. Even if the foot is manually extended, weight bearing is compromised.

DIFFERENTIAL DIAGNOSIS

Suprascapular Nerve

- Other causes of forelimb lameness
- Avulsion of the brachial plexus
- Radial nerve paralysis

Radial Nerve

- Other causes of forelimb lameness
- Avulsion of the brachial plexus
- Suprascapular nerve palsy
- Fracture of the humerus

Avulsion of the Brachial Plexus

- Other causes of forelimb lameness
- Radial nerve palsy
- Suprascapular nerve palsy

Sciatic Nerve

- Fractures of the hindlimb or pelvis
- Other causes of hindlimb lameness
- Peroneal or tibial nerve palsy

Peroneal Nerve

- Fractures of the hindlimb or pelvis
- Other causes of hindlimb lameness
- Sciatic or tibial nerve palsy

TREATMENT

Suprascapular Nerve

- Initially, conservative treatment involving rest and anti-inflammatory therapy is indicated. In cases where signs persist, surgical exploration and removal of any tissue entrapping the nerve are possible. Further details of treatment are given in Chapter 4 in the section on "sweeny."

Radial Nerve

- Treatment is normally directed at ameliorating clinical signs. This may involve anti-inflammatory therapy such as flunixin meglumine (1.1 mg/kg IV q12-24h; Treatment No. 52) and possibly DMSO (1 g/kg slowly IV as a 10-20% solution in dextrose q24h; Treatment No. 34). In most cases where radial nerve palsy occurs as a result of pressure during general anesthesia, remission of signs often occurs within hours to days.

Avulsion of the Brachial Plexus

- Treatment is as described for radial nerve palsy. Improvement in signs occurs in some cases in about 1 month. In those cases that do not improve in this time, the prognosis is guarded.

Sciatic Nerve

- Principles of therapy are similar to those described for radial nerve palsy. The prognosis must always be guarded.

Peroneal Nerve

- Principles of therapy are similar to those described for radial nerve palsy. Return to normal function in cases associated with general anesthesia is relatively common. Direct trauma to the nerve in the form of a kick or cut has a more variable prognosis.

Shivers

Shivers is a rare and poorly understood neurologic condition, characterized by bilateral muscle fasciculations and tremors involving the muscles of the pelvis, pelvic limbs, and tail. The cause is unknown.

KEY POINT

Shivers generally is a progressive disease affecting primarily draft horses of any age.

HISTORY AND PRESENTING SIGNS

- Mostly large heavy adult horses affected
- Variable presentation, although hindlimb muscle and tail trembling usually are noted at rest

CLINICAL FINDINGS AND DIAGNOSIS

- Although muscle fasciculations and tremors may affect the forelimbs, frequently with carpal elevation and abduction, the predominant muscles affected are those of the hindquarters.
- Signs usually worsen when the horse is backed or circled with sudden jerky extensor movements of the tail. Signs also may occur when limbs are lifted for farrier work.
- In severe cases, hyperflexion and abduction of one hindlimb can occur with the limb remaining in this position and trembling (similar to stringhalt). Signs subside quickly but recur if the horse is backed again. With progression of the disease, muscle atrophy, weakness, and debilitation also may be observed.

KEY POINT

Shivering probably involves hypertonia of flexor and/or extensor muscles and potentially

results from a sensory or motor neuropathy, myopathy, or spinal cord disease of unknown origin.

- Diagnosis is based on recognition of clinical signs and ruling out other problems such as those listed below. Electromyography may reveal fibrillation potentials.

DIFFERENTIAL DIAGNOSIS

- EMND
- Polysaccharide storage myopathy
- Stringhalt
- Equine protozol myeloencephalitis
- Myotonia
- HyPP

TREATMENT

- Although there are anecdotal reports of variable success using agents such as phenytoin (10-12 mg/kg PO q12h for 3-4 days, then 10-12 mg/kg q24h for 3-4 days, then 5-6 mg/kg q24h; Treatment No. 91) or baclofen (this is very expensive), at present no reliable treatment has been documented.
- In many horses the clinical signs remain static and the animals continue to be useful. Improvement after a prolonged period of rest and spontaneous recovery can occur.

Stringhalt

Stringhalt refers to intermittent or continuous exaggerated flexion of the hindlimbs (one or both) when the horse moves. This disease occurs as an isolated event in adult light horses and in an outbreak form as described elsewhere in the text (see Chapter 4).

Multifocal Neurologic Diseases

Equine Degenerative Myeloencephalopathy

Equine degenerative myeloencephalopathy (EDM) is a diffuse, symmetric, degenerative disease of the spinal cord and brain, similar to neuraxonal dystrophy in people and Morgan horses.

KEY POINT
EDM is one of the most common causes of ataxia in young horses of many breeds and both sexes.

The disease has been diagnosed in Great Britain, continental Europe, and North America and is reported to occur more frequently in northeastern United States. The specific etiology of EDM is unknown. However, confinement to dirt paddocks, exposure to either insecticides (in particular those containing pyrethrins) or creosote-based wood preservatives, or consumption of processed or pelleted feeds are thought to be risk factors for the disease. In addition, hypovitaminosis E and copper deficiency during the first year of life have been implicated as causative factors in the development of EDM. Low serum vitamin E concentration is likely to be the result of insufficient intake or a defect in vitamin E metabolism.

KEY POINT
Foals born to dams that have previously had a foal with EDM also appear much more likely to develop the disease.

HISTORY AND PRESENTING SIGNS

- Young horses less than 2 years of age. The mean age of onset is approximately 5 months, although onset of disease has been observed in horses as late as 12 years of age
- Horses in dirt yards fed pelleted or processed rations that are deficient in vitamin E
- Can occur in outbreaks
- Application of insecticides containing pyrethrins or pyrethroids or exposure to creosote-based wood preservatives
- Insidious onset of incoordination and stumbling progressing to bilaterally symmetric ataxia, paresis, and spasticity usually affecting all four limbs. Abrupt onset of signs may occur
- Familial predisposition is reported in Thoroughbreds, Standardbreds, Appaloosas, Morgans, Arabians, Paso Fino, and Norwegian Fjord horses

CLINICAL FINDINGS AND DIAGNOSIS

- Signs of ataxia and paresis commonly begin in the hindlimbs and progress to the forelimbs, although severity can be equal in all limbs.
- Gait and postural abnormalities include dragging of toes, proprioceptive deficits such as pivoting and circumduction, and difficulty backing, circling, and negotiating hills.
- Cranial nerve involvement, muscle atrophy, and changes in skin sensation or tail tone are not features of EDM.
- In horses that have been affected for a long period, there may be evidence of generalized

hyporeflexia, as demonstrated by reduced or absent cervical, cervicofacial, cutaneous trunci, and laryngeal adductor reflexes. This assists in identification of a diffuse thoracic spinal cord lesion and decreases the likelihood of the ataxia being due to cervical stenotic myelopathy.

KEY POINT

Despite progression of clinical signs early in the disease, signs appear to plateau once the horse matures (or reaches 2 years of age), and tetraplegia rarely develops. Horses that survive to 2 to 3 years of age commonly exhibit lifelong stable neurologic deficits.

- Diagnosis of EDM is based on clinical signs and by ruling out other possible causes of ataxia using cervical radiographs with or without myelography and CSF analysis as diagnostic aids.
- Some horses with the disease will have low serum α-tocopherol concentrations (<1.5 mg/mL; normal >2.0 mg/mL). However, given the potential daily variation in a-tocopherol values in individual animals and the delay that may exist between the presence of hypovitaminosis E and occurrence of the disease, measurement of serum a-tocopherol values is more likely to be of use as a herd-screening test in outbreaks.
- Definitive diagnosis depends on histopathologic examination of the brainstem and spinal cord and demonstration of neuraxonal degeneration (dystrophy) with prominent neuronal swelling, depletion, atrophy, and lipofuscinosis.

DIFFERENTIAL DIAGNOSIS

- Equine cervical stenotic myelopathy
- Equine herpesvirus myeloencephalopathy
- Equine protozoal myeloencephalitis
- AO malformation
- Cerebellar abiotrophy
- Meningitis
- Trauma
- Neuritis of the cauda equina
- Vertebral osteomyelitis
- Verminous myelitis
- Rabies

TREATMENT

- Although no specific treatment for EDM is available, affected horses can improve when administered large doses of oral vitamin E (Rovimix 20, Hoffman-LaRoche, Nutley, NJ; 6000 IU/day of D,l-a-tocopherol acetate in 60 mL of corn oil mixed in 1 L of sweet feed or grain) up to the third year of life. Horses with low serum vitamin E concentrations also may benefit from 1000 (foals and yearlings) to 2000 (adults) IU of vitamin E in oil given intramuscularly every 10 days. Administration of vitamin E intravenously in horses should be avoided because of increased risk of sudden death following injection.
- Clinical signs of improvement can be noted within 2 to 3 weeks, with continued progress over the next 6 to 12 months.
- A balanced diet including fresh green feed with or without vitamin E supplementation should be provided. Prophylactic treatment with vitamin E (1000-2000 IU/day) should be used in all foals considered to be at risk based on familial history and management regardless of baseline serum vitamin E concentrations.

KEY POINT

Once signs have developed, full recovery is unlikely. However, the best recovery usually is seen in horses in which the disease is diagnosed and treated in the early stages.

- Prevention of EDM may be related to ensuring that foals are not kept in dirt paddocks, are not exposed to insecticides and wood preservatives, and receive a balanced diet, preferably at pasture, that is rich in green feed.

Equine "Grass Sickness" (Equine Dysautonomia)

Equine grass sickness is an idiopathic invariably fatal neurologic disease associated with the selective degeneration of neurons of the autonomic nervous system. Reports of affected horses are confined to northwestern Europe and South America. The disease currently is not recognized as occurring in the United States, Australia, or New Zealand.

KEY POINT

The etiology of equine grass sickness is still unknown, although an ingested neurotoxin is implicated. It is proposed that this toxin concentrates at intestinal locations (in particular the ileum) where absorption is greatest. Selective binding of the neurotoxin to autonomic nerve terminals in the enteric plexuses and retrograde axonal transport to other autonomic neurons results in significant neuronal degeneration and depletion.

Intestinal dysfunction also has been postulated to be the result of excessive quantities of norepinephrine or nitric oxide produced by neurons in the enteric plexuses of affected horses.

HISTORY AND PRESENTING SIGNS

- No sex or breed predilection
- Mainly in grazing horses 2 to 7 years old during the spring and summer
- Greater risk when horses are turned out to pasture after winter confinement, if low-lying pastures adjacent to streams are used or if horses are moved to new pasture
- Signs of depression or moderate to severe abdominal pain with abdominal distention
- Thick saliva may drool from the mouth and inability to swallow may be noted.
- Patchy sweating over the flank and behind the shoulder

CLINICAL FINDINGS AND DIAGNOSIS

KEY POINT

Most clinical signs may be attributed to disordered autonomic innervation of the alimentary tract from the pharynx to the rectum.

Acute Grass Sickness

- The major findings in acutely affected horses include mild to severe colic characterized by abdominal distension, gastric reflux (up to 20 L) when intubated, spontaneous nasogastric regurgitation, and tachycardia (70-120 beats/min). Sudden death also is common.
- Gastrointestinal atony results in decreased or absent borborygmi on auscultation, whereas secondary tympany, large colon impaction, and dry fecal balls are palpable per rectum. Prominent loops of distended small intestine also may be detected during rectal examination.
- Of critical importance to improving accuracy of diagnosis is observation of subtle signs such as ptosis, dry rhinitis causing a snuffling sound, and prominent dysphagia. Affected horses are slow to masticate food and are either unable to swallow food and water or unable to swallow without pain, often inducing regurgitation. However, dysphagia can be difficult to assess in affected horses that refuse to eat or drink.
- Patchy sweating also may be seen on the flank and behind the shoulders, whereas fine muscle fasciculations may affect the triceps and hip/stifle areas.

Chronic Grass Sickness

- Progressive emaciation, dehydration, anorexia, and signs of depression are the main clinical findings in horses with chronic grass sickness.
- Affected horses may wander aimlessly and develop intermittent signs of colic, usually the result of ileus rather than colonic impaction or gastric distention. Scant, dry feces covered with mucus are present in the rectum, although diarrhea sometimes is observed.
- Other physical findings include absent borborygmi, normal to moderately increased heart rate, sweating over the flanks, and muscle tremors involving the triceps and hips. In addition, swallowing generally is slow and may appear painful, whereas blockage of the nasal passages with mucopurulent material frequently occurs.

KEY POINT

Diagnosis of grass sickness rests with recognition of subtle clinical signs, which may require the horse to be evaluated more than once. Results of histopathologic assessment of ileal biopsies at laparotomy, or as part of a postmortem examination, are necessary for definitive diagnosis.

- Nonspecific clinicopathologic abnormalities are consistent with hypovolemia, stress, and anorexia. Analysis of peritoneal fluid samples invariably shows increased protein concentrations and normal nucleated cell counts.
- Histologic examination of ileal biopsies obtained at exploratory laparotomy (usually performed to eliminate the possibility of the existence of a surgical lesion) reveal characteristic noninflammatory neuronal degeneration and loss. Other findings during laparotomy may include extensive, segmental impaction of the large colon, gastric dilatation, and poor gastrointestinal motility.
- Decrease in the number of peptide-specific neurons observed in histologic sections of rectal mucosal biopsies may provide further support for a diagnosis antemortem.
- Esophageal motility also may be assessed using contrast radiography and fluoroscopy if available.
- Although usually limited to university or referral institutions, demonstration of high circulating concentrations of histamine, adrenocorticotropin hormone, Cortisol, and norepinephrine also can aid in the diagnosis.

DIFFERENTIAL DIAGNOSIS

- Small or large intestinal obstruction (e.g., intraluminal and extraluminal causes)

- Paralytic ileus associated with peritonitis, proximal enteritis, or toxemia
- Primary or secondary gastric dilation or impaction
- Other causes of dysphagia (e.g., botulism, pharyngeal paralysis)
- Esophageal obstruction
- Other causes of chronic weight loss in which there also is intermittent abdominal pain (e.g., cyathostomiasis, sand impaction, infiltrative inflammatory bowel diseases)

TREATMENT

- In acute cases of grass sickness, treatment with intravenous fluid therapy, analgesia, and repeated gastric decompression may be attempted until a clinical diagnosis becomes more certain. However, marked degeneration and loss of enteric intramural plexuses in horses with acute grass sickness resulting in complete gastrointestinal atony, colonic impaction, and dysphagia probably are incompatible with recovery, and euthanasia is recommended.

> KEY POINT
>
> *Success may be achieved in treating some chronic cases because neuronal degeneration is relatively mild. With intensive commitment from owners and veterinary staff and careful and appropriate case selection, approximately 40% of horses with chronic grass sickness have been reported to recover and return to work.*

- In general, horses likely to survive are those that have less difficulty swallowing, have few and less severe episodes of colic, are prepared to eat, have mild purulent nasal discharge, and are able to rise from recumbency without difficulty.
- Supportive treatment in suitably selected cases involves close attention to feed, fluid, and exercise requirements; provision of analgesia when necessary; and prompt correction of any complications such as diarrhea, esophageal choke, and inhalation pneumonia.
- Although intravenous fluid therapy and providing food often with liquid paraffin by stomach tube may be necessary in the early stages, most cases that have a chance of survival generally do not require these treatments. A commercial or homemade diet high in energy and protein that is easily swallowed should be fed (e.g., oats mixed with warm diluted molasses, cut grass, bran mash, carrots, apples). However, appetite often varies in terms of quantity consumed and food preference. For further details on the feeding of debilitated horses, see Chapter 18.
- Short walks initially to maintain interest and gut motility can be followed by gradually increasing lengths of time spent on pasture.
- In cases that show mild signs of colic, often after eating, nonsteroidal anti-inflammatory drugs such as phenylbutazone (Treatment No. 89) or flunixin meglumine (Treatment No. 52) can be used at recommended dosages. Cisapride (0.1 mg/kg IM q12h or 0.5-0.8 mg/kg PO q8h) also can be administered for 7 days to increase intestinal motility.
- Other supportive measures include application of a blanket to assist in maintenance of body temperature, frequent grooming, and removal of nasal crusting.
- Occasionally, diarrhea or esophageal choke occurs, although most cases resolve without treatment. However, sudden onset of signs of inhalation pneumonia results in a fatal outcome despite institution of appropriate antibiotic therapy.
- Recovery from chronic grass sickness with excellent supportive care can take at least 6 to 8 weeks during which there is continuing but diminishing rate of weight loss, and fewer signs of dysphagia, sweating, muscle tremors, nasal discharge, and colic. Many horses require up to a year or more before returning to work and most are susceptible to future problems (e.g., dysphagia and choke) if subjected to excessive stress. In addition, horses that have recovered may retain their winter coat, develop small areas of erect hair, or lose coat color.

Equine Herpesvirus 1 Myeloencephalopathy

Equine herpesvirus type 1 (EHV 1) is one of five distinct herpesviruses known to cause disease in horses. Both EHV 1 and EHV 4 are enzootic in the horse population, and most horses show serologic evidence of exposure to these viruses. Most horses that have recovered remain latently infected with EHV 1 and/or EHV 4 for life. However, stress-associated recrudescence of latent infections can result in clinical disease or horses may become shedders of virus. Although EHV 4 is a major cause of respiratory tract disease in horses, EHV 1 is responsible for abortions, birth of dead or weak foals, mild respiratory infections, and myeloencephalopathy. The latter disorder is due to a diffuse hemorrhagic vasculitis and thrombosis of arterioles in the brain and spinal cord resulting in infarction of neural tissue. The patho-

genesis probably is related to direct viral infection of neural tissues associated with an immune-mediated type III hypersensitivity reaction in central nervous system vessel walls.

KEY POINT

Natural infection with EHV 1 occurs by inhalation of aerosols of virus-containing secretions (e.g., saliva, nasal, and ocular discharges and secretions from EHV 1 abortions) or ingestion of infective virus from the products of EHV 1 abortions and possibly feces.

Clinically affected horses, subclinically affected horses, or shedding carrier horses can disperse EHV 1 for up to 2 weeks, and virus may remain infective in the environment for up to 14 days or 5 to 6 weeks on suitable fomites such as horse hair.

Although EHV 1 myeloencephalopathy can occur as sporadic individual cases, more commonly, outbreaks in breeding and racing establishments with morbidity up to 40% and relatively high mortality (up to 90%, in some reports) may occur. In general, cases of neurologic EHV 1 infection occur concurrently with or subsequent to outbreaks of abortion or, less frequently, respiratory disease. However, recrudescence of latent infection or contact with inapparently affected horses may result in outbreaks of neurologic disease in the absence of other signs of EHV 1 infection and without the introduction of new horses.

KEY POINT

Distribution of EHV 1 myeloencephalopathy is worldwide, although most outbreaks have been reported in Europe, North America, and Australia.

HISTORY AND PRESENTING SIGNS

- Occurs in horses of any breed or sex and at all ages, although pregnant mares and mares nursing foals appear to be at increased risk
- Exposure to horses with current or recent history of respiratory tract disease, neurologic deficits, abortion, and neonatal deaths or foal diarrhea
- Recent weaning, castration, parturition, transport, racetrack work, use of exogenous glucocorticosteroids, and possibly use of live-virus EHV 1 vaccines may increase risk
- Can affect a single horse or occur in outbreaks
- Acute onset of incoordination, weakness, or recumbency that may be preceded or accompanied by signs of respiratory tract infection and signs of depression, inappetence, limb edema, and ocular discharge within the previous 10 to 14 days

CLINICAL FINDINGS AND DIAGNOSIS

- Neurologic signs usually appear 6 to 10 days after exposure to the virus. There may be fever or hypothermia at the onset of signs and lower limb edema and scrotal edema in colts and stallions.

KEY POINT

Neurologic deficits can be highly variable depending on location and severity of lesions. Acute onset of symmetric ataxia and paresis of trunk and limbs most commonly is noted, progressing rapidly for 1 to 2 days before stabilizing.

- The hindlimbs generally are more severely affected with thoracic limb involvement less commonly observed. Severely affected horses show profound limb weakness that may progress to paraplegia, with dog sitting or complete teraplegia and recumbency. In recumbent horses, spinal reflexes are normal to increased, whereas perineal reflexes are preserved.
- Asymmetric signs, signs of hemiparesis or unilateral hindlimb or forelimb lameness progressing to more generalized ataxia, paresis, and recumbency, also infrequently occur.
- Dribbling of urine secondary to urinary retention, penile prolapse, fecal retention, and hypotonia and hypalgesia of the tail and perineal regions commonly but not invariably are found.
- Most horses with the disease remain alert and have good appetite. Rarely, there are signs of brain involvement and cranial nerve dysfunction with vestibular signs (e.g., nystagmus, circling with head tilt) with or without facial nerve palsy, and dysphagia due to lingual, mandibular, and pharyngeal paresis.

KEY POINT

A fourfold or greater increase in serum neutralizing or complement-fixing antibody titer to EHV 1 between samples taken during the acute stages of the disease and 2 to 3 weeks later is strong evidence of a recent tion with the virus. As well, the presence of antibodies to EHV J in the CSF of affected horses can assist in diagnosis, particularly if the titer is higher than in the serum.

- Caution should be exercised in interpretation of EHV 1 neutralizing antibody titers in CSF be-

cause blood contamination during collection and other diseases that cause an increase in the permeability of the BBB may falsely elevate CSF antibody titers if serum titers also are high.

- Other findings on analysis of CSF include xanthochromia and an increase in protein (albumin) concentration, with values increasing to 1.0 to 3.0 g/L (100-300 mg/dL). The total nucleated cell count usually is normal but occasionally is increased. Changes in CSF resolve quickly and therefore may not be present at the onset of clinical signs.
- Serologic testing in many individual horses can be unrewarding because peak antibody titers may already have dissipated before neurologic signs appear. However, testing of paired serum samples from affected and unaffected in-contact horses is recommended to provide indirect evidence of infection with EHV 1.
- Viral isolation and identification of EHV 1 can be undertaken during outbreaks of neurologic disease via collection of nasopharyngeal swabs, tracheal fluids, and samples of blood (buffy coat) from affected and preferably unaffected in-contact horses. Although strongly supportive of a diagnosis of EHV 1 myeloencephalopathy, interpretation of positive cultures may be confusing, because EHV 1 has been isolated from the respiratory tracts of normal horses. Isolation of EHV 1 from CSF of affected horses confirms the diagnosis but seldom is successful.
- Additional future diagnostic procedures include indirect immunoperoxidase staining and the polymerase chain reaction (PCR) technique for detection of viral antigen in nasopharyngeal, buffy coat, or CSF samples.

KEY POINT

Antemortem diagnosis of EHV 1 myeloencephalopathy in individual horses frequently is not possible. Definitive diagnosis is based on histologic demonstration of widespread vasculitis and white and gray matter malacia and detection of viral antigen-containing cells in the central nervous system using PCR or indirect immunoperoxidase methods.

DIFFERENTIAL DIAGNOSIS

- Equine cervical stenotic myelopathy
- Equine protozoal myeloencephalitis
- Neuritis of the cauda equina
- Viral encephalitides
- Meningitis
- Cranial or spinal cord trauma
- Equine degenerative myeloencephalopathy
- Vertebral osteomyelitis
- Verminous myelitis
- Brain abscess
- Rabies
- Botulism
- Plant or chemical intoxication
- Congenital abnormalities

TREATMENT

- Horses suspected of being affected should be isolated with treatment directed toward supportive and nutritional care and by reducing central nervous system immune-mediated inflammation. Although no specific antiviral therapy has been evaluated, one report suggests acyclovir (8-16 mg/kg PO q8h for 10-14 days) may be beneficial in neonates affected with EHV1 myeloencephalopathy, in particular if used as a preventative for unaffected in-contact foals. Although expensive, a similar dose regimen may be beneficial in adults. *Note:* renal function should be monitored when administering acyclovir.
- Mildly affected horses often stabilize rapidly over a period of hours to a few days and if eating, drinking, and walking adequately may subsequently recover completely with little or no therapy.
- Anti-inflammatory therapy using intravenous DMSO (1 g/kg slowly IV as a 10-20% solution in normal saline q12h for 1 to 3 days; Treatment No. 34) and dexamethasone (0.05-0.2 mg/kg IV or IM q12h for 1-3 days, then tapering over 3-5 days; Treatment Nos. 29 and 30) can be beneficial in the first 24 to 48 hours.
- Supportive therapy is critical. Horses with severe ataxia require restriction of movement and ready access to water and palatable feeds, including fecal softeners such as bran mashes, mineral oil, or psyllium to reduce intestinal impaction. Frequent manual emptying of the rectum or administration of enemas also may be necessary.
- Horses with urinary retention require bladder catheterization and evacuation at least twice daily until normal micturition returns. In cases where urinary incontinence persists, placement of an indwelling Foley catheter (24-28Fr) is indicated. In stallions and geldings, this may need to be performed via a perineal urethrostomy. Once the catheter is inserted, the balloon is inflated, and the free end of the catheter is attached (with Supaglue or Krazy Glue) to a urine collection bag or old 5-L fluid bag. This bag is then taped to the horse's leg to allow accumulation of urine by gravity flow. Provision of the bag prevents urine scald. In fractious horses, maintenance of the leg bag may be dif-

ficult, although attaching the Foley catheter to 3 to 4 feet of a valved stomach tube braided into the tail may be a suitable alternative.
- Cystitis is common secondary to urine stasis and bladder catheterization. As a result, prophylactic antibiotic coverage (e.g., trimethoprim-sulfadiazine at 15-20 mg/kg PO q12h; Treatment No. 108) is indicated.
- Recumbent patients provide a significant therapeutic challenge and usually have the worst prognosis for recovery. Complications such as myonecrosis, decubital ulcers, respiratory tract infections and gastrointestinal tract obstruction, or ulceration frequently occur in recumbent horses.
- Apart from therapies described above, recumbent horses should be maintained in sternal position and rolled frequently. An appropriately fitted sling can be beneficial in moderately affected horses that are too weak to rise but are able to maintain a standing position with minimal assistance or stress.

KEY POINT

Complete recovery may require several weeks to more than a year in some cases. Control of urination usually returns before resolution of gait abnormalities. Residual deficits may be seen in some horses after convalescence.

- Control measures during outbreaks include
  - o Isolation of affected and nonaffected in-contact horses.
  - o Minimizing traffic of horses and people on and off infected premises until at least 3 weeks after resolution of acute signs in the last clinical case. New arrivals also should be isolated for 3 weeks before joining the herd.
  - o Disinfection of infected equipment and horse stalls.
  - o Disposal of aborted fetuses, fetal membranes, and bedding contaminated with fetal fluids.
  - o Reduction of stresses that may induce recrudescence of latent EHV 1 infection.

KEY POINT

Use of modified live and inactivated EHV 1 vaccines does not prevent infection with EHV 1 or prevent the possibility of abortion, myeloencephalopathy, or establishment of the carrier state. However, there is strong ancedotal evidence suggesting these vaccines induce some immunity to respiratory disease and may reduce the incidence of abortion.

- Vaccination of unexposed horses, although controversial, may limit spread of the virus or the possibility of viremia. However, first-time vaccination of in-contact animals during an outbreak is not recommended because of risk of precipitating disease.

Equine Protozoal Myeloencephalitis

EPM is a common, although sporadic, neurologic disease reported only in horses from the American continents. Serologic surveys suggest that approximately 40 to 50% of horses in the United States, most of which are racehorses, have been exposed to the causative organism.

KEY POINT

The disease is caused by the protozoan parasite Sarcocystis neurona *and results in progressive clinical signs involving the spinal cord and brain, the severity of which is related to the size of the infective dose, immunocompetence of the host, and degree of environmental stress.*

Horses are an aberrant intermediate dead-end host of *S. neurona* and are infected by ingesting *S. neurona* sporocysts in feed or water contaminated by the feces of opossums *(Didelphis virginiana),* the definitive hosts. *{Note:* Until recently, *S. neurona* was considered to be *S. falcatula,* a well characterized *Sarcocystis* sp. of opossums. However, current research has demonstrated that *S. falcatula* and *S. neurona* are distinct species and that avians are the intermediate host of *S. neurona.*

In most horses, the organism is cleared by a competent immune response, whereas in others tachyzoites gain access to the central nervous system. Here, the organisms undergo asexual reproduction intracellularly in neurons and microglial cells, without forming tissue cysts. Therefore, horses cannot transmit *S. neurona* to other animals. Although earliest clinical signs of EPM can be observed in horses 4 weeks after ingestion of sporocysts, some horses can take as long as 2 years to develop signs.

KEY POINT

EPM is the most common form of multifocal neurologic disease in the horse.

HISTORY AND PRESENTING SIGNS

- All ages can be affected but most affected horses are 1 to 5 years of age.
- Affected horses often have a history of recent stress (e.g., transport, training, or foaling).

- Identified in most breeds, but Standardbreds and Thoroughbreds are overrepresented. Ponies appear to have resistance.
- Onset of signs may be acute or insidious and usually are progressive.
- Presenting signs are highly variable—atypical lameness, gait asymmetry, difficulty maintaining a specific limb lead or negotiating turns on a racetrack, frequent stumbling, and behavioral problems (e.g., head tossing, training difficulties) are commonly reported early signs.
- Atrophy, particularly involving the gluteal muscles, may be evident.
- Signs of depression, head tilt, dysphagia, and paralysis of tongue and larynx.

CLINICAL FINDINGS AND DIAGNOSIS

KEY POINT

Neurologic signs are referable to the site or sites of infection in the central nervous system and are the result of direct neuronal damage by the parasite and indirect damage from edema and inflammation. Multifocal disease is typical of EPM.

- Signs of spinal cord involvement alone are observed more commonly than signs of brain disease or the combination of spinal cord and brain signs.
- Asymmetric or symmetric truncal and limb ataxia and weakness occur, whereas LMN signs result in asymmetric atrophy primarily of the gluteal, biceps femoris, and infraspinatus/supraspinatus muscles.
- All four limbs are involved if the spinal cord is affected cranial to T3, whereas the hindlimbs only are involved if the cord is affected caudal to T3. Lesions behind S3 result in signs of cauda equina syndrome, which may include degrees of rectal, anal, bladder, and penile paralysis and hypalgesia of the skin of the tail and perineum.
- Signs progressively worsen over hours to years with recumbency the end result.
- Demarcated areas of spontaneous sweating, hypalgesia, or complete loss of cutaneous sensation on the head, neck, or body also may be demonstrated.

KEY POINT

A wide range of mild early signs of EPM may be detected by careful observation of horses during walking, "breaking," or training. These may include asymmetric stride length, toe dragging, spasticity, frequent bucking, head tossing or shaking, and upward fixation of the patella and back soreness caused by weak or asymmetric use of muscle groups.

- Approximately 10% of horses with EPM develop brain disease, with the brainstem and cranial nerves most commonly affected. Asymmetric vestibular dysfunction with head tilt, nystagmus, and base-wide stance may be observed. Other findings are facial paralysis, dysphagia, laryngeal hemiplegia, tongue paralysis, strabismus, and weak jaw tone. On occasion there may be involvement of the cerebrum, reticular activating system, and cerebellum manifest as seizures, central blindness with loss of the menace response, behavioral abnormalities, profound signs of depression, or ataxia.

KEY POINT

EPM should be suspected in any horse with acute onset of progressive, asymmetric, multifocal *neurologic disease characterized by ataxia, weakness, and muscle wasting. However, definitive diagnosis relies on analysis of CSF, preferably, taken from the LS space.*

- Cytologic analysis of CSF may reveal evidence of xanthochromia, moderate elevations in total protein concentration (>0.8 g/L or >80 mg/dL), or an increase in total nucleated cell count (>10 X 10^6/L or >10/μL), predominantly lymphocytes and mononuclear cells. Eosinophils may be seen on occasion. CK activity also may be high, although spurious increases occur as a result of contamination of the CSF with inadvertent inclusion of epidural fat or dura during collection. However, these alterations in CSF are not observed consistently nor are they specific for EPM.
- Positive identification of CSF antibody produced in response to antigens unique to *S. neurona* using the Western immunoblot test (Equine Biodiagnostics, Lexington, KY), is approximately 90% sensitive and specific for the diagnosis of EPM.
- A positive result for CSF indicates that parasites have penetrated the BBB and have stimulated a local immune response. In contrast, positive immunoblot analysis *of serum* indicates exposure to the organism only and does not necessarily implicate EPM as the cause of neurologic signs, if they are present.

KEY POINT

High prevalence of CSF-positive results occasionally has been found among groups of young clinically normal horses suggesting that

subclinical central nervous system infections and production of CSF antibody may occur. However, the significance of positive CSF immunoblots from clinically normal horses currently is unclear.

- If there is inflammatory compromise of the BBB, which may occur as a result of EHV 1, trauma, meningitis, or EDM, circulating serum antibodies to *S. neurona* may leak across the barrier and produce a false-positive CSF test result. A false-positive immunoblot test of CSF also may be observed if the sample of CSF is contaminated with antibody-containing blood during collection.
- Measurement of CSF albumin quotient may be useful to interpret the role of potential leakage or contamination by blood proteins in CSF when results of immunoblot analysis for *S. neurona* are positive. An albumin quotient greater than 2.0 is evidence that contamination or leakage of protein from blood or serum into the CSF has occurred and a positive CSF immunoblot result must be considered false. Another CSF aspirate should be collected at a later date.
- Other reasons for false-positive results of immunoblot analysis of CSF includes laboratory or technical error and an immune response to infection, preventing clinical signs.
- False-negative results are rare and may occur in horses that are tested before the development of sufficient antibody response, in those that have been previously treated with antifolate drugs or corticosteroids, and in those with chronic inactive foci of organisms.

KEY POINT

Immunoblot analysis of CSF is more reliable for identification of horses affected with EPM than is analysis of serum, in particular when combined with the results of cytology and CSF albumin quotient. Widespread use of the immunoblot test in the diagnosis of EPM has contributed to an increased awareness of the disease.

- In acute cases before an antibody response has developed or in chronic indolent cases in which the serologic response has waned or never developed, PCR testing (Equine Biodiagnostics, Inc.) of CSF or blood collected in EDTA may be used. Detection of the presence of parasite DNA using PCR does not rely on antibody production and, if used in combination with immunoblot, becomes highly specific and sensitive for diagnosis of EPM.
- *Note:* Some investigators have found PCR testing of limited use as a method for diagnosing EPM because of high rates of false-positive results. It is proposed that minimal protozoal DNA is present in CSF for detection by PCR testing. As well, protozoal organisms are not found in CSF cytology and rarely are observed in lesions at postmortem.

DIFFERENTIAL DIAGNOSIS

- EDM
- Equine cervical stenotic myelopathy
- Equine herpesvirus myeloencephalopathy
- Traumatic spinal cord injury
- Neuritis of the cauda equina
- Viral encephalitides
- Meningitis
- Vertebral osteomyelitis
- Verminous myeloencephalitis
- Rabies
- Brain abscess
- Congenital abnormalities

TREATMENT

- At least 75% of horses with EPM improve when treatment with sulfadiazine-sulfonamide (20-30 mg/kg q12h PO; Treatment No. 108) plus pyrimethamine [Daraprim; 1 mg/kg q24h PO) is instituted.
- Horses should be treated with both drugs without variation in dosage for at least 90 days or until they become immunoblot negative in CSF. Use of lower doses or administering a single antiprotozoal agent only may encourage selection for a resistant population of protozoa within the horse that may lead to persistence of intrathecal antibody, relapses after cessation of therapy, or worsening of clinical signs during treatment.
- Horses that remain positive on CSF immunoblot testing should receive continued antimicrobial therapy until they test negative. Treatment for these chronic cases may be augmented with the use of immunostimulants such as killed *Propionibacterium acne* (Equistim), mycobacterial cell wall extract (Equimune IV), levamisole, or a-interferon.
- Antibiotic medications are best administered by syringe into the mouth and preferably at least 1 hour before hay and grain is fed, because these feeds may interfere with absorption.
- A complete blood count is recommended approximately every 2 weeks during therapy because pyrimethamine (and trimethoprim-sulfonamide if used) can interfere with folic acid metabolism in the horse, resulting in folic acid

deficiency, manifested by cytopenia (leukopenia, thrombocytopenia, anemia). This side effect is rare and usually mild, necessitating only halving the dose of pyrimethamine and providing high-quality folate-rich green forage. If severe bone marrow suppression occurs or the PCV drops below 20%, antimicrobial therapy should be discontinued and folinic acid (Leucovorin, Immunex, Seattle, WA) at 0.1 to 0.3 mg/kg q24h administered orally until hematologic values return to an acceptable range. Although poorly absorbed, folic acid (40 mg q24h PO) also can be used.

- If clinical signs initially are severe or rapidly progressive, anti-inflammatory and antioxidant therapy should be used to minimize neuronal dysfunction secondary to protozoa-induced inflammation. Flunixin meglumine (1.1 mg/kg q12h for 3-4 days, then q24h for 4 days; Treatment No. 52) used together with DMSO (1 g/kg as a 10% solution slowly IV or PO q12h for 3 days; Treatment No. 34) may provide some symptomatic relief early in the disease. Acute onset of severe brain disease resulting from EPM also may warrant a brief course of dexamethasone (0.05 mg/kg q12h IV; Treatment No. 30).
- Use of antioxidant therapy during the treatment period using oral vitamin E supplementation (10-20 IU/kg q24h) may aid to promote healing of central nervous system damage.
- Recent preliminary research has shown that the oral administration of triazine-based coccidiocidal agents, diclazuril (Clinacox 0.5%; Janssen Pharmaceuticals, Beerse, Belgium) and toltrazuril (Bayer Pharmaceuticals, Leverkusen, Germany) at a dose rate of 5 mg/kg q24h and 10 mg/kg q24h, respectively, resulted in improvement in a small number of EPM-affected horses. However, therapeutic specifics for these agents in the horse remain to be determined.
- Most horses with EPM will show clinical improvement within several weeks of commencement of therapy. Residual neurologic deficits may still exist in 25 to 50% of affected horses at the conclusion of therapy. Subsequently, horses should be carefully monitored within the first few months for early signs of recurrence. Treatment of horses that relapse usually is less successful, and duration of the second treatment may need to be at least twice as long as the first or for the life of the horse.
- Prevention of EPM is best accomplished by denying access of opossums to horse-feeding areas. Trapping and relocating opossums may decrease morbidity of EPM in endemic areas.

Polyneuritis Equi (Neuritis of the Cauda Equina)

The cauda equina in horses begins at approximately the lumbosacral junction and includes the terminal segments of the spinal cord and the nerve roots of all five sacral and at least five coccygeal spinal cord segments. These sacrococcygeal nerve roots give rise to peripheral nerves that provide motor and/or sensory supply to muscles and skin of the pelvic limb, perineum, rectum, anus, scrotum, udder, and tail, as well as parasympathetic innervation to the bladder, urethra, rectum, anus, and genital erectile tissue.

Disorders of the cauda equina in horses most commonly are the result of sacrococcygeal vertebral trauma, EHV 1 myeloencephalopathy, or polyneuritis equi (PNE). The latter disease will be discussed further.

PNE, or neuritis of the cauda equina, is an uncommon neurologic disease characterized by granulomatous perineuritis that involves the cauda equina and other spinal and cranial nerve roots (in particular cranial nerves V, VII, and VIII). The disease has been reported in Europe, Canada, and the United States.

KEY POINT

The disorder is thought to be immune-mediated, possibly the result of viral (e.g., EHV 1 or adenovirus) or bacterial (e.g., streptococci or possibly C. tetani*) infection.*

HISTORY AND PRESENTING SIGNS

- Usually adult horses (youngest horse affected was 17 months of age).
- There is no sex or breed predisposition.
- Acute signs of hypersensitivity of hind regions or head progressing to tail weakness or paralysis, urinary dribbling and scalding of the hindlimbs, penile paralysis and hindlimb ataxia.

CLINICAL FINDINGS AND DIAGNOSIS

- Acute signs with head and/or perineal hyperesthesia often may not be identified. Vague signs of intermittent, low-grade colic due to fecal retention or evidence of self-mutilation may be all that is observed.
- Subsequently, signs tend to become chronic and are characterized by progressive paralysis of the tail with reduced "tail clamp" reflex, reduced anal tone, and insensitivity to painful stimulation around the anus and perineum. Reduced or

absent rectal and bladder tone results in retention of feces and urine (with extreme bladder distention).
- The penis often protrudes from the prepuce and is insensitive to painful stimuli.
- Less commonly, there is mild symmetric hindlimb ataxia and paresis and atrophy of the muscles of the hindlimbs and rump.
- Although sacrococcygeal lesions predominate, head signs also can occur. The latter often are characterized by asymmetric lesions of cranial nerves V, VII, and VIII, resulting in a head tilt, unilateral facial paralysis, masseter muscle atrophy, and difficulty in chewing. Involvement of cranial nerves II, III, IV, VI, IX, X, and XII also has been reported.
- These signs of cranial nerve dysfunction can appear suddenly, sometimes without signs of cauda equina disease, then wax and wane.

KEY POINT

Diagnosis is based on the history, clinical signs, CSF findings, and evidence of progression of the disease.

- CSF is abnormal in some cases, with increases in protein (1.0-3.0 g/L or 100-300 mg/dL) and total nucleated cell count (>8-200 cells/μL and predominantly lymphocytes) being demonstrated most frequently.
- Hematology can reveal evidence of chronic inflammation, as reflected by leukocytosis, hyperfibrinogenemia, and mild-moderate anemia.
- Radiography, nuclear scintigraphy, or transrectal ultrasound examination may help to rule out trauma to the tail head or head.
- The presence of circulating antibodies to myelin protein P2 in serum is good supportive evidence of polyneuritis equi. Increases in the concentration of anti-P2 antibodies in the CSF of some affected horses also have been demonstrated. However, the same antibody has been detected in horses with EHV 1 and equine adenovirus infections. Also, assays to test for P2 myelin antibody are not readily available.
- Definitive diagnosis is by postmortem examination.

DIFFERENTIAL DIAGNOSIS

- Cranial or sacrococcygeal trauma
- Equine herpesvirus myeloencephalopathy
- Equine protozoal myeloencephalitis
- Vertebral osteomyelitis
- Verminous myelitis
- Equine cervical stenotic myelopathy
- Equine motor neuron disease
- Rabies

TREATMENT

- There is no effective specific therapy for polyneuritis equi. Because nerve root degeneration apparently is irreversible by the time of clinical presentation, there usually is little response to immunosuppressive therapy using glucocorticosteroids or cytotoxic drugs such as cyclophosphamide and azathioprine.
- Supportive therapy involves attending to feed and water requirements, manual evacuation of the rectum, catheterization of the bladder (see Equine Herpesvirus Myeloencephalopathy), and appropriate antibiotic therapy for cystitis. Unfortunately, the disease progresses insidiously despite therapy in most cases and prognosis usually is poor.
- Euthanasia should be recommended in severely affected horses.

Rabies

Rabies is a disease associated with profound public health implications because it can occur in all warm-blooded domestic species and humans. Equine rabies has been reported in most countries although England, New Zealand, Australia, Hawaii, and some Caribbean and Pacific islands are free from the disease. While mandatory vaccination of dogs has made the disease rare in North America, it remains a significant threat to horses in South and Central America.

In horses, rabies virus normally is spread by bites from infected wild or domestic animals, particularly raccoons, skunks, bats, dogs, cats, other horses, and probably foxes. Rabies virus also can be transmitted by droplet inhalation, orally, or transplacentally. Inoculation of rabies-infected saliva into myocytes results in variable periods of viral replication followed by infection of peripheral motor and sensory nerves. This initial infection of nerves produces local hyperactivity, reflected by hyperesthesia and tremors. The virus ascends neuronal axons and once within the central nervous system rapidly multiplies and spreads throughout the spinal cord, brain, and sympathetic trunk, as well as passively moving into CSF and blood. High concentrations of virus particles subsequently are released in saliva, making it extremely infectious.

There are three main forms of the disease.

However, combinations of the three syndromes frequently are encountered. When there are signs of cerebral disease, the disorder is referred to as the *furious form* of rabies. Signs reflecting predominantly brainstem disease are referred to as the *dumb form.* A third manifestation of the disease relates to the spinal cord and is referred to as the *paralytic form* of rabies. The dumb and paralytic forms of the disease most commonly affect horses.

HISTORY AND PRESENTING SIGNS

- No age, gender, or breed predilection
- History of a bite by an infected wild or domestic animal, although the bite may be difficult to find or may occur months before the onset of signs
- Occurrence sometimes coincides with epizootics in sylvatic reservoirs or when wildlife populations are at a peak
- Very early signs may include intense rubbing or biting, which may appear as self-mutilation
- Behavioral changes may include aggression and indiscriminate attack
- Other presenting signs include colic, signs of depression, sweating, drooling, obscure lameness, gait abnormalities, and recumbency

CLINICAL FINDINGS AND DIAGNOSIS

- In all cases, progression of the disease is rapid, with death from cardiac or respiratory arrest normally occurring within 5 to 10 days of the onset of signs.

KEY POINT

In general, the incubation period and clinical signs are related to the dose and pathogenicity of the viral strain, the host's immune status, and the proximity of the bite wound site to the central nervous system.

- In the cerebral or furious form, clinical findings may include aggression, hyperesthesia, photophobia and extraocular muscle spasms, blindness, straining, inappetence, and in the terminal stages chewing and seizures.
- With the brainstem, or dumb form, signs of depression, ataxia, circling, head tilt, facial paralysis, and dysphagia with increased drooling of saliva occur.
- In the spinal cord or paralytic form of rabies, the most predominant signs are of progressive ascending paresis or paralysis. Ataxia or shifting lameness with regional hyperesthesia, flaccid tail and anus, urinary incontinence, and a tendency for self-mutilation also may be observed.
- In the paralytic form of the disease, signs will progress to involve the brainstem. Once brainstem signs are present, horses rapidly become recumbent and death ensues. However, in some cases, death may not occur until there is involvement of the cerebrum.

KEY POINT

Rabies should always be considered in any rapidly progressive, multifocal neurologic disorder in a horse. Because there are no pathognomonic signs for rabies, antemortem diagnosis is difficult.

- If rabies is suspected, the clinician must be cautious regarding further handling of the animal, particularly during collection of fluid samples (e.g., blood, urine, milk, or CSF) for diagnostic testing. However, the zoonotic risk of handling these fluids from an affected horse is unknown and currently there are no reports of transmission of rabies from horses to humans.
- There are no specific changes in hematologic variables or serum biochemical analysis.
- Analysis of CSF can be normal or may show xanthochromia (especially early in the course of the disease), moderate increase in total protein concentration (0.6-2.0 g/L or 60-200 mg/dL), and a pleocytosis, with predominance of lymphocytes and macrophages.
- Fluorescent antibody tests on CSF, skin, tactile hairs, cornea, saliva, or salivary gland can be performed but have limited usefulness because of false-positive and false-negative results and difficulties in their interpretation. Samples should be collected, chilled, and sent to a laboratory capable of performing the test. Only positive results are of diagnostic value.
- Serum and CSF neutralization tests also are of limited use because of similar problems with interpretation.

KEY POINT

In a dead horse, the head (and possibly the spinal cord if only spinal cord signs are present) should be shipped unfrozen on ice to a diagnostic laboratory equipped to handle tissues from rabies suspects.

- The laboratory should be contacted regarding the most appropriate tissues and methods of handling, storage, and transportation. Diagnosis is based on the presence of fluorescent antibodies and histopathologic demonstration of intracytoplasmic negri bodies, although not all horses

show these viral inclusions. However, horses that survive 4 or more days are more likely to have negri bodies in neural tissues.

DIFFERENTIAL DIAGNOSIS

- Togaviral encephalitides
- Equine protozoal myeloencephalitis
- Equine herpesvirus myeloencephalopathy
- Hepatoencephalopathy
- Cranial or spinal cord trauma
- Botulism
- Bacterial meningitis
- Leukoencephalomalacia
- Nigropallidal encephalomalacia
- Verminous encephalitis
- Neuritis of the cauda equina
- Parasitic thromboembolism
- Equine stenotic myelopathy
- Choke
- Neoplasia

TREATMENT

- There is no successful treatment for rabies Supportive and anti-inflammatory therapy may prolong the course. To avoid the risk of exposure to people and other animals, horses suspected of having rabies should be isolated and handled as little as possible.

KEY POINT

Management of a rabies suspect should be undertaken in consultation with state or federal authorities.

- Vaccinated or unvaccinated horses that have been bitten by a wild animal should be considered exposed to rabies, regardless of whether the wild animal is available for testing. Exposed vaccinated horses should be revaccinated and observed for 45 days. An exposed but unvaccinated horse probably should not be vaccinated but be kept quarantined for observation for 6 months. All wounds should be cleaned with an appropriate disinfectant (e.g., iodine) and rabies antiserum, if available, infiltrated around the bite wound. If a rabies-exposed horse subsequently develops neurologic signs, it should be euthanized.
- *Prevention* of equine rabies is based on vaccination with killed-virus vaccines given intramuscularly into the thigh. In areas where the disease is endemic, vaccination is recommended at 3 months or older, followed by a second dose at 1 year of age, and annual boosters thereafter. Mares should receive boosters before breeding rather than during pregnancy.

KEY POINT

Veterinarians, veterinary students, animal health technicians, and animal caretakers should be routinely vaccinated against rabies.

Togaviral Encephalomyelitides

Infection with alphaviruses is responsible for induction of eastern (EEE), western (WEE), and Venezuelan (VEE) equine encephalomyelitis. These viruses have birds, rodents, and reptiles as their reservoir host, with mosquitoes being responsible for spread of the virus to horses.

In general, disease associated with EEE, WEE, and VEE is restricted to the Western Hemisphere and ranges from temperate to desert climates. However, disease has been identified in the Philippines and possibly in Europe. The viral encephalitides have a reasonably predictable geographic distribution, with cases of EEE and WEE being most common in the United States (US). As the names of the diseases imply, WEE occurs in western, midwestern and southern US, whereas EEE is found in the eastern US. There is a large area, approximately 1600 km (1000 miles) wide, down the center of the US where both diseases occur. In contrast, VEE only occurs in the most southern regions of the US. All three viral encephalitides occur in Mexico and South America.

KEY POINT

The range of positive serology for the viruses often is far greater than the range of clinical disease.

Spread of the disease is dependent on the presence of the reservoir hosts and insect vectors. As a result, in the US, viral encephalitides can occur year-round in the southeast, whereas they are more common in the north during the summer and fall (June to November).

HISTORY AND PRESENTING SIGNS

- No apparent age or gender susceptibility, although viral encephalitides are rare in young foals
- Acute onset of lethargy, signs of depression, and colic
- Signs progress to include alterations in behavior and other neurologic abnormalities

CLINICAL FINDINGS AND DIAGNOSIS

- Affected horses may present with similar or different clinical signs depending on the type of virus, the ability of the host to clear the virus, and the pathogenicity of the virus. Also, signs are more profound in unvaccinated animals.
- Persistent fever, anorexia, and signs of depression occur acutely and may last for up to 5 days. While many cases of WEE do not progress beyond this point, progression is more common in horses with EEE and VEE.
- Following acute infection, cerebral signs such as head pressing, behavioral changes, circling, blindness, seizures, and coma may develop although these signs can manifest at any time. These signs are the result of cerebral invasion, necrosis, and inflammation in response to the virus.
- In horses affected with VEE, diarrhea, signs of depression, recumbency, and death may precede neurologic deficits. Other findings associated with VEE include abortion, oral ulceration, and pulmonary hemorrhage and epistaxis.
- Infection with EEE and VEE often is fatal, whereas death following WEE is less frequent.

KEY POINT

Human disease often coincides with, or is preceded by, equine epizootics. Clinical signs in humans include fever, headache, stupor, and seizures, with mortality rates highest in cases of EEE and lowest with WEE and VEE.

- Diagnosis is based on the history, with emphasis being focused on the time of the year and geographic location, the clinical signs, and the results of diagnostic tests.
- Analysis of the CSF reveals xanthochromia, increased total protein (>1.5 g/L or 150 mg/dL), and leukocytosis (50-400 X 10^6/L or 50-400/μL). With EEE, the leukocytosis is often the result of a neutrophilia, whereas a monocytic pleocytosis tends to occur with WEE.
- *Serology* may be useful in assisting with diagnosis. However, seroconversion and peak titers usually have occurred by the time encephalitic signs occur. Therefore, a fourfold increase in titer between serum samples collected when encephalitic signs are present and those collected 10 to 14 days later may not occur.
- A combination of serologic tests (e.g., complement fixation, hemagglutination inhibition, and neutralizing antibody assays) increases the likelihood of diagnosis. If increased titers exist for all three tests in an unvaccinated horse with clinical signs of cerebral disease, then a presumptive diagnosis can be made on a single serum sample.
- In horses suspected of VEE, an ELISA is available and can detect viral-specific IgM antibodies to surface glycoprotein by 3 days after the onset of clinical signs.
- *Postmortem* analysis of tissues reveals relatively characteristic changes (e.g., neuronal degeneration, mononuclear cell meningeal inflammation, gliosis) in brain and sometimes spinal cord. Fluorescent antibody, ELISA, and viral isolation are useful in identifying the offending virus in central nervous system tissues.

DIFFERENTIAL DIAGNOSIS

- Other togaviral encephalitides
- Hepatoencephalopathy
- Trauma
- Equine protozoal myeloencephalitis
- Bacterial meningitis
- Rabies
- Leukoencephalomalacia
- Verminous encephalitis

TREATMENT

- There is no specific therapy for the viral encephalitides. As a result, treatment is supportive and mainly involves nursing care.
- NSAIDs such as flunixin meglumine (1.1 mg/kg q12-24h; Treatment No. 52) or phenylbutazone (2.2-4.4 mg/kg q12-24h; Treatment No. 89) is indicated to control pyrexia, inflammation, and discomfort. In addition, slow intravenous administration of DMSO (1.0 g/kg IV as a 10 to 20% solution q12-24h for 1 to 3 days; Treatment No. 34) appears to provide some anti-inflammatory and analgesic effects. Use of corticosteroids for short periods early in the disease, such as dexamethasone (0.1-0.2 mg/kg IV q6-8h for 1-2 days; Treatment Nos. 29 and 30), may be of benefit although there is risk of development of secondary bacterial infections and laminitis.
- If seizures are occurring, administration of diazepam (0.05-0.5 mg/kg IV as required; Treatment No. 32) or phenobarbital (4-10 mg/kg IV slowly; Treatment No. 88) often is useful.
- Attention to maintenance of hydration and nutritional status is important. Administration of mineral oil (Treatment No. 77) may be indicated to assist in prevention of gastrointestinal impaction. Anorexic horses can be fed pelleted gruels via stomach tube (see Chapter 18) or dietary supplements (Osmolite HN, Ross Laboratories, Columbus, OH).
- Full recovery from the viral encephalitides is

rare. If neurologic signs are present, mortality is common, being greater than 75 to 100% for horses with EEE, up to 50% for WEE, and up to 80% for VEE.
- *Prevention* of these diseases has been afforded by insect-control programs and vaccination.
- Insecticides and repellants should be used when practical and standing water should be removed.

KEY POINT

Administration of formalin-inactivated vaccines (e.g., monovalent, divalent, or trivalent vaccines containing EEE, WEE, or VEE) to horses at risk provides good protection (Treatment No. 109).

- However, there is increased specific antibody production to all viruses when trivalent vaccines are administered.
- If vaccinating for protection against VEE, a trivalent vaccine containing inactivated EEE, WEE, and VEE should be given. Vaccination should always be conducted in the spring before the insect numbers increase. In endemic areas, or if there is a prolonged presence of vectors, vaccination should be repeated every 4 to 6 months. Mares should be vaccinated in the month prior to foaling.
- Vaccination in the face of an outbreak is indicated.

Verminous Myelitis/Encephalitis

A number of species of internal parasites have been documented to occasionally undergo aberrant migration through the spinal cord and brain of horses. Parasites reported within the central nervous system include *Strongylus vulgaris, Draschia megastoma, Hypoderma* spp., *Setaria* spp., and *Halicephalobus deletrix* (formerly *Micronema deletrix).* Reports of infection with the latter organism recently have increased, particularly in North America. Horses acquire *H. deletrix* from contaminated soil, and penetration across the mucosae of the oral and nasal passages is the likely route of infection.

HISTORY AND PRESENTING SIGNS

- No breed, age, or sex predilection
- Usually an acute onset of signs that generally is progressive
- Dramatic alterations in neurologic function are commonly reported

CLINICAL FINDINGS AND DIAGNOSIS

- Clinical findings in verminous myelitis depend on the number and size of parasites involved and on the sites and duration of migration.

KEY POINT

Signs are highly variable and include blindness, cranial nerve deficits, head tilt, depression, circling or aimless walking, seizures, ataxia, weakness, recumbency, and death.

- In most circumstances, verminous myelitis results in asymmetrical and multifocal neurologic signs.
- Horses infected with *H. deletrix* also may develop signs associated with nasal and oral granulomas, arthritis, granulomatous nephritis, as well as central nervous system disease.
- Although an increase in (β-globulin concentrations has been documented with parasitic infestations, no characteristic hematologic changes generally are observed in horses with verminous myelitis. Rarely, *H. deletrix* larvae are found in urine, whereas the presence of additional *Hypoderma* larvae subcutaneously might support the diagnosis.
- CSF analysis sometimes provides useful information. Xanthochromia, increased total protein, and elevations in the total number of cells (including neutrophils and eosinophils) may occur. These changes are more commonly identified early in the course of the disease.

KEY POINT

Diagnosis is difficult to establish antemortem and relies on ruling out other common causes of spinal cord or brain disease. History of acute onset of neurologic signs, clinical signs revealing asymmetrical deficits, and CSF findings can assist the clinician in compiling a list of differential diagnoses, of which verminous myelitis may be under strong consideration.

- Definitive diagnosis usually is only made following necropsy and histopathologic examination of central nervous system tissues.

DIFFERENTIAL DIAGNOSIS

- Equine protozoal myeloencephalitis
- Equine herpesvirus myeloencephalopathy
- Equine degenerative myeloencephalopathy
- Trauma

- Viral encephalitides
- Equine stenotic myelopathy
- Vertebral osteomyelitis
- Leukoencephalomalacia
- Brain abscess
- Rabies
- Neoplasia
- Congenital abnormalities

TREATMENT

Since a definitive diagnosis is rare, treatment tends to be empirical and supportive.

KEY POINT

Accurate information regarding dose and efficacy of antiparasitic drugs for the treatment of parasitic migration through the central nervous system of horses is limited.

- Fenbendazole (60 mg/kg once or 50 mg/kg q24h for 3 days or 7.5 mg/kg q24h for 5 days PO; Treatment No. 51), thiabendazole (440 mg/kg q24h for 1 or 2 days) and diethylcarbamazine (DEC; 20 to 40 mg/kg PO for 1 to 3 days) all have been recommended. Organophosphates (e.g., trichlorfon at 40 mg/kg PO once) have been used with apparent success in *Setaria* infections.
- Although ivermectin (0.2 mg/kg PO once; Treatment No. 62) has good efficacy against most parasites, it is not recommended in treating parasite migration because the drug may take up to 2 weeks to kill larvae. However, in less severe cases, ivermectin still may be beneficial.
- In acute neurologic disease, concomitant anti-inflammatory therapy is essential. Good choices include flunixin meglumine (1.1 mg/kg IV q24h; Treatment No. 52) or phenylbutazone (4.4 mg/kg PO q24h; Treatment No. 88). Dexamethasone (0.1-0.2 mg/kg IV q24h for 3-4 days; Treatment Nos. 29 and 30) also can provide benefit. However, if equine protozoal myeloencephalitis is strongly suspected, glucocorticosteroids *should not* be administered. Intravenous administration of mannitol (0.25 mg/kg IV as a 20% solution q4-6h for 1 day; Treatment No. 68) and/or DMSO (1 g/kg slowly IV as a 10-20% solution q12-24h for 1 to 3 days; Treatment No. 34) may be valuable because increased intracranial pressure is likely.
- Good supportive care, including provision of shelter, water, and palatable, nutritious feeds, is important.
- The prognosis for verminous myelitis must always be guarded. Cases with mild deficits may survive or even return to almost normal function. However, in our experience, many cases have been progressive or resulted in significant residual deficits. In these cases, euthanasia should be considered.

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CHAPTER 15

Endocrinology

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Endocrine disorders are rare in horses and mostly are found in older animals. In most cases, endocrine disorders present with nonspecific signs such as weight loss, lethargy, skin or hair abnormalities, and change in appetite. When such signs occur, endocrine diseases should be on a list of differential diagnoses.

EXAMINATION OF THE ENDOCRINE SYSTEM

The endocrine system primarily consists of the pituitary, adrenal, thyroid and parathyroid glands, and the pancreas. In most cases, these organs are not accessible for examination. However, the thyroid gland situated at the level of the larynx is easily palpable and often visible.

History

Important questions when considering the possibility of endocrine disturbances include

- What breed and how old is the horse?
- Is the problem acute or chronic?
- Have there been increases or decreases in appetite?
- What does the diet consist of?
- Has there been weight loss?
- Are there signs of decreased exercise capacity?
- Have there been signs of lameness or incoordination?
- Has there been a change in water consumption or urine output?
- Is there an alteration in the horse's behavior?
- Is there a change in the appearance of the skin or hair coat?

Physical Examination

In endocrine diseases, the nonspecific nature of the history and clinical signs emphasizes the importance of careful examination of all body systems. Specific abnormalities involving the skin, musculoskeletal, and neurologic systems may alert the clinician to an endocrine disorder. For example, in dysfunction of the pars intermedia of the pituitary gland resulting in hyperadrenocorticism (or Cushing's disease), abnormal characteristics of the skin and hair coat (e.g., hirsutism, failure to shed, patchy alopecia, and excessive sweating) frequently are found on physical examination. Chronic, recurrent laminitis or sole abscesses and in some cases neurologic dysfunction (e.g., blindness or diminished responsiveness to pain) also are observed with hyperadrenocorticism. As well, quantitation of urinary output and water intake are important in the investigation of diabetes insipidus, tumors of the adrenal medulla (pheochromocytoma), pancreatic diseases, and pituitary dysfunction. Although nonspecific, the presence of a stiff gait and myositis or muscle tremor may be suggestive of hypothyroidism and pheochromocytoma, respectively. In addition, in diseases of the parathyroid glands in which there is a disturbance of calcium homeostasis, signs of ataxia, muscle spasm, and synchronous diaphragmatic flutter may be noted. Finally, in some cases, signs of chronic infectious disease (e.g., pneumonia or sinusitis) may be evidence for decreased immune function as is often observed in hyperadrenocorticism.

DIAGNOSTIC AIDS

If there is a suspicion of endocrine disease, one should commence with a simple clinicopathologic

investigation, followed by more extensive hormonal function testing.

Hematology

The hemogram is an essential part of examination for a potential endocrine disorder. The neutrophil-to-lymphocyte ratio is useful as a crude screening test for alterations in serum Cortisol values. Lowered Cortisol values are associated with a low neutrophil-to-lymphocyte ratio, whereas normal values are around 1.5:1. High values for plasma Cortisol are reflected by neutrophil-to-lymphocyte ratios above 2:1, often with decreased numbers of eosinophils in the peripheral circulation. However, in many endocrine disorders, horses have normal complete blood counts (as well as serum/plasma biochemical profiles).

Serum/Plasma Biochemical Analyses

Serum or plasma biochemistry profiles may be useful to rule out specific organ diseases, particularly renal and hepatic disease, which may present with similar features to some endocrine diseases. In particular, measurements of glucose, calcium, phosphorus, magnesium, sodium, potassium, and chloride should be monitored by taking sequential samples at times that avoid stress, feeding, and exercise. This will enable clinicians to distinguish between transient reversible conditions and persistent, chronic disorders. Although uncommon in the horse, diseases of the adrenal medulla (e.g., pheochromocytoma), pituitary gland, and pancreas (e.g., chronic generalised pancreatitis) invariably result in persistent hyperglycemia. In contrast, transient hyperglycemic states are relatively common as a result of consuming diets rich in carbohydrates, stress, exercise, obesity, pregnancy, and glucocorticosteroid or α_2-agonist (e.g., xylazine) administration. Normal values for various biochemical measurements are presented in Appendix 2.

Dynamic Hormonal Function Tests

Routine hormonal measurements using either serum or plasma include Cortisol, corticotropin (ACTH), triiodothyronine (T_3), thyroxine (T_4), parathyroid hormone (PTH), and insulin. Reference values for these are given in Table 15—1, together with various suppression or stimulation tests to enable assessment of endocrine function. Before performing an endocrine test, the clinical laboratory should be consulted about availability, validation, and cost of assays and the preferred methods of sample handling. In general, for Cortisol, insulin, PTH, T_3, and T_4, blood samples should be collected into plain (red top) Vacutainer tubes and stored in the refrigerator for 6 to 8 hours to allow clotting. At least 1 mL of serum subsequently is placed into plastic tubes and refrigerated or frozen before delivery to the laboratory, preferably within 48 hours of collection. For ACTH, blood samples should be collected into sodium EDTA (purple top) Vacutainer tubes, transferred to plastic tubes, and centrifuged within 3 hours of collection. These first two steps can be done at ambient temperature, without significantly affecting the ACTH measurement. Separated plasma should be frozen at — 20°C and transported, preferably on dry ice, to the appropriate laboratory.

KEY POINT

Measurement of any plasma or serum hormone concentration at a single point is not recommended to confirm a diagnosis of endocrine disease. This is because of the wide range in reference values and several nonendocrine factors (e.g., time of day, stress, recent meal, diet, and certain drugs) that may result in falsely high or low concentrations of the hormone (in particular Cortisol, insulin, T_3, and T_4).

DEXAMETHASONE SUPPRESSION TEST

Evaluation of the capacity for negative feedback of Cortisol on the secretion of ACTH from the pituitary gland can be performed safely and efficiently using the overnight dexamethasone suppression test:

- Blood (serum) is drawn for baseline Cortisol between 4 and 6 PM.
- Dexamethasone at 40 (μg/kg (20 mg for a 500-kg horse) is administered intramuscularly.
- A postdexamethasone blood sample is collected between 10 AM and noon the following day (approximately 19 hours after dexamethasone administration).

Interpretation. In normal horses the predexamethasone plasma Cortisol concentration is >2 μg/dL (20 ng/mL), whereas 19 hours after dexamethasone injection, serum Cortisol concentrations are decreased by approximately 80% (or is < 1 μg/dL [10 ng/mL]). A serum concentration of Cortisol > 1 μg/dL 19 hours after dexamethasone injection is considered diagnostic for Cushing's disease.

KEY POINT

The overnight protocol is an excellent and convenient screening test. However, a

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TABLE 15—1. Normal Hormonal Values and Endocrine Function Tests in Horses

Test	Reference Value or Normal Response
Serum (or plasma) Cortisol	<13 μg/dL
Plasma ACTH	<32 pg/mL for horses and <14 pg/mL for ponies
Serum insulin	Stallions and colts: 4.9-28.0 μIU/mL Geldings: 5.5-143.6 (μIU/mL Pregnant mares: 6.3-45.5 μTU/mL Nursing foals: 8.7-22.8 μIU/mL
Serum PTH	Mares: 0.32-0.52 ng/mL Geldings: 0.03-0.9 ng/mL Welsh ponies: 0.27-0.64 ng/mL
Plasma norepinephrine	Calm ponies: 140-450 pg/mL Excited ponies: 400-1200 pg/mL Horses: 120-300 pg/mL
ACTH stimulation test	
1 IU/kg ACTH gel IM or 100 IU synthetic ACTH IV	Doubling or trebling of Cortisol values by 2-4 h
Dexamethasone suppression test	
0.04 mg/kg given IM	80% suppression in serum Cortisol by 19 h (or Cortisol concentration < 1 μg/dL)
Glucose tolerance test	
0.5 g/kg of 50% dextrose solution IV	Threefold increase in plasma glucose and fivefold increase in serum insulin level by 15-30 min, returning to baseline in 1.5-3 h for glucose and 3-5 h for insulin
Insulin tolerance test	
0.4 IU/kg protamine zinc insulin	76% decrease in glucose
0.05 IU/kg regular insulin	30-45% decrease in glucose at 15 min, 60% decrease at 30 min with normal values at 2 h
Serum T_3	90 ± 20 ng/dL (foals 100-300 ng/dL)
Serum T_4	1.8 ± 0.8 μg/dL (foals 3-5 μg/dL)
TSH response test	
2.5-5 IU TSH IV	Twofold increase over baseline in serum T_4 and T_3 at 4 to 6 h
5 IU TSH IV in foals	50% increase in T_3 above baseline
TRH response test	
0.5-1.0 mg TRH IV	Twofold increase over baseline in serum T_4 and T_3 at 2 and 4 h
Serum Cortisol before and 15 and 90 min after 1 mg TRH IV	No change or slight decrease in Cortisol levels

Sources: Beech, J.: Tumors of the pituitary gland (pars intermedia). In N.E. Robinson (ed.), *Current Therapy in Equine Medicine 2.* Philadelphia: W.B. Saunders, 1987, with permission.

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standard protocol in which a baseline Cortisol sample is drawn at midnight followed by plasma samples taken 8, 12, 16, 20, and 24 hours after intramuscular injection of 40 μg/kg dexamethasone can be used to assess the degree of loss of pituitary function or improvement in treated cases. Normal horses have concentrations of Cortisol / μg/dL 24 hours after administration of dexamethasone.

PLASMA ENDOGENOUS ACTH CONCENTRATION

Although sampling of plasma for ACTH determination requires special handing (see above), mea-

surement is valuable and accurate in the diagnosis of Cushing's disease. Blood samples should be collected from horses resting quietly in box stalls or yards between 9 AM and noon.

Interpretation. Plasma ACTH values >50 pg/mL for horses and >27 pg/mL for ponies are highly suggestive of Cushing's disease. However, plasma ACTH values between 32 and 50 pg/mL in horses and ACTH concentrations between 14 and 27 pg/mL in ponies are equivocal.

ACTH STIMULATION TEST

Adrenocortical insufficiency (hypoadrenocorticism) can be demonstrated using the ACTH stimulation test:

- Blood (serum) is drawn for pre-ACTH Cortisol concentration between 8 and 10 AM.
- A dose of 1 IU/kg (500 IU/500-kg horse) ACTH gel (corticotropin gel; Adrenomone, Burns Biotec Laboratory, Oakland, CA) is administered intramuscularly or 100 IU (or 1 mg) of synthetic ACTH (Cosyntropin; Cortrosyn, Organon, Inc., West Orange, NJ) intravenously.
- Serum samples for measurement of Cortisol concentration are taken at 2 and 4 hours after ACTH administration.

Interpretation. In normal (and some Cushing's disease) horses, the pre-ACTH baseline Cortisol concentration is 1 μg/dL, whereas the post-ACTH Cortisol concentration should be two to three times higher than baseline. A concentration of Cortisol below 1 (μg/dL with little variation after ACTH stimulation is indicative of adrenal insufficiency.

KEY POINT

Although some cases of Cushing's disease show an exaggerated (more than fourfold) increase in Cortisol concentrations 2 hours after ACTH stimulation, this test does not adequately distinguish between normal horses and those with Cushing's disease. As well, although a combination of dexamethasone suppression and ACTH stimulation tests has been advocated as a practical means of evaluating the pituitary-adrenal axis, these tests also can fail to distinguish normal horses from those with Cushing's disease and cannot be considered diagnostic.

SERUM ENDOGENOUS INSULIN CONCENTRATION, GLUCOSE TOLERANCE TEST, AND INSULIN TOLERANCE TEST

In horses with *persistent hyperglycemia* unassociated with stress or drug administration, measurement of fasting insulin concentration may aid in differentiating horses with hyperinsulinemia, from those with hypoinsulinemia. *Hyperglycemic horses with hyperinsulinemia* include cases with insulin resistance such as Cushing's disease, pheochromocytoma, and non-insulin dependent (type 2) diabetes mellitus. *Hyperglycemic horses with hypoinsulinemia* include those with generalized pancreatic disease, which has led to secondary beta cell damage and insulin-dependent (type 1) diabetes mellitus. The glucose tolerance test, with concurrent measurement of insulin concentration, and the insulin tolerance test also can be useful to further define etiology.

Endogenous Insulin Concentration

Determination of insulin concentration should be performed on two or three nonhemolyzed serum samples at different times, in particular avoiding times close to feeding or exercise (by 4-6 hours), except in nursing foals.

Interpretation

1. Hyperinsulinemia—In hyperglycemic horses with insulin resistance, insulin concentrations usually are well above reference range (see Table 15-1).
   - o Cushing's disease, the most common cause of this abnormality, should be investigated using specific testing as previously described. However, the combined glucose tolerance and insulin concentration test or the insulin tolerance test also can be used.
   - o Cases of pheochromocytoma that also may have a degree of insulin resistance can be diagnosed by determination of blood and/or urinary catecholamine concentrations. However, confirmation with a laboratory about the availability and validity of tests for horses is essential.
   - o Type 2 (insulin-resistant) diabetes mellitus should be suspected in horses after exclusion of other causes of insulin resistance.

2. Hypoinsulinemia—Although an extremely rare cause of hyperglycemia in the horse, cases of type 1 diabetes mellitus usually have decreased concentrations of insulin (<4.9 μIU/mL) and may be definitively diagnosed using the insulin tolerance test. In addition, it is possible (although unconfirmed) that horses with pheochromocytoma may have lowered insulin values as a result of catecholamine-induced suppression of insulin secretion.

KEY POINT

In cases where the concentration of insulin is close to the normal range, causes of transient

hyperglycemia should be ruled out before performing an insulin tolerance test, glucose tolerance and insulin concentration test, or other endocrine tests (primarily for Cushing 's disease). If assays for measurement of insulin are unavailable, the insulin tolerance test is most useful to distinguish between pancreatic dysfunction (resulting in type 1 diabetes) and insulin resistance, caused most commonly by Cushing's disease.

Glucose Tolerance Test

In the combined glucose tolerance and insulin concentration tests, 0.5 g/kg (250 g in a 500-kg horse) of 50% dextrose is administered intravenously. Blood (serum) subsequently should be collected at 15, 60, 90, and 180 minutes after the infusion for measurement of glucose and insulin concentrations. This test may be performed in nonfasted horses and is repeatable, fast, and accurate for establishing glucose tolerance status. Also, the test is less subject to confounding factors than the oral glucose absorption test (see Chapter 7).

KEY POINT

//' blood samples for glucose estimation cannot be processed soon after collection, the blood should be taken into tubes containing fluoride oxalate to inhibit glycolysis.

Interpretation. In normal horses there is a threefold increase in glucose and fivefold increase in insulin concentration by 15 minutes after the infusion. Return to baseline occurs within 1.5 to 3 hours for glucose and 3 to 5 hours for insulin. Horses with Cushing's disease have a less than twofold increase in glucose by 15 minutes, and concentrations remain persistently high. Also, insulin concentrations do not increase. In addition, horses with type 1 diabetes mellitus show a similar response with a blunted increase in glucose concentrations without an accompanying increase in insulin concentrations.

Insulin Tolerance Test

Insulin tolerance testing is most useful to differentiate between insulin resistance (e.g., as in Cushing's disease) and type 1 diabetes mellitus from pancreatic dysfunction. *(Note:* Information is unavailable about the response of other causes of insulin resistance to this test.) Fasting baseline plasma glucose measurements are made before intravenous injection of regular (crystalline) insulin (0.05 IU/kg or 25 U for a 500-kg horse) or protamine zinc insulin (0.4 IU/kg or 200 IU for a 500-kg horse). Blood samples for plasma glucose concentrations subsequently are taken every 15 minutes for 3 hours.

Interpretation. Horses with Cushing's disease do not show a significant decrease in blood glucose. In contrast, horses with insulin-dependent diabetes usually show an appropriate decrease in glucose concentrations by 3 hours after insulin infusion. Published reference ranges for insulin in the horse vary depending on age and sex (see Table 15-1).

URINARY CORTISOL/CREATININE RATIO

Measurement of urinary Cortisol/creatinine ratio (Cort:Cr) is a useful although nonspecific screening test for horses that have a history and clinical evidence supportive of a diagnosis of Cushing's disease. A morning urine sample is collected into a plastic container and, after centrifugation, to remove debris, the sample is analyzed or kept frozen at - 20°C.

Interpretation. Normal horses have a urinary Cort:Cr between 4.7 and 16 ($x10^{-6}$), whereas values reported for Cushing's horses range from 7.5 to 52 ($X\ 10^{-6}$). Horses from which equivocal results are obtained should undergo further hormonal testing.

THYROID-STIMULATING HORMONE (TSH) AND THYROTROPIN-RELEASING HORMONE (TRH) STIMULATION TESTS

If T_3 and T_4 are abnormally low in the absence of nonthyroidal factors that influence their concentrations, function tests using TSH and TRH can be used to determine if the thyroid gland is able to respond to stimulation.

KEY POINT

To avoid confounding nonthyroidal factors, function testing should be performed in horses that have been off medication for at least 1 month and have not been exposed to goitrogenic plants, fescue, excess energy, excess protein, or high levels of zinc, copper, and iodine. Unfortunately, at present TSH and TRH are expensive and not easily obtainable for function testing in the horse. Also, there is no assay available for measurement of equine TSH.

TSH Stimulation Test

The TSH stimulation test determines primary hypothyroidism as a result of reduction in functional thyroid tissue:

- Blood is collected into plain tubes for baseline measurement of T_3 and T_4.
- 2.5 to 5 IU of bovine TSH (Sigma Chemical Co., St. Louis, MO) is administered intravenously. Five international units is used in foals.
- T_3 and T_4 are determined at 2 hours and either at 4 or 6 hours post-TSH. In foals, measurement of T_3 only is made 3 hours after TSH injection.

Interpretation. Normal adult horses show an increase in T_3 (more than two times baseline) at 2 hours and an increase in T_4 (more than two times baseline) at 4 and 6 hours. In foals, T_3 should be at least 50% greater than the baseline value. In primary hypothyroidism, TSH insignificantly affects the low baseline of T_3 and T_4 at 2, 4, or 6 hours. Importantly, however, a normal response may be noted in horses with secondary hypothyroidism in which there is a deficiency in TSH as a result of hypothalamic or pituitary problems.

TRH Stimulation Test

Administration of TRH tests both the ability of the pituitary to respond to TRH and release TSH and the thyroid gland's ability to respond to endogenous TSH and produce thyroid hormones. Used together with the TSH response test, the TRH stimulation test is useful to document secondary hypothyroidism:

- Blood (serum) is collected for baseline measurement of T_3 and T_4.
- 0.5 to 1.0 mg of TRH (Sigma Chemical Co.) is administered intravenously.
- T_3 and T_4 concentrations are determined 2 and 4 hours later.

Interpretation. In normal horses, T_3 and T_4 increase (twice the baseline) at 2 and 4 hours, respectively, after TRH administration. A functional problem of the hypothalamus, pituitary, or thyroid exists if no response is observed. However, if there is a positive response to TSH stimulation, then the hypothyroidism is secondary to a hypothalamic or pituitary disorder, resulting in lack of endogenous TSH.

KEY POINT

Apart from stimulation of TSH and thyroid hormone release, the TRH response test also can he used in horses with Cushing's disease because tumors of the pars intermedia of the pituitary gland are stimulated by TRH to produce ACTH or ACTH-like peptides.

Performed in a similar manner to the ACTH stimulation test, the intravenous administration of 1 mg of TRH increases the serum/plasma Cortisol concentration by 50% to 100%, 15 minutes after injection, for up to 90 minutes. In normal horses, the Cortisol concentration decreases slightly after the administration of TRH. This test is safe, excludes the use of exogenous dexamethasone when administration might be undesirable (e.g., in horses with laminitis), and results in higher Cortisol concentrations in cases of Cushing's disease that have Cortisol concentrations within normal reference range.

PARATHYROID HORMONE DETERMINATION

Parathyroid dysfunction should be considered in the differential diagnosis of any horse exhibiting hypercalcemia, hypocalcemia, or calcium/phosphorus ratio imbalance. Serum PTH concentration can be evaluated using a radioimmunoassay (IRMA, Nichols Institute, San Juan Capistrano, CA) that has been validated for horses and should be interpreted in light of serum calcium and phosphorus values. The normal reference range in mares, geldings, and ponies is between 0.03 and 0.9 ng/mL.

Urinalysis

Urinalysis is important in several endocrine diseases and, in particular, to investigate causes of polyuria and polydipsia. As well, renal losses of glucose in horses with hyperglycemia may be documented, whereas fractional urinary excretion of electrolytes (e.g., calcium, magnesium, and sodium) often is essential to better define cases of parathyroid dysfunction or adrenal insufficiency.

Radiologic Evaluation

Although available only in specialized university facilities or referral centers, computed tomography and magnetic resonance imaging have been used in horses to examine the pituitary gland for evidence of enlargement. As well, a technique of ventrodorsal radiography with contrast venography to demonstrate an enlarged pituitary gland has been described.

Endocrine Diseases

ADRENAL DISORDERS

The paired adrenal glands in the horse consist of an outer cortex and central medulla. The adrenal

cortex comprises three zones that produce mineralocorticoids, glucocorticoids, and androgens, respectively, whereas the adrenal medulla produces catecholamines.

Primary diseases of the adrenal cortex or medulla are extremely rare in horses. Several cases of hypoadrenocorticism (or acute adrenocortical insufficiency) in horses, with inadequate production of glucocorticoids, mineralocorticoids, or both, have been reported. As well, there are a few case reports of tumors of the adrenal medulla (pheochromocytomas) with excessive production of catecholamines. In contrast, hyperadrenocorticism as a primary adrenal disorder has not been recognized in the horse, although hyperadrenocorticism as a manifestation of pituitary dysfunction is well documented.

Hypoadrenocorticism (Addison's Disease)

Primary hypoadrenocorticism is a rare disorder in horses. It has been attributed to poor performance as a result of adrenal insufficiency secondary to intense training programs, "turn-out" or "let-down" syndrome noted in horses suddenly rested after prolonged racing seasons, and to adrenal atrophy and suppression secondary to chronic administration of exogenous corticosteroids. In addition, the adrenal glands are shock organs in the horse and consequently can be damaged during bouts of endotoxemia, severe colic, or anaphylaxis, which may predispose to insufficiency.

KEY POINT

Lower than normal serum Cortisol values may be present for up to 1 to 2 weeks after a single 1M dose of long-acting corticosteroids (e.g., triamcinolone). In contrast, as little as 4 mg (total dose) of dexamethasone causes 18 to 24 hours of pituitary-adrenal suppression. Therefore, long-acting corticosteroids must be regarded as more capable of producing adrenal suppression than are short-acting corticosteroids (e.g., dexamethasone or prednisolone).

HISTORY AND PRESENTING SIGNS

- Prolonged corticosteroid administration (e.g., oral or repeated long-acting injectable forms)
- Horse that has recently come off the track or completed some other form of intensive training
- Recent history of severe colic or periods of dehydration
- Signs of depression and weakness
- Reduced exercise tolerance
- Loss of weight
- Dull hair coat

CLINICAL FINDINGS AND DIAGNOSIS

- Apart from the above nonspecific presenting signs, there are few other documented clinical findings. Polyuria and polydipsia have not been reported in horses with hypoadrenocorticism as is often noted in other species.
- Hematologic and biochemical findings may include leukopenia with a neutrophil-to-lymphocyte ratio of less than 1, mild hyponatremia, hypochloremia, hyperkalemia, and/or hypoglycemia. Also, fractional urinary excretion of sodium may be increased, suggestive of a deficiency of aldosterone.
- Baseline Cortisol should be measured, followed by an ACTH stimulation test, as outlined previously. In horses with hypoadrenocorticism, the ACTH stimulation test results in a blunted Cortisol response.

DIFFERENTIAL DIAGNOSIS

- Chronic inflammatory disease, in particular of the respiratory tract
- Endotoxemia and acidosis
- Renal failure
- Hypothyroidism
- Pancreatic disorders

TREATMENT

- Avoidance of high prolonged doses of corticosteroids, gradual withdrawal of the drug, and administration during the morning hours are the best prevention. Although it has been reported that treatment with <40 mg of dexamethasone/day in a 500-kg horse for 30 days or less can be abruptly withdrawn without adverse effects, gradual reduction in dose by 0.01 mg/kg daily over a period of 7 to 21 days is recommended.
- If primary hypoadrenocorticism is definitively diagnosed, therapy includes reduction of environmental stress and prolonged rest supplemented with small intermittent doses of ACTH (i.e., up to 1 IU of ACTH gel intramuscularly).
- Frequent monitoring of clinicopathologic data should be performed. In particular, supportive therapy in the form of fluid, electrolyte, and glucose supplementation may be required.
- Although some clinicians have used mineralocorticoids and glucocorticoids in an attempt to ameliorate the signs of the disorder, such therapy has not been adequately evaluated in the horse.

Pheochromocytoma

Adrenal medullary tumors have been reported infrequently in horses. They are either nonfunctional asymptomatic masses noted incidentally at necropsy or produce clinical signs referable to increased circulating catecholamines. Functional pheochromocytomas are thought to produce either epinephrine or norepinephrine. There is no breed or sex predilection, and although both adrenal glands may be affected, most documented cases of pheochromocytoma in the horse are unilateral and do not appear to metastasize.

HISTORY AND PRESENTING SIGNS

- Usually an older horse (>12 years)
- Excessive sweating in the absence of exertion
- Muscle tremors and anxiety
- Excessive urinating and drinking
- Signs of low-grade recurrent colic
- Exercise intolerance

CLINICAL FINDINGS AND DIAGNOSIS

- Predominant clinical signs are associated with excessive secretion of adrenaline and/or noradrenaline that stimulate α- and β-adrenergic receptors, mediate splenic contraction and vasoconstriction, antagonize or suppress insulin, and promote gluconeogenesis.

KEY POINT

Suspicion of pheochromocytoma should be aroused when a mature horse has episodes of or a sudden onset of sweating, tachycardia, tachypnea, and muscle tremors and signs of apprehension.

- Other frequent findings include polyuria and polydipsia, dilated pupils, diarrhea, ileus with secondary gaseous distension, and abdominal pain, whereas less common signs are cardiac arrhythmias and myopathy.
- Although malignant pheochromocytoma has been reported, tumors rarely metastasize. However, pheochromocytomas are predisposed to hemorrhage, and continual bleeding within the capsule of the adrenal gland may lead to rupture of the tumor, resulting in signs of colic and hemoperitoneum.
- Activation of a previously quiescent pheochromocytoma may be provoked by procedures or events that exert mechanical influences on the tumor (e.g., advanced gestation).
- Findings on clinicopathologic analysis are nonspecific, although in most reported cases there is neutrophilia, lymphopenia, hemoconcentration, increased concentrations of creatine kinase, hyperglycemia with concomitant glucosuria, and low urine specific gravity.
- Compromised renal function may occur as a result of norepinephrine-induced vasoconstrictive renal ischemia. Azotemia, hyponatremia, hyperkalemia, metabolic acidosis, and, rarely, hypocalcemia and hypophosphatemia may be found.

KEY POINT

Antemortem diagnosis of pheochromocytoma has not been made in the horse. All documented cases were unexpected and were diagnosed at postmortem.

- Determination of venous norepinephrine concentrations in normal horses and ponies has been described, although problems associated with sample preparation and sophisticated assay techniques (using high-performance liquid chromotography) somewhat preclude routine use of these assays in a practice setting. Blood samples are collected into EDTA tubes containing an antioxidant (e.g., 100 |xL of 0.25 M sodium bisulfate) to prevent norepinephrine degradation. Samples should be kept on ice for no longer than 1 hour and spun in a refrigerated centrifuge before laboratory analysis.

KEY POINT

Because of the extremely short half-life of the catecholamines in blood, urinary measurements of their breakdown products may be more useful in future.

- Diagnosis also may be aided by confirming persistent elevations in blood pressure. As well, palpation per rectum or ultrasonographic demonstration of an enlarged mass in the craniodorsal abdomen may be possible in cases in which hemorrhage has occurred.

DIFFERENTIAL DIAGNOSIS

- Other causes of colic (e.g., nonstrangulating obstructions, enteritis)
- Rhabdomyolysis
- Acute laminitis
- Hyperkalemic periodic paralysis
- Renal failure
- Type 1 and type 2 diabetes mellitus
- Cushing's disease
- Pancreatic disease

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TREATMENT

- If antemortem diagnosis of pheochromocytoma can be established in horses, surgical removal may be considered in conjunction with administration of α-adrenergic blockers and use of appropriate hypotensive drugs. Tyrosine analogues (e.g., α-methyl tyrosine), which inhibit the rate-limiting step in catecholamine production, also might be used.

Pancreatic Diseases

Acute and Chronic Pancreatitis

Although rare in the horse, diseases of the pancreas may be evident as either acute episodes of severe inflammation or chronic nonspecific destruction secondary to abscessation (e.g., *Streptococcus equi* var. *equi* or *Corynebacterium* spp.), eosinophilic granulomas, or fibrosis due to strongyle migration (generally *Strongylus equinus).* If generalized, chronic pancreatitis sometimes results in beta cell damage and subsequent type 1 (insulin-dependent) diabetes mellitus. In addition, a case of an insulin-producing pancreatic tumor has been reported.

KEY POINT

Although pancreatic disorders are infrequently diagnosed, the actual prevalence of disease may be underestimated. Clinicians should be alert to the possibility of pancreatic disease in horses that are presented either with acute severe colic or polyuria, hyperglycemia, and glucosuria.

HISTORY AND PRESENTING SIGNS

Acute Pancreatitis

- Acute severe colic
- Signs of depression and shock
- Abdominal distension

Chronic Pancreatitis

- Usually an older horse and in particular in those with an irregular deworming history
- Weight loss or signs of ill thrift
- Signs of depression
- Polyuria and polydipsia
- Polyphagia

CLINICAL FINDINGS AND DIAGNOSIS

Acute Pancreatitis

- Clinical signs of acute pancreatitis are nonspecific and frequently resemble those found in cases of small intestinal obstruction or severe inflammation. Apart from severe abdominal pain, signs of hypovolemic shock including tachycardia, tachypnea, prolonged capillary refill time, congested mucous membranes, and dehydration often predominate. Gastric reflux and reduced gastrointestinal motility invariably are present.
- Documented clinicopathologic findings in horses with pancreatitis confirmed at necropsy include increases in serum amylase and lipase (>700 and 3.5 Sigma-Teitz [S-T] units/L, respectively), hypocalcemia, and increased fractional urinary excretion of amylase (>1%).

KEY POINT

Although reference values for normal horses are reported for serum amylase (15-50 IU/L), serum lipase (<0.2 S-T U/L), and fractional urinary excretion of amylase (<1%), reference ranges should be established for each laboratory because testing methods vary widely.

- Analysis of peritoneal fluid samples may reveal a brown or hemorrhagic color, elevated numbers of neutrophils, and the presence of fat globules. Also, peritoneal amylase concentration, which generally is less than serum concentrations, is increased.

KEY POINT

In general, increases in amylase associated with other causes (e.g., intestinal ileus, intestinal mucosal damage, mesenteric infarction, and conditions resulting in reduced glomerular filtration rate) usually are less than those occurring with pancreatitis.

Chronic Pancreatitis

- In most cases, chronic pancreatic damage is subclinical. However, extensive damage ultimately may result in fibrotic replacement of the islets of Langerhans and hypoinsulinemia (type 1 [insulin-dependent] diabetes mellitus).
- Apart from polyphagia, polyuria, and polydipsia, clinical laboratory analysis consistently reveals persistent hyperglycemia, glucosuria, and ketonuria. However, definitive diagnosis of type 1 diabetes mellitus can be achieved by measuring endogenous insulin concentrations (hypoinsulinemia) and by performing a glucose tolerance test and/or insulin tolerance test. Pituitary function tests for Cushing's disease, prompted by the hyperglycemia, are normal.

KEY POINT

In the single case of an insulin-secreting tumor in a 12-year-old pony, clinical signs included episodic ataxia, disorientation, sweating, dilated pupils, seizures, and generalized clonic muscle contractions. Differential diagnosis included hepatic disease, hypoadrenocorticism, and central nervous system lesions. Hypoglycemia resulting from hyperinsulinemia was isolated to the time of feeding, whereas glucose concentrations were normal at other times of the day.

DIFFERENTIAL DIAGNOSIS

Acute Pancreatitis

- Small intestinal obstruction or enteritis
- Endotoxemia
- Hypoparathyroidism

Chronic Pancreatitis

- Equine Cushing's disease
- Pheochromocytoma
- Hypoadrenocorticism
- Type 2 (insulin-resistant) diabetes mellitus

TREATMENT

Acute Pancreatitis

- Acute pancreatitis should be treated by supportive therapy (see Medical Treatment of Colic, Chapter 7). Large volumes of balanced intravenous fluids with added calcium if necessary, continuous gastric decompression, and analgesics are essential. Broad-spectrum antibiotics may be indicated if secondary bacterial infections become a problem.

Chronic Pancreatitis

- Although there is little published relating to use of insulin in the treatment of type 1 diabetes mellitus, protamine zinc insulin at doses of 0.5 and 1.0 units/kg have been used successfully in the few reported cases.

Parathyroid Diseases

Horses have two pairs of parathyroid glands, usually located in association with the lateral lobe of the thyroid gland and the lower trachea at the level of the first rib. PTH acts to increase calcium and decrease phosphorus concentrations in serum. Secretion is stimulated when serum ionized calcium is decreased (e.g., secondary to decreased dietary intake, increased phosphorus or oxalate intake, and pancreatic disease), when serum magnesium concentration is decreased, due to increased adrenergic stimulation, and as a result of parathyroid adenoma, hyperplasia, or carcinoma.

Primary and secondary hyperparathyroidism and primary hypoparathyroidism have been reported in the horse.

Hyperparathyroidism

- Only a few cases of primary hyperparathyroidism have been reported as a result of parathyroid adenoma, parathyroid hyperplasia, or carcinoma. Clinical and pathologic findings in these cases are not well defined, although invariably there is intermittent weakness, persistent hypercalcemia, and hypophosphatemia and increased fractional urinary excretion of phosphorus. Also, PTH concentration generally is increased.
- Definitive diagnosis can be obtained by histopathology at postmortem examination, but often there is difficulty identifying the parathyroid glands.
- More commonly described are cases of secondary hyperparathyroidism as a sequel to excessive intake of dietary phosphorus and exposure to pastures rich in oxalates. Nutritional secondary hyperparathyroidism is discussed in Chapter 4.
- In addition, neoplastic diseases such as gastric squamous cell carcinoma, lymphosarcoma, and mesothelioma may elaborate a PTH-like product and produce pseudohyperparathyroidism and hypercalcemia.

Hypoparathyroidism

One case of primary hypoparathyroidism has been documented in a 4-year-old Standardbred racehorse. Although a definitive cause for the deficiency in PTH was not identified in this horse, functional hypoparathyroidism due to prolonged hypomagnesemia was suspected. Autoimmune destruction of the glands also may produce primary hypoparathyroidism in the horse.

Presenting signs included severe ataxia in all four limbs, mild colic, facial muscle twitching, and synchronous diaphragmatic flutter.

Initial clinical examination revealed tachycardia and tachypnea, whereas hematologic and biochemical findings included neutrophilia, lymphopenia (corticosteroid-mediated leukogram), hyperglycemia, hypocalcemia, hypomagnesemia, hyperglobulinemia, and metabolic alkalosis. As well, there was a significant and persistent increase in urinary fractional excretion of calcium, phosphorus, and magnesium.

Diagnosis of primary hypoparathyroidism was made on the basis of persistently low serum calcium, magnesium, and PTH concentrations measured in repeated blood samples. Also, administration of magnesium sulphate (5 g in 500 mL of 5% dextrose solution) did not increase PTH levels as had been previously reported in normal horses. Detection of anti-parathyroid antibodies as a means of diagnosing autoimmune destruction of the glands also may be useful for definitive diagnosis if an equine assay becomes available. In addition, horses suspected to be hypoparathyroid may be challenged by administering a chelating agent (e.g., sodium EDTA) and measuring PTH values 30 to 60 minutes after administration. Normal horses should respond with a marked increase in PTH.

Differential diagnoses considered included other causes of hypocalcemia (e.g., idiopathic hypocalcemia, pancreatitis, renal disease, blister beetle or cadmium toxicosis, exertional rhabdomyolysis, and malabsorption syndromes) and other causes of persistent hypomagnesemia (e.g., malabsorption or increased renal excretion due to an unknown mechanism).

Treatment consisted of intravenous infusions of a balanced electrolyte solution with 500 mEq of calcium gluconate as a 23% solution (calcium gluconate 23% solution, Phoenix Pharmaceutical Inc., St. Joseph, MO) added. Because of recurrence of signs of latent tetany (ie., restlessness, tongue rolling, lip and mouth movements) and signs of acute renal failure, administration of calcium-containing fluids (400 mEq calcium gluconate/day in 0.9% NaCl with 20 mEq/L of potassium chloride) was continued for 1 week.

Diet consisted of ad libitum alfalfa hay (~8 kg) supplemented with calcium carbonate (30 g q6h of limestone powder). Maintenance at this dose of calcium was necessary to prevent recurrence of hypocalcemic crises even though the calcium requirement for a normal 500-kg horse is approximately 23 g/day.

Vitamin D_3 (100,000 IU IM) also may be useful in cases of primary hypoparathyroidism.

Prognosis is good if oral calcium replacement therapy is maintained and patients remain clinically stable. Five months after presentation, the horse of this report was clinically normal despite persistent low-grade hypocalcemia.

Pituitary Pars Intermedia Dysfunction (Cushing's-Like Disease)

Equine Cushing's-like disease is caused by hypertrophy and hyperplasia of the pars intermedia of the pituitary gland as a result of a decrease or loss of the neurotransmitter dopamine in the innervation of the pars intermedia. In some cases, this process may progress until expanding adenomas develop. Diffuse enlargement of the pars intermedia is associated with increased secretion of ACTH, (β-endorphin (β-END), and-melanophore-stimulating hormone (α-MSH), which together result in variable elevations in serum Cortisol concentrations and a loss of the circadian rhythmicity of corticosteroid secretion. The complex array of clinical signs noted in Cushing's disease consequently arises as a sequela to excess circulating ACTH (the action of which is enhanced by β-END and α-MSH), the metabolic changes associated with increased glucocorticoid concentrations, and physical destruction of the posterior lobe of the pituitary, the hypothalamus, or the optic chiasma.

HISTORY AND PRESENTING SIGNS

- Usually in older horses (average age 19-21 years) of either sex and any breed, although the incidence appears to be higher in ponies than horses
- Weight loss or muscle wasting, decreased muscle tone, and abnormal bulging of supraorbital fat pads
- Thick, long, and wavy hair coat with abnormal shedding pattern
- Excessive urination and drinking
- Lethargy
- Persistent sweating
- Chronic recurrent laminitis
- Chronic recurrent skin, pulmonary, urinary, or sinus infections
- Vision disturbances

KEY POINT

The presenting complaint for horses with Cushing 's disease frequently is not directly related to the pituitary dysfunction. A careful history and physical examination, in particular in debilitated horses with chronic nonresponsive intercurrent disease problems including laminitis, should lead the clinician to suspect Cushing's disease.

CLINICAL FINDINGS AND DIAGNOSIS

- Approximately 85% of horses affected with long-standing Cushing's disease exhibit hirsutism. The long, thick, curly hair that frequently fails to shed often is preceded by months to years of subtle coat variations. For example,

sparse long hair in the jugular groove, along the chin, or on the legs often is retained after shedding in spring. Also, patchy slow shedding during late spring or delayed shedding with alopecia may be observed.

KEY POINT

The resting Cortisol concentration rarely is elevated in horses with Cushing's disease. Recent evidence suggests that the increased concentrations of ACTH, β-END, and α-MSH and subsequent loss in circadian rhythmicity in Cortisol secretion together *may play a direct, although varied, role in the pathogenesis of many of the clinical and metabolic signs found in Cushing 's disease.*

- For example, the combination of these hormonal factors may lead to insulin resistance (with subsequent hyperinsulinemia, hyperglycemia, glucosuria, and osmotic diuresis) and antagonism of antidiuretic hormone activity (contributing to polyuria and polydipsia). Also, catabolism, which may result in muscle wasting, may be promoted, whereas the digital vasoconstrictive effects of catecholamines may be augmented (contributing to chronic and/or recurrent laminitis with or without associated hoof abscessation). As well, immunosuppression may be promoted, resulting in chronic parasitic disease and infectious disorders such as pneumonia, sinusitis, or periodontitis.
- Other less common clinical findings include distension of the supraorbital fossae, tachypnea, tachycardia, infertility, and rarely hypertrophic osteopathy.
- Other laboratory findings include relative neutrophilia and lymphopenia, whereas in more advanced cases, hyperlipemia with rapidly developing liver failure and coagulopathy may be found.
- Dorsal expansion of the tumor rarely can produce central effects including blindness with mydriasis and interference with antidiuretic hormone (ADH) secretion or thermoregulation (which may explain hirsutism).
- Intermittent hyperhidrosis and anhidrosis also are noted in many horses with Cushing's disease, although the mechanism for these conditions is unknown.
- Diagnostic confirmation of Cushing's disease is made by selective use of screening tests and more definitive dynamic endocrinologic methods. Details of these tests are provided previously.

KEY POINT

The dexamethasone suppression test currently is the most sensitive and specific test available for diagnosis of Cushing's disease. Although the risk of steroid-induced laminitis cannot be underestimated, studies in horses in which the overnight dexamethasone suppression test was used showed no clinical evidence of laminitis or other complications.

DIFFERENTIAL DIAGNOSIS

- Other causes of laminitis
- Renal failure
- Primary or psychogenic diabetes insipidus
- Hyperlipemia
- Type 1 and type 2 diabetes mellitus
- Pheochromocytoma

TREATMENT

- In affected horses that are not persistently hyperglycemic with no evidence of intercurrent infections or laminitis, regular dental care (every 6 months), deworming (every 8 weeks), and hoof care with aggressive treatment of any sole abscesses are all that is required. Good nutritional management (e.g., pelleted, all-in-one type rations) avoiding both obesity and leanness and regular body clipping with appropriate blanketing also are essential.

KEY POINT

With excellent husbandry, feeding practices, vigilance for any secondary complications, and frequent monitoring of blood or urinary glucose, many horses with Cushing's disease have been successfully and inexpensively managed for up to 10 years.

- In horses with Cushing's disease that are hyperglycemic, hyperlipemic, or afflicted with laminitis, apart from the above recommendations, medical intervention is necessary. Currently, the most effective approach is treatment with the long-acting type-2 dopaminergic receptor agonist, pergolide mesylate (Permax, Athena Neurosciences, South San Francisco, CA). Substantial anecdotal evidence supports the use of 0.5 to 2 mg/day PO for a 500-kg horse as an effective and safe means of providing dopamine replacement therapy.
- In general, horses with Cushing's disease are started at 0.001 mg/kg/day PO (0.5 mg/day for a 500-kg horse or 0.25 mg/day PO for ponies) with clinical improvement (i.e., decrease in fre-

quency of urination and drinking, shedding of hair coat, and normoglycemia) observed usually within 6 weeks. If normal blood glucose is not achieved by this time, the dose can be increased in 0.25-mg increments every 3 to 4 days to a maximum of 5 mg/day. Reported side effects include diarrhea, colic, anorexia, and signs of depression.

- Establishing normoglycemia and clinical improvement does not always coincide with return to normal pituitary function as assessed by a dexamethasone suppression test.

KEY POINT

Because the drug is expensive and horses must be maintained on medication for life, the lowest effective dose of pergolide should be found. Most horses respond to doses between 0.75 and 1.25 mg/day, although ponies can be successfully maintained on 0.25 mg/day.

- Bromocriptine, another dopaminergic receptor agonist, also has been used successfully without any side effects at a dose of up to 0.04 mg/kg PO or SC (20 mg for a 500-kg horse) in the morning and 0.02 mg/kg in the evening. However, it has been reported that oral absorption is poor and reactions to subcutaneous injection can occur.
- Cyproheptadine hydrochloride (Periactin, Merck & Co., West Point, PA), an anticholinergic, antihistaminic, and antiserotonergic drug, also has been used with limited success in horses with Cushing's disease. Although serotonin appears to play little if any role in the pathogenesis of Cushing's disease, anecdotal reports of clinical improvement in 35% to 69% of cases suggest that a nonpituitary mechanism may exist. A dose of 0.25 mg/kg/day PO (125 mg/500-kg PO) given once in the morning is safe and relatively inexpensive. If after 2 to 3 weeks there is no response to therapy (i.e., no reduction in blood glucose or no clinical improvement), the dose can be doubled (0.5 mg/kg/day) and given as a divided dose twice daily for a further 4 to 8 weeks. In most horses, polyuria and polydipsia begin to decrease within 1 to 2 weeks of the initiation of therapy. If there is still no improvement, the drug should be discontinued and a dopaminergic agonist or combination therapy considered for use.

KEY POINT

Regardless of relatively poor clinical response, cyproheptadine use continues primarily because of the expense of the dopaminergic agonists.

Thyroid Diseases

The thyroid gland is a freely moveable, round, bilobed structure connected by a narrow fibrous isthmus. Each lobe is approximately 2.5 X 2.5 X 5 cm and in normal horses usually is not visible in the throat region.

Although frequently blamed or cited as a cause of various clinical disorders, naturally occurring thyroid disease in adult horses rarely has been documented. In adults, hypothyroidism has been suggested as a cause of alopecia, anhidrosis, episodic rhabdomyolysis, myositis, exercise intolerance, laminitis, cresty or thickened necks, infertility, agalactia, and poor growth. In neonatal foals, congenital hypothyroidism and dysmaturity syndrome have been well described. There have been no reported cases of equine hyperthyroidism associated with neoplasia or an autoimmune condition in horses. However, a syndrome of tremors, excitability, tachycardia, sweating, and weight loss despite good appetite has been described in racehorses with high serum thyroid hormone concentrations. Improvement was observed after administration of antithyroid treatment (1 g potassium iodide q24h PO). In addition, horses are at risk for accelerated thyroid hormone production as a result of exposure to increased quantities of iodine found in expectorants, contrast media, drugs (e.g., iodochlorhydroxyquin), and leg paints and shampoos containing iodine. Severe distress as a consequence of iatrogenically induced hyperthyroidism may be alleviated with administration of glucocorticoids.

HYPOTHYROIDISM

Hypothyroidism may result from a disruption to any phase of the thyroid (T_3 and T_4)-pituitary (TSH)-hypothalamic (TRH) axis. Primary hypothyroidism is due to inadequate production of T_3 and T_4 from the thyroid gland. It has been documented in horses and foals by a failure to respond as expected to TSH stimulation testing and produced experimentally by surgical thyroidectomy in adults. Secondary hypothyroidism is due to disorders of the anterior pituitary resulting in lowered production and/or release of TSH and subsequent decreased thyroid hormone release. Secondary hypothyroidism has been described in horses in which response to both TRH and TSH tests was abnormal and has been associated with pituitary

dysfunction resulting in typical Cushing's-like clinical signs. Tertiary hypothyroidism, in which severe disease of the hypothalamus results in decreased secretion of TRH, has not been reported in horses.

HISTORY AND PRESENTING SIGNS

Foals

- Full-term foals of any breed or sex in which gestation length usually is prolonged (average 360 days) and premature lactation, dystocia, or abortion may have occurred
- Ingestion by the dam during pregnancy of excess iodine in the form of kelp-containing feed supplements or goitrogenic plants; also ingestion of irrigated pasture or greenfeed containing high levels of nitrate
- Weakness, incoordination, and other signs of dysmaturity (e.g., poor sucking, lax tendons and joints, silky hair coat)
- Flexural deformities and rupture of the common digital extensor tendon

Adults

- Primary hypothyroidism is evident most commonly with exercise intolerance and muscle pain. However, thyroidectomized horses present with retarded growth, decreased feed consumption, increased sensitivity to cold, delayed shedding of hair coat, lethargy, poor exercise performance, rear limb edema, dull hair coat, and a coarse thickened face
- Enlarged thyroid glands in horses fed diets containing deficient or excessive amounts of iodine and in horses with adenoma, carcinoma, or parafollicular cell tumors of the thyroid gland

CLINICAL FINDINGS AND DIAGNOSIS

Foals

- Foals with low serum concentrations of thyroid hormones are characterized by signs of dysmaturity, poor righting reflexes, hypothermia, and sometimes an enlarged thyroid gland. Mandibular prognathia and incomplete ossification of the carpal and tarsal bones are identified in almost all cases.
- Flexural deformities (e.g., tendon contracture) with rupture of at least one common digital extensor tendon frequently are encountered, often in foals with normal T_4 and T_3 levels. Severity and distribution of deformities vary widely between cases, and skeletal lesions may appear a few weeks after birth.
- Incomplete closure of the abdominal wall and poor muscle development, particularly evident over the sternum, also have been reported.

Adults

- Clinical findings in adult horses with primary hypothyroidism include poor cardiac function with decreased resting heart rate, poor performance, and increased electrocardiographic intervals. Lowered body temperature also is noted. Thyroidectomized horses also develop anemia, leukopenia, and hypercholesterolemia but do not become obese or laminitic.
- In most instances, thyroid adenoma is an incidental finding, whereas thyroid carcinoma and parafollicular cell tumors have been associated with work intolerance, weight loss, and respiratory embarrassment.

KEY POINT

Although decreased resting serum T_3 and T_4 concentrations can be used to suggest a diagnosis of hypothyroidism, these tests alone are unreliable in differentiating normal from hypothyroid horses because of the many nonthyroid factors affecting thyroid hormone levels.

Considerable variation may be found in normal values for T_3 and T_4 as a result of

- *Age:* concentrations of T_3 and T_4 in normal foals at birth are extremely high and decline steadily through the first 28 days of life toward adult levels. Therefore, comparison of thyroid hormone concentration from foals suspected of congenital hypothyroidism should be made with those of exactly age-matched controls.
- Sex: concentrations are higher in stallions.
- *Climate:* concentrations are higher in cold weather.
- *Diet:* diets high in carbohydrate, protein, zinc, copper, and iodine (from kelp or seaweed supplementation) and ingestion of endophyte-infected fescue grass can cause decreased T_4 levels.
- *Drugs:* administration of iodine-containing drugs and corticosteroids and phenylbutazone result in reduced T_3 and T_4.
- *Other factors:* long-standing debilitating diseases and food deprivation lower thyroid hormone concentrations, whereas the values of T_3 and T_4 increase in response to training; also there is a mild diurnal variation in T_3 and T_4 concentrations.

For this reason, a diagnosis of primary or secondary hypothyroidism can only be reached by

measuring T_3 and T_4 after conducting a TSH or TRH stimulation test, as described previously. Unfortunately, however, exogenous TSH and TRH are expensive and difficult to obtain except for research purposes. Therefore, the equine clinician must rely on measurement of T_3 and T_4 concentrations to evaluate thyroid function, but the results must be interpreted with caution. In addition, fine-needle aspiration followed by true-cut biopsy is useful for definitive diagnosis of hypothyroidism in foals, thyroid adenoma, carcinoma, and parafollicular cell tumors. Thyroid scintigraphy also has been used to diagnose thyroid adenocarcinoma.

DIFFERENTIAL DIAGNOSIS

Foals

- Neonatal maladjustment syndrome
- Septicemia and toxemia
- Meningitis
- Other lesions of the central nervous system (e.g., hemorrhage, edema)
- Birth trauma or asphyxia
- Other congenital malformations (e.g., arthrogryposis)

Adults

- Cushing's disease
- Retropharyngeal lymph node abscess
- Goiter without neoplasia
- Other causes of poor performance and myopathy

TREATMENT

- Treatment for congenital hypothyroidism and dysmaturity syndrome in foals consists of prevention of failure of passive transfer, splints, physical therapy, and nutritional supplementation. Thyroid supplementation (8.8 X [serum T_4] (μg/L) PO once daily) may be effective in foals with deficient hormone levels, although developmental lesions cannot be reversed. Prognosis varies with the severity of the condition, although future musculoskeletal soundness in surviving foals usually is poor.
- In horses in which hypothyroidism is suspected and low resting concentrations of thyroid hormones are found in the absence of nonthyroidal factors, thyroid hormone supplementation may be beneficial.
- Administration of synthetic sodium levothyroxine (Synthroid, Flint, MI) at a dose rate of 20 μg/kg PO q24h (10 mg for a 500-kg horse) has been reported to be effective. Serum T_4 and T_3 concentrations should be monitored every 30 to 90 days and the replacement dose adjusted accordingly.

KEY POINT

Thyroid hormone supplementation is of no benefit and may be detrimental in horses with low serum T_4 and T_3 concentrations caused by concurrent disease. Also, a positive response to thyroid therapy may actually be a spontaneous resolution of another problem and is not a good basis for confirming a diagnosis of hypothyroidism.

- If therapy is discontinued because of failure of a horse to clinically respond after 6 weeks of supplementation, horses should be weaned from the drug to allow atrophied thyroid tissue to return to normal.
- Iodinated casein also has been described as effective at a dose rate of 5 g PO q24h.
- The iodine requirement for a 450-kg horse is 1 to 2 mg/day, which is easily met using iodized salt or provision of a commercial dietary supplement. However, products with concentrated iodine should be used cautiously or not at all in pregnant mares.
- The treatment of choice for thyroid carcinoma and parafollicular cell tumors is complete surgical removal.

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CHAPTER 16

Practical Clinical Pathology

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Submission of blood and serum for hematologic and biochemical assessment is a routine part of the diagnostic evaluation of many equine cases. Results from these assays may provide a diagnosis, indicate the need for further testing, or assist in clinical management.

KEY POINT

Equine practitioners must recognize the limitations of requested tests and guard against overinterpretation or relying too heavily on test results for a diagnosis. It is essential that a thorough history and complete physical examination be performed before samples for laboratory analyses are submitted. Interpretation of laboratory results must subsequently be correlated with the findings from the history and clinical examination so that a logical plan for further diagnostic tests can be formulated.

The decision to run a "battery of tests" versus a single test frequently is based on economic grounds, although many laboratories now have sophisticated autoanalyzers, making batch testing more cost effective. However, in most circumstances, results of a few specific tests are likely to provide the greatest diagnostic yield.

SAMPLE COLLECTION AND QUALITY

Preparation of samples of blood for hematology is discussed in Chapter 12. Venous blood samples for serum biochemical assays should be collected into clean glass tubes (green-top [lithium heparin anticoagulant] or red-top [no anticoagulant] Vacutainer tubes) and allowed to clot and retract. Samples should be submitted as soon after collection as possible. If storage for 12 to 24 hours is required, serum or plasma should be separated from the red cells *within an hour of collection* and kept refrigerated in a clean plastic vial. This will avoid misleading increases in aspartate aminotransferase (AST), lactate dehydrogenase (LDH), and potassium due to red cell lysis and decreases due to glycolysis. Hemolyzed, icteric, or lipemic plasma samples also may interfere with accuracy of biochemical analysis.

SOURCES OF VARIATION AND CHOICE OF LABORATORY

A number of variables affect results of laboratory tests, including breed, age, sex, and nutritional status of the horse. Current medications being used, sample collection and handling, and the laboratory equipment and methodology also may affect laboratory results. Variation in hematologic tests due to some of these factors is discussed in Chapter 12. The most important variation in biochemical results is due to age. In general, foals have higher values for alkaline phosphatase (ALP), creatine kinase (CK), AST, bilirubin, cholesterol, gamma glutamyltransferase (GGT), glucose, phosphorus, potassium, and triglyceride than adults, whereas lower values are expected in foals for albumin, chloride, creatinine, globulins, total protein, and urea.

The choice of laboratory to analyze submitted samples is also important. Ideally, the laboratory performing the tests should provide

Accurate results derived by the most current analytic methods and validated for the equine species.
Reference ranges derived from clinically normal horses, preferably grouped according to breed, sex, age, and use.
Standardized units of measure with which the clinician is familiar (either SI [mmol/L, g/L, or IU/L] or conventional [mg/dL or mEq/L]). Table 16-1 shows conversion factors for most clinical chemistry data.
Interpretation of results by personnel highly skilled in analysis of cytologic variables.
Prompt and economic service.

CLINICAL CASES

The following case studies were selected to illustrate the principles of interpretation of clinicopathologic data frequently encountered by the equine practitioner. A brief case history and summary of clinical findings is given with results of relevant hematologic and biochemical data. Abnormalities in laboratory data are indicated as mild ( ), moderate ( ), or marked ( ). Values for results not shown should be presumed to be within reference range. The reference ranges used for the interpretation of the data in these cases are given adjacent to the data and also are provided in Table 16-2.

TABLE 16-1. Clinical Chemistry Data: Conversion from Conventional Units to SI Units

Component	Conventional Unit	Conversion Factor	SI Unit
Albumin	g/dL	10	g/L
Alkaline phosphatase (ALP)	U/L	Same	U/L
Aspartate aminotransferase (ALT)	U/L	Same	U/L
Bile acids, total (TBA; fasting)	μmol/L	Same	μmol/L
Bilirubin, total	mg/dL	17.10	μmol/L
Blood gases			
pH			
HCO_3^-	mEq/L	1.0	mmol/L
Pco_2	mm Hg	0.1333	kPa
Po_2	mm Hg	0.1333	kPa
Calcium	mg/dL	0.2495	mmol/L
Chloride	mEq/L	1.0	mmol/L
Cortisol, baseline	μg/dL	27.59	nmol/L
Creatine kinase (CK)	U/L	Same	U/L
Creatinine	mg/dL	88.4	μmol/L
Fibrinogen	mg/dL	0.01	g/L
Gamma glutamyltransferase (GGT)	U/L	Same	U/L
Globulin, total (α, β, and γ)	g/dL	10	g/L
Glucose	mg/dL	0.0555	mmol/L
Iron, serum	μg/dL	0.1791	μmol/L
Iron-binding capacity, total (TIBC)	μg/dL	0.1791	μmol/L
Lactate	mg/dL	0.111	mmol/L
Lactate dehydrogenase (LDH)	U/L	Same	U/L
L-iditol dehydrogenase (L-iDH or SDH)	U/L	Same	U/L
Magnesium	mg/dL	0.4114	mmol/L
Osmolality	mOsmol/kg	Same	mOsmol/kg
Phosphate	mg/dL	0.3229	mmol/L
Potassium	mEq/L	1.0	mmol/L
Protein, total (plasma)	g/dL	10	g/L
Protein, total (serum)	g/dL	10	g/L
Sodium	mEq/L	1.0	mmol/L
Thyroxine, baseline (T_4)	μg/dL	12.87	nmol/L
Triglycerides	mg/dL	0.0113	nmol/L
Triiodothyronine, baseline (T_3)	ng/dL	0.01536	nmol/L
Urea nitrogen (BUN)	mg/dL	0.3570	mmol urea/L

From Duncan, J. R., Prasse, K. W., and Mahaffey, E. A.: Veterinary laboratory medicine. In *Clinical Pathology,* 3rd ed. Iowa: Iowa State University Press, 1994.

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TABLE 16-2. Reference Values for Serum or Plasma Biochemical Measurements

Biochemistry Value*	SI Units (reference range)	Conventional Units (reference range)
Plasma total protein†	60-75 g/L	6.0-7.5 g/dL
Serum total protein‡	57-73 g/L	5.7-7.3 g/dL
Albumin	29-35 g/L (BCG)§	2.9-3.5 g/dL
	27-34 g/L	2.7-3.4 g/dL
Total globulins‡	28-38 g/L	2.8-3.8 g/dL
α-Globulin	7-17 g/L	0.7-1.7 g/dL
β-Globulin	6-20 g/L	0.6-2.0 g/dL
γ-Globulin	8-16 g/L	0.8-1.6 g/dL
Fibrinogen	1-4 g/L	100-400 mg/dL
Urea nitrogen	4.0-8.0 mmol/L	11.2-22.4 mg/dL
Creatinine	100-160 μmol/L	1.13-1.81 mg/dL
Creatinine kinase (CK)	60-330 U/L	60-330 U/L
Aspartate aminotransferase (AST)	160-412 U/L	160-412 U/L
Alkaline phosphatase (ALP)	138-251 U/L	138-251 U/L
Gamma glutamyltransferase (GGT)	10-40 U/L	10-40 U/L
Glutamate dehydrogenase (GLDH)	0-11.8 UL	0-11.8 U/L
Total bile acids (TBA)	5-28 μmol/L	5-28 μmol/L
L-iditol dehydrogenase (L-iDH or SDH)	0-8 U/L	0-8 U/L
Lactate dehydrogenase (LDH)	112-456 U/L	112-456 U/L
Total bilirubin	10-50 μmol/L	0.58-2.92 mg/dL
Sodium	132-142 mmol/L	132-142 mEq/L
Potassium	3.2-4.2 mmol/L	3.2-4.2 mEq/L
Chloride	94-104 mmol/L	94-104 mEq/L
Calcium	2.78-3.32 mmol/L	11.1-13.3 mg/dL
Magnesium	0.58-0.95 mmol/L	1.4-2.3 mg/dL
Phosphate	0.75-1.25 mmol/L	2.3-3.9 mg/dL
Glucose	4.1-6.4 mmol/L	75-115 mg/dL
Triglycerides	0.06-0.61 mmol/L	5.3-54.0 mg/dL
Iron (serum)	13.1-25.1 μmol/L	73-140 μg/dL
Total iron-binding capacity (T1BC)	48.9-72.0 μmol/L	273-402 μg/dL
Pco_2	36-46 mm Hg (arterial)	36-46 mm Hg (arterial)
	38-48 mm Hg (venous)	38-48 mm Hg (venous)
Po_2	80-112 mm Hg (arterial)	80-112 mm Hg (arterial)
	37-56 mm Hg (venous)	37-56 mm Hg (venous)
HCO_3^-	22-29 mmol/L	22-29 mEq/L
PH	7.34-7.47 (arterial); 7.34-7.43 (venous)	7.34-7.47 (arterial); 7.34-743 (venous)

*Unless otherwise stated, measurements were calculated using automated techniques (SMAC Autoanalyser, Technicon, NY).
†Plasma total protein was measured using a temperature-compensated refractometer (American Optical Co, NY).
‡Serum protein electrophoresis was used to determine serum total protein, albumin, and globulins.
§BCG, bromcresol green method.
Data from Duncan, J. R., Prasse, K. W., and Mahaffey, E. A.: *Clinical Pathology,* 3rd ed. Ames: Iowa State University Press, 1994; Jain, N. C: *Essentials of Veterinary Hematology.* Philadelphia: Lea and Febiger, 1993; Kaneko, J. J.: *Clinical Biochemistry of Domestic Animals,* 4th ed. San Diego: Academic Press, 1989; Lumsden, J. H., Rowe, R., and Mullen, K.: Hematology and biochemistry reference values in the light horse. *Can. J. Comp. Med.* 44:32, 1980; Rose, R. J., Ilkiw, J. E., Martin, I.C.A.: Blood gases, acid-base and hematological values in horses during an endurance ride. *Equine Vet. J.* 11:56, 1979.

These cases should be reviewed with the following questions in mind:

- What is your interpretation of results and what are the most significant findings?
- What are your differential diagnoses?
- What (if any) further diagnostic tests would you perform?
- How would you manage this case?

Our suggestions for interpretation of laboratory data are subsequently given. A list of the key

problems obtained from the data is provided. This will assist in identifying critical issues relating to each case and will focus attention on a logical diagnostic plan. A summary at the end of each case gives results of further diagnostic tests and indicates the most likely diagnosis based on all available information.

CASE 1 **Signalment:** 16-year-old Shetland pony stallion used for breeding purposes.
History and Clinical Signs: Low-grade intermittent abdominal pain and anorexia observed for several days. Frequent posturing to urinate with urine dribbling from penis also had been noticed.

Laboratory Data

Component	SI Units (reference range)	Conventional Units (reference range)	Mild/ Moderate/ Marked Change
Hematology			
Packed cell volume (PCV)	0.36 L/L (0.30-0.48)	36% (30-48%)	
Red cell count (RCC)	8.41 X 10^{12}/L (7.5-11.0)	8.41 x 10^{6}/ML (7.5-11.0)	
Hemoglobin	136 g/L (110-160)	13.6 g/dL (11-16)	
Red cell morphology: normal			
White cell count (WCC)	17.3 X 10^{9}/L (6.0-11.0)	17.3 x 10^{3}/ML (6.0-11.0)	
Neutrophils	13.1 X 10^{9}/L (2.5-7.0)	13.1 X 10^{3}/µL (2.5-7.0)	
Band neutrophils	0.35 X 10^{9}/L (0-0.24)	0.35 X 10^{3}/µL(0-0.24)	
Lymphocytes	2.6 X 10^{9}/L (1.6-5.4)	2.6 X 10^{3}/µL (1.6-5.4)	
Monocytes	1.2 X 10^{9}/L (0-0.7)	1.2 X 10^{3}/µL (0-0.7)	
Eosinophils	0 X 10^{9}/L (0-0.5)	0 x 10^{3}/µL (0-0.5)	
White cell morphology: normal			
Platelets	250 X 10^{9}/L (100-300)	250 X 10^{3}/µL100-300)	
Biochemistry			
Blood urea nitrogen	58.3 mmol/L (4.0-8.0)	163.3 mg/dL (11.2-22.4)	
Creatinine	980 µmol/L (100-160)	11.1 mg/dL (1.13-1.81)	
Plasma total protein	86 g/L (60-75)	8.6 g/dL (6.0-7.5)	
Serum total protein	78 g/L (57-73)	7.8 g/dL (5.7-7.3)	
Albumin	22 g/L (29-35)	2.2 g/dL (2.9-3.5)	
Globulin	56 g/L (28-38)	5.6 g/dL (2.8-3.8)	
A:G ratio	0.39 (0.6-1.4)	0.39(0.6-1.4)	
Fibrinogen	7.7 g/L (1-4)	770 mg/dL (100-400)	
CK	459 U/L (60-330)	459 U/L (60-330)	
AST	432 U/L (160-412)	432 U/L (160-412)	
ALP	370 U/L (138-251)	370 U/L (138-251)	
GGT	35 U/L (10-40)	35 U/L (10-40)	
Total bilirubin	58 µmol/L (10-50)	3.39 mg/dL (0.58-2.92)	
Calcium	2.7 mmol/L (2.78-3.32)	10.8 mg/dL (11.1-13.3)	
Phosphate	0.6 mmol/L (0.75-1.25)	1.86 mg/dL (2.3-3.9)	
Sodium	123 mmol/L (132-142)	123 mEq/L (132-142)	
Potassium	7.8 mmol/L (3.2-4.2)	7.8 mEq/L (3.2-4.2)	
Chloride	87 mmol/L (94-104)	87 mEq/L (94-104)	
Triglycerides	2.0 mmol/L (0.06-0.61)	177 mg/dL (5.3-54)	
Blood gases and acid-base analysis (venous)			
Pco_2	45 mm Hg (38-48)	45 mm Hg (38-48)	
$HC0_3^-$	33 mmol/L (22-29)	33 mEq/L (22-29)	
PH	7.5 (7.34-7.43)	7.5 (7.34-7.43)	

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Summary of Problems and Interpretation

1. **Inflammatory leukogram.** Moderate leukocytosis, mild left shift, and monocytosis indicate inflammatory demand. This is supported by the presence of hyperfibrinogenemia and hyperglobulinemia.
2. **Azotemia.** Marked increases in blood urea nitrogen (BUN) and creatinine in the horse can indicate prerenal, renal, or postrenal azotemia or combinations of these mechanisms. Increased concentrations of BUN usually are modest in renal failure in the horse because of intestinal excretion of urea. Creatinine is a more sensitive indicator of renal disease than BUN because the potential for gastrointestinal excretion is less than with urea. High concentrations of BUN and creatinine coupled with severe hyperkalemia in this case supports acute oliguric/anuric renal failure or postrenal urinary tract obstruction or bladder rupture. Prerenal factors (e.g., reduced glomerular filtration rate [GFR] due to hypovolemia/dehydration) may have further increased urea concentration.
3. **Electrolyte derangements.**
   - Primary hyperkalemia (in nonhemolyzed blood samples) may develop as a result of acute renal failure, renal shutdown with metabolic acidosis, urinary tract obstruction or leakage, and during episodes of hyperkalemic periodic paralysis. Marked increases can have profound neuromuscular (in particular, cardiac) effects that are largely the result of changes in cell membrane potential.
   - Common reasons for mild to moderate decreases in sodium and chloride concentrations include diarrhea, excessive sweating, high-volume gastric reflux, renal disease, and sequestration of fluid due to peritonitis or ruptured bladder.
   - Total calcium concentration in serum is approximately 50% ionized, 40% protein-bound (mostly to albumin), and 10% complexed with anions. Only ionized calcium is biologically active. Therefore, in hypoalbuminemia, the albumin-bound fraction of calcium and thus the total serum calcium concentration will be decreased (sometimes referred to as "pseudohypocalcemia"). However, ionized calcium remains normal, and clinical signs of hypocalcemia are not observed.
   - Serum phosphate concentrations are not always an accurate guide to phosphorus balance in the horse, although levels may be decreased in chronic renal failure or due to dietary deficiency.
4. **Hyperproteinemia and hypoalbuminemia.** Moderate increases in globulins and fibrinogen can increase the plasma/serum total protein concentration. Hypoalbuminemia (with decreased albumin/globulin ratio) frequently is associated with hyperglobulinemia as a result of decreased production of albumin by the liver rather than albumin loss. Increased albumin catabolism to provide necessary amino acids for immunoglobulin production secondary to chronic antigenic stimulation also may contribute to decreased albumin concentrations.
5. **Hyperlipidemia.** Concentrations of triglycerides <5.6 mmol/L (or <500 mg/dL) but above reference range are consistent with hyperlipidemia but not with hyperlipemia (i.e., triglyceride concentration >5.6 mmol/L or >500 mg/dL). Any primary disease, particularly in ponies, resulting in anorexia may cause hyperlipidemia that may progress to hyperlipemia.
6. **Hyperbilirubinemia.** Moderate elevations in bilirubin develop in response to anorexia due to reduced hepatic uptake and secretion. As well, minor

hepatocellular damage and cholestasis may cause increased concentrations of bilirubin.

7. **Elevations in CK, AST, and ALP.** Mild elevations in these variables may suggest nonspecific muscle damage (CK, AST) and possibly hepatobiliary damage (AST, ALP). Recumbency, intramuscular injections, transport, and trauma can result in modest release of CK and AST into the circulation without producing histologic evidence of muscle damage.

Key Problems

1. The most significant changes in this case include marked azotemia and hyperkalemia associated with a renal and/or postrenal disorder.
2. The moderate inflammatory response also is significant and probably is related to inflammation of the urinary tract.
3. Also important is hyperlipidemia, which in an anorexic pony can rapidly develop into hyperlipemia with other subsequent complications. Repeated measurements for triglyceride and BUN/creatinine are essential to monitor progression of hyperlipidemia and azotemia/renal function, respectively.

Differential Diagnosis (see also Chapter 10)

- Urolithiasis (urethral, bladder, bilateral renal, ureteral)
- Ruptured bladder
- Urinary tract neoplasia
- Acute or chronic renal failure
- Bilateral pyelonephritis

Suggestions for Further Diagnostic Tests

- Rectal examination. In particular, palpation of left kidney, bladder, and proximal urethra. Urethral calculi are most commonly lodged just below the anus in horses.
- Thorough external palpation of penis and distal urethra.
- Passage of urinary catheter.
- Ultrasound examination performed per rectum and transabdominally of all urinary tract structures.
- Endoscopic visualization of urethra, bladder, and ureters.
- Abdominocentesis and peritoneal fluid creatinine-to-serum creatinine ratio. Peritoneal fluid creatinine concentration is at least two times higher than the serum creatinine concentration in horses with uroperitoneum. Peritoneal fluid potassium concentrations above 10 mmol/L also are indicative of urine in the peritoneal cavity.
- Urinalysis. In particular, measurement of specific gravity and complete cytologic and (if indicated) bacteriologic assessment.

Suggestions for Additional Diagnostic Tests

- Urinary GGT/creatinine ratio. Damage to the brush border of renal tubular cells may lead to release of GGT into the tubular lumen and the urine. However, increases in GGT relative to creatinine in the urine as an index of acute renal tubular damage is of questionable use in the horse, because this test has been considered too sensitive to be of practical value.

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- Fractional excretion (FE) of urinary electrolytes. Increases in the FE of sodium are noted with renal tubular damage and impaired sodium resorption, whereas low sodium FE may indicate prerenal azotemia.

Additional Notes

- Single BUN and creatinine determinations are not reliable prognostic indicators. It is essential to use serial evaluation, noting progressive changes in values and response to treatment.
- Other abnormalities associated with decreased GFR include hypercalcemia, hyperphosphatemia, and acidemia.
- FE of urinary electrolytes (i.e., determination of renal electrolyte clearance relative to endogenous creatinine clearance) can be useful in the detection of renal tubular damage and specific dietary deficiencies (e.g., calcium and sodium deficiency, phosphorus excess). The FE of electrolytes can fluctuate in response to physical activity and after feed and water intake. The reference range is consequently wide.
- Albumin/globulin ratio can be used as an aid to interpretation of total protein values, with abnormalities in the ratio observed if an alteration in one fraction predominates.

Case Resolution

A urethral calculus was visualized on urethral endoscopy at a point located below the anus and was removed via perineal urethrostomy.

CASE 2

Signalment: 4.5-year-old Paint gelding.

History and Clinical Signs: Anorexia, marked ill-thrift, signs of depression, and intermittent recumbency noted over a 2-week period. Icteric gums and dark yellow urine also recently observed.

Laboratory Data

Component	SI Units (reference range)	Conventional Units (reference range)	Mild/ Moderate/ Marked Change
Hematology			
PCV	0.28 L/L (0.30-0.48)	28% (30-48%)	
RCC	8.42 X 10^{12}/L (7.5-11.0)	8.42 X 10^{6}/µL (7.5-11.0)	
Hemoglobin	103 g/L (110-160)	10.3 g/dL (11-16)	
Red cell morphology: marked anisocytosis, poikilocytosis, schistocytosis, and acanthocytosis observed			
WCC	9.96 X 10^{9}/L (6.0-11.0)	9.96 X 10^{3}/ML (6.0-11.0)	
Neutrophils	5.46 X 10^{9}/L (2.5-7.0)	5.46 X 10^{3}/µL (2.5-7.0)	
Lymphocytes	2.5 X 10^{9}/L (1.6-5.4)	2.5 X 10^{3}/ML (1.6-5.4)	
Monocytes	1.9 x 10^{9}/L (0-0.7)	1.9 x 10^{3}/µL (0-0.7)	
Eosinophils	0 X 10^{9}/L (0-0.5)	0 X 10^{3}/ML (0-0.5)	
White cell morphology: normal			
Platelets	100 10^{9}/L (100-300)	100 X 10^{3}/µL (100-300)	

Biochemistry		
BUN	20.1 mmol/L (4.0-8.0)	56.3 mg/dL (11.2-22.4)
Creatinine	155 |xmol/L (100-160)	1.75 mg/dL (1.13-1.81)
Plasma total protein	69 g/L (60-75)	6.9 g/dL (6.0-7.5)
Serum total protein	68 g/L (57-73)	6.8 g/dL (5.7-7.3)
Albumin	23 g/L (29-35)	2.3 g/dL (2.9-3.5)
Globulin	45 g/L (28-38)	4.5 g/dL (2.8-3.8)
A:G ratio	0.5 (0.6-1.4)	0.5(0.6-1.4)
Fibrinogen	0.8 g/L (1-4)	80 mg/dL (100-400)
CK	3337 U/L (60-330)	3337 U/L (60-330)
AST	2080 U/L (160-412)	2080 U/L (160-412)
ALP	826 U/L (138-251)	826 U/L (138-251)
GGT	420 U/L (10-40)	420 U/L (10-40)
GLDH	267 U/L (0-11.8)	267 U/L (0-11.8)
Total bilirubin	173.3 (xmol/L (10-50)	10.1 mg/dL (0.58-2.92)
Total bile acids	90 jjumol/L (5-28)	90 (xmol/L (5-28)
Glucose	2.08 mmol/L (4.1-6.4)	37.5 mg/dL (75-115)
Calcium	2.48 mmol/L (2.78-3.32)	9.94 mg/dL (11.1-13.3)
Phosphate	1.21 mmol/L (0.75-1.25)	3.75 mg/dL (2.3-3.9)
Sodium	140 mmol/L (132-142)	140 mEq/L (132-142)
Potassium	3.84 mmol/L (3.2-4.2)	3.84 mEq/L (3.2-4.2)
Chloride	100 mmol/L (94-104)	100 mEq/L (94-104)

Summary of Problems and Interpretation

1. **Anemia.** Mild anemia most likely is the result of inadequate erythropoiesis associated with chronic inflammatory or neoplastic disorders (and iron sequestration into the reticuloendothelial system). Immune-mediated hemolytic anemia (IMHA) secondary to infection or neoplasia also is possible in this case.
2. **Erythrocyte morphologic changes.**
   - Anisocytosis is variation in erythrocyte size. Macrocytes rarely may be observed in regenerative anemias in horses. Microcytes may be noted with oxidative injury to red cells or in iron or pyridoxine deficiency, in which they may be associated with a low mean corpuscular volume (MCV). Spherocytes associated with immune-mediated anemias also are a type of microcyte, although these cells rarely are observed on smears of horse blood.
   - Poikilocytes are abnormally shaped erythrocytes, examples of which include schistocytes (irregular erythrocyte fragments) and acanthocytes (spiculated erythrocytes). Schistocytes are indicative of microcirculatory changes such as occur in disseminated intravascular coagulation (DIC), neoplasia, or inflammation of highly vascular organs (e.g., liver, spleen, lung, bone marrow). Acanthocytes often are observed in horses with hemangiosarcoma or liver disease.
3. **Marked hepatic damage.** Increased activity of AST and GLDH indicates hepatocellular damage, which can be due to hypoxia, necrosis, neoplasia, lipidosis, infections, and toxins. There is poor correlation between the degree by which AST and GLDH are increased and the extent of liver damage. Elevations in GGT and ALP indicate hepatobiliary or cholestatic disease. Cholestasis in this case also is indicated by hyperbilirubinemia, whereas increased bile acids could be due to cholestasis and decreased functional hepatic mass. Other tissue sources of GGT in horses include pancreas and kidney, whereas ALP also is found in bone, intestine, and placenta. Although rarely observed, low

concentrations of fibrinogen, glucose, and albumin could be due to decreased functional hepatic mass. However, in this case, function apparently has not decreased enough to affect urea synthesis. BUN usually decreases in cases of acute hepatic necrosis or hepatic failure, concomitant with elevation in ammonia concentrations.

4. **Azotemia.** Mild elevations in BUN in the absence of obvious prerenal mechanisms (e.g., dehydration) may be the result of increased protein catabolism and muscle damage caused by recumbency. Creatinine is not significantly affected by catabolic factors but is affected by changes in muscle mass.
5. **Myopathy.** Moderate elevation in CK indicates concurrent muscle damage probably secondary to recumbency or repeated intramuscular injections. Increase in AST activity, therefore, is the result of both liver and muscle damage in this case.

Key Problems

1. The key features of the laboratory data are marked elevations in all liver-related enzymes associated with significant erythrocyte morphologic changes indicating severe chronic hepatic disease.

Differential Diagnosis (see also Chapter 7)

- Pyrrolizidine alkaloid toxicosis
- Chronic active hepatitis
- Serum-associated hepatitis (Theiler's disease)
- Cholangitis or cholangiohepatitis
- Cholelithiasis or other biliary obstruction
- Aflatoxicosis
- Hepatic lipidosis
- Hepatic abscess

Suggestions for Further Diagnostic Tests

- Request measurement of unconjugated (indirect) and conjugated (direct) forms of total bilirubin concentration.
- Liver ultrasonography
- Coagulation profile. Although a rare complication of liver disease in horses, coagulopathy caused by decreased synthesis of clotting factors may increase the risk of hemorrhage associated with liver biopsy.
- Liver biopsy

Suggestions for Additional Diagnostic Tests

- Urinalysis to confirm presence of bilirubinuria and/or myoglobinuria
- Iron analysis. Low serum iron concentration and high serum total iron-binding capacity (TIBC) are consistent with iron deficiency due to chronic external blood loss. Low serum iron with normal TIBC suggests anemia of chronic disease.

Additional Notes

- Myoglobinuria, if present, also may contribute to nephropathy, which may exacerbate azotemia.

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- It is important to note that the presence of dehydration (as determined by clinical variables) also may explain mild azotemia and suggests that the horse was more hypoalbuminemic and anemic than indicated by the laboratory data.
- Most hepatic functions do not become detectably altered until more than 50% of the hepatocellular mass is nonfunctional. For example, 60 to 80% of the functional mass of the liver must be lost before hypoalbuminemia results.
- Although none of the alterations in serum/plasma proteins is entirely specific for liver dysfunction, the combination of hypoalbuminemia and hyperglobulinemia frequently is observed in chronic liver disease. Increased concentrations of globulins (primarily gamma globulins) are the result of excessive antigenic stimulation of lymphoid tissues by enteric-derived foreign protein. Diminished mass and function of hepatic Kupffer cells prevent normal removal of these antigens.
- Although alanine aminotransferase (ALT) is an important liver-specific enzyme in small animal clinical pathology, it has not been shown to be useful for evaluation of liver disease in horses.
- Enzymes that are liver specific or have high concentrations primarily in hepatic tissue include L-iditol dehydrogenase (L-iDH, formerly sorbitol dehydrogenase [SDH]), arginase, and GLDH. These enzymes increase and decrease rapidly in response to a single insult to the liver. Enzymes that are not liver specific and are found in high concentrations in other tissues in addition to the liver include AST, LDH, and ALP**.** These enzymes generally have a longer half-life, and levels should be interpreted in conjunction with other more specific tests.
- Although total LDH concentration is not liver specific, the isoenzyme LDH-5 is a specific indicator of hepatocellular damage. A specific assay is currently available in North America.
- Serum **GGT** is found primarily in biliary tissue and is a good indicator of intra- or extrahepatic cholestasis.
- In general, unconjugated (indirect-reacting) bilirubin predominates in normal horses and in most cases with hyperbilirubinemia. Although both unconjugated and conjugated (direct-reacting) bilirubin may increase in hepatic and extrahepatic diseases, hyperbilirubinemia with >20% conjugated bilirubin indicates an extrahepatic disorder (e.g., biliary obstruction).

Case Resolution

The results of the coagulation profile were within reference limits, eliminating the risk of hemorrhage after liver biopsy. Liver biopsy revealed histologic changes characteristic of pyrrolizidine alkaloid toxicosis.

CASE 3

Signalment: 8-year-old female Thoroughbred in race training.
History and Clinical Signs: Profuse diarrhea, anorexia, and signs of depression noted over 24 hours. There also was a possibility of antibiotics having been given.

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Laboratory Data

Component	SI Units (reference range)	Conventional Units (reference range)	Mild/ Moderate/ Marked Change
Hematology			
PCV	0.57 L/L (0.30-0.48)	57% (30-48%)	t T
RCC	20.6 X 10^{12}/L (7.5-11.0)	20.6 X 10^{6}/µL (7.5-11.0)	T T
Hemoglobin	190 g/L (110-160)	19 g/dL (11-16)	T T
Red cell morphology: normal			
WCC	3.0 x 10^{9}/L (6.0-11.0)	3.0 x 10^{3}/µL (6.0-11.0)	4 4
Neutrophils	0.3 X 10^{9}/L (2.5-7.0)	0.3 X 10^{3}/µL (2.5-7.0)	4- * 4-
Band neutrophils	0.7 x 10^{9}/L (0-0.24)	0.7 X 10^{3}/ML (0-0.24)	T T
Lymphocytes	1.9 X 10^{9}/L (1.6-5.4)	1.9 X 10^{3}/ML (1.6-5.4)	
Monocytes	0.1 x 10^{9}/L (0-0.7)	0.1 X 10^{3}/ML (0-0.7)	
Eosinophils	0 X 10^{9}/L (0-0.5)	0 x 10^{3}/µL (0-0.5)	
White cell morphology: marked "toxic" change of neutrophils and "ring forms" apparent; also difficult to differentiate neutrophils from monocytes			
Platelets	120 X 10^{9}/L (100-300)	120 X 10^{3}/µL (100-300)	
Biochemistry			
BUN	18 mmol/L (4.0-8.0)	50.4 mg/dL (11.2-22.4)	
Creatinine	250 µmol/L (100-160)	2.83 mg/dL (1.13-1.81)	
Plasma total protein	55 g/L (60-75)	5.5 g/dL (6.0-7.5)	
Serum total protein	50 g/L (57-73)	5.0 g/dL (5.7-7.3)	
Albumin	23 g/L (29-35)	2.3 g/dL (2.9-3.5)	
Globulin	27 g/L (28-38)	2.7 g/dL (2.8-3.8)	
Fibrinogen	7 g/L (1-4)	700 mg/dL (100-400)	
CK	145 U/L (60-330)	145 U/L (60-330)	
AST	256 U/L (160-412)	256 U/L (160-412)	
ALP	146 U/L (138-251)	146 U/L (138-251)	
GGT	33 U/L (10-40)	33 U/L (10-40)	
Total bilirubin	65 µmol/L (10-50)	3.8 mg/dL (0.58-2.92)	
Calcium	2.5 mmol/L (2.78-3.32)	10.0 mg/dL (11.1-13.3)	
Phosphate	1.1 mmol/L (0.75-1.25)	3.41 mg/dL (2.3-3.9)	
Sodium	118 mmol/L (132-142)	118 mEq/L (132-142)	
Potassium	4.1 mmol/L (3.2-4.2)	4.1 mEq/L (3.2-4.2)	
Chloride	88 mmol/L (94-104)	88 mEq/L (94-104)	
Blood gases and acid-base analysis (venous)			
Pco_2	35 mm Hg (38-48)	35 mm Hg (38-48)	
HCO_3^-	15 mmol/L (22-29)	15 mmol/L (22-29)	
pH	7.24 (7.34-7.43)	7.24 (7.34-7.43)	

Summary of Problems and Interpretation

1. **Acute inflammation.** Leukopenia due to neutropenia with a degenerate left shift indicates overwhelming tissue demand for leukocytes. Toxic neutrophil changes suggest toxemia, frequently the result of increased absorption of endotoxin. Further evidence for endotoxemia secondary to gram-negative infection (e.g., salmonellosis) is the presence of

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neutrophilic "ring forms." Hyperfibrinogenemia confirms the inflammatory character of the disease.

2. **Severe dehydration and hypovolemia.** Moderate elevations in erythrocyte variables (PCV, RCC, and hemoglobin) suggest serious hypovolemia and dehydration. Although elevations in total protein and albumin usually are noted in horses as a result of fluid loss, in this case, protein did not reflect the degree of dehydration because of concomitant protein loss through an inflamed intestine (see below). Degree of dehydration should be estimated clinically and fluid replacement plans calculated accordingly.
3. **Marked hyponatremia.** Typically, marked losses of sodium and bicarbonate occur in acute diarrhea. Although plasma potassium concentration is normal as a result of shifts from the intracellular fluid (ICF) to the extracellular fluid (ECF) caused by acidemia, whole body potassium will be severely depleted with time, in particular if the horse remains anorexic. This emphasizes the importance of potassium supplementation in maintenance fluids and by oral administration if possible, when hypovolemia is corrected (see Chapter 19).
4. **Azotemia.** Generally, hypovolemia results in prerenal azotemia, although if volume replacement is delayed or insufficient, hemodynamic renal failure with renal azotemia can occur. As well, toxemia and use of renal toxic drugs such as nonsteroidal anti-inflammatory medications (which are commonly used in cases of acute diarrhea) may further exacerbate renal azotemia.
5. **Metabolic acidosis.** Severe metabolic acidosis is characterized by marked decreases in pH and bicarbonate concentration and commonly occurs as a result of gastrointestinal loss of bicarbonate secondary to diarrhea or strangulated bowel. Metabolic acidosis also results from an accumulation of lactate (plus hydrogen ions) associated with toxemia, shock, and ischemia. Initial respiratory compensation is reflected by low Pco_2, whereas long-term correction requires renal bicarbonate retention and enhanced acid secretion.
6. **Protein-losing enteropathy.** Although plasma total protein (TP) increases initially due to hypovolemia, enteric loss of protein, particularly albumin, rapidly occurs as a result of widespread mucosal or endothelial damage. Therefore, in this case, total protein and albumin concentrations were significantly lower than these data indicate because of the effect of hypovolemia.

Key Problems

1. In this case, all the data highlighted above are significant and are indicative of acute severe gastrointestinal inflammation with concomitant toxemia, fluid, and protein loss. Repeat hematologic and relevant biochemical studies (i.e., PCV, TP, electrolytes, creatinine, and blood gases) for purposes of monitoring the horse's response to treatment and the prognosis are critical.

Differential Diagnosis (see also Chapter 7)

- Acute undifferentiated (idiopathic) diarrhea
- Antibiotic-related diarrhea
- Salmonellosis

- Equine monocytic ehrlichiosis (Potomac fever)
- Intestinal clostridiosis *(Clostridium difficile, C. perfringens)*
- Endotoxemia/gram-negative sepsis
- Sudden change in diet/overfeeding
- Toxic causes (e.g., phenylbutazone, blister beetle, oleander, acorn)
- Cyathostomiasis

Suggestions for Further Diagnostic Tests

- Fecal analysis:
  - Bacterial culture for *Salmonella* spp., C *difficile,* and C. *perfringens* (at least three serial samples [20-50 g or 20-30 mL] required)
- Demonstration of clostridial toxins
- Paired sera for *Ehrlichia risticii*

Suggestions for Additional Diagnostic Tests

- Strongyle eggs may be observed on fecal flotation, whereas *cyathostomes* (small strongyles) can be subsequently cultured.
- Rectal biopsy:
  - Encysted larvae in rectal biopsies may indicate diffuse cyathostomiasis.
  - Culture for *Salmonella* spp.
- Detection of *Salmonella* spp. in feces using a polymerase chain reaction.

Additional Notes

- A "degenerative left shift" occurs when the absolute number of immature neutrophils (bands and metamyelocytes) exceeds the number of mature segmented neutrophils.
- A "regenerative left shift" occurs when the absolute number of immature neutrophils is less than the number of mature neutrophils.
- Persistence of neutropenia and a degenerative left shift and hypoproteinemia with development of dependent edema suggest a guarded prognosis.
- Peracute cases of acute colitis may present similarly without diarrhea.
- The clinical presentation and laboratory data above should alert clinicians to the potential for serious complications such as renal failure, laminitis, DIC, and ischemic bowel necrosis, for which immediate preventive measures should be instituted. Care also should be taken when using nonsteroidal anti-inflammatory drugs in horses with hypovolemia because of the high potential of these agents for accentuating renal tubular necrosis and failure.
- Most horses recovering from salmonellosis (or with subclinical infection) cease to shed *Salmonella* over days to weeks (usually 3-16 weeks after acute *Salmonella* colitis). Isolation of the horse from others, in particular neonates, for a period of 4 to 6 weeks or until five negative fecal cultures are obtained is considered safe practice when attempting to prevent cross-infection. *Salmonella* is zoonotic, and attention to hygiene cannot be overemphasized.
- Neutrophilic "ring forms" in smears of horse blood often are observed in acute toxemic disorders, frequently as a result of acute gastrointestinal disorders.

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Case Resolution

The horse was refractory to intensive medical therapy and subsequently developed oliguric renal failure. The owners of the horse requested euthanasia. Culture of *Salmonella typhimurium* from feces and biopsy specimens from mesenteric lymph nodes and colonic contents at postmortem confirmed a diagnosis of salmonellosis.

CASE 4

Signalment: 14-year-old Standardbred gelding used for occasional riding.
History and Clinical Signs: Ill-thrift, signs of depression, inappetence, exercise intolerance, and ventral edema observed over previous 2 weeks. Pale oral mucous membranes and fever of unknown origin also noted on clinical examination.

Laboratory Data

Component	SI Units (reference range)	Conventional Units (reference range)	Mild/ Moderate/ Marked Change
Hematology			
PCV	0.20 LA (0.30-0.48)	20% (30-48%)	
RCC	5.8 x 10^{12}/L (7.5-11.0)	5.8 X 10^6/ML (7.5-11.0)	
Hemoglobin	95 g/L (110-160)	9.5 g/dL (11-16)	
Red cell morphology: normal			
WCC	13.3 x 10^9/L (6.0-11.0)	13.3 X 10^3/µL (6.0-11.0)	
Neutrophils	8.1 x 10^9/L (2.5-7.0)	8.1 x 10^3/|ML (2.5-7.0)	
Lymphocytes	3.2 x 10^9/L (1.6-5.4)	3.2 X 10^3/µL (1.6-5.4)	
Monocytes	1.8 X 10^9/L (0-0.7)	1.8 X10^3/ML (0-0.7)	
Eosinophils	0.2 x 10^9/L (0-0.5)	0.2 X 10^3/ML (0-0.5)	
White cell morphology: normal			
Platelets	45 X 10^9/L (100-300)	45 X 10^3/µL (100-300)	
Biochemistry			
BUN	7.3 mmol/L (4.0-8.0)	7.3 mg/dL (11.2-22.4)	
Creatinine	150 µmol/L (100-160)	1.7 mg/dL (1.13-1.81)	
Plasma total protein	74 g/L (60-75)	7.4 g/dL (6.0-7.5)	
Serum total protein	68 g/L (57-73)	6.8 g/dL (5.7-7.3)	
Albumin	25 g/L (29-35)	2.5 g/dL (2.9-3.5)	
Globulin	43 g/L (28-38)	4.3 g/dL (2.8-3.8)	
A:G ratio	0.58 (0.6-1.4)	0.58 (0.6-1.4)	
Fibrinogen	5.5 g/L (1-4)	550 mg/dL (100-400)	
CK	143 U/L (60-330)	143 U/L (60-330)	
AST	215 U/L (160-412)	215 U/L (160-412)	
ALP	141 U/L (138-251)	141 U/L (138-251)	
GGT	33 U/L (10-40)	33 U/L (10-40)	
Total bilirubin	85.3 µmol/L (10-50)	5.0 mg/dL (0.58-2.92)	
Calcium	2.8 mmol/L (2.78-3.32)	11.22 mg/dL (11.1-13.3)	
Phosphate	0.87 mmol/L (0.75-1.25)	2.7 mg/dL (2.3-3.9)	
Sodium	139 mmol/L (132-142)	139 mEq/L (132-142)	
Potassium	3.3 mmol/L (3.2-4.2)	3.3 mEq/L (3.2-4.2)	
Chloride	101 mmol/L (94-104)	101 mEq/L (94-104)	

Summary of Problems and Interpretation

1. **Anemia.** Moderate anemia may be due to hemolysis or blood loss (regenerative anemias) or inadequate erythropoiesis as a result of

chronic disease/inflammation (nonregenerative anemia). It is important to note that determination of erythrocyte regeneration in the horse is difficult because equine erythrocytes are not released into the circulation until mature, even when there is intense erythropoiesis. Therefore, *reticulocytosis, nucleated red cells, polychromasia, macrocytosis, and anisocytosis, common in other domestic species, are not present in equine regenerative anemias.*

2. **Inflammatory leukogram.** Mild leukocytosis due to neutrophilia and monocytosis and concomitant hyperfibrinogenemia and hyperglobulinemia support the diagnosis of a chronic or low-grade inflammatory process.
3. **Thrombocytopenia.** Significant decreases in platelet numbers may indicate sampling error, increased usage (consumption), decreased bone marrow production, or increased destruction. With EDTA-dependent pseudothrombocytopenia (see Chapter 12), platelet counts performed from blood collected into heparinized or citrated tubes shoud be normal. Consumption (e.g., hemorrhage, DIC, acute infection, or endotoxemia) is the most common cause of "true" thrombocytopenia in horses. However, the history and clinical signs in this case suggest secondary immune-mediated destruction of platelets (e.g., as a result of equine infectious anemia [EIA], or lymphosarcoma) as the most likely cause of thrombocytopenia.
4. **Hypoalbuminemia.** Mild decreases in albumin concentration may be observed secondary to parasitism or chronic antigenic stimulation/ inflammation (often associated with hyperglobulinemia and a decreased A:G ratio).
5. **Hyperbilirubinemia.** In the absence of elevations in liver enzymes, a mild increase in bilirubin usually is the result of inappetence or anorexia. This is associated with reduced hepatic uptake of bilirubin as a result of decreased ligandin concentrations and not due to liver disease. Clinical jaundice is frequently observed. Acute hemolytic anemia also may produce hyperbilirubinemia.

Key Problems

1. The main laboratory findings are moderate anemia and thrombocytopenia. Additional information about the etiology of the anemia cannot be obtained from the above laboratory data, highlighting the difficulty associated with approach to the diagnosis of anemia in the horse and the necessity for further diagnostic testing.
2. The mild inflammatory process also should not be ignored and steps taken to locate its origin.

Differential Diagnosis (see also Chapter 12)

- Infectious hemolytic anemia (e.g., EIA, ehrlichiosis [unlikely])
- IMHA and immune-mediated thrombocytopenia
- Oxidant-induced hemolysis (e.g., onion, rape, kale, wilted red maple leaves)
- Chronic inflammatory disease (e.g., retropharyngeal abscess, chronic pleuritis, or abdominal abscess)
- Chronic neoplastic disease (e.g., gastric squamous cell carcinoma, urogenital neoplasia)

Suggestions for Further Diagnostic Tests

- Repeat hematologic tests with particular attention to
  - Confirmation of thrombocytopenia. Using citrated tube for blood collection and excellent technique should eliminate the possibility of pseudothrombocytopenia and sample error, respectively.
  - Detection of red-tinged plasma, indicating hemolysis.
  - Detection of Heinz bodies using new methylene blue stain, verifying oxidant injury to erythrocytes.
  - Erythrocyte agglutination after mixing with one part blood to four parts 0.9% saline, suggesting immune-mediated anemia.
  - Response of PCV. Increased values over time represent regeneration of erythrocytes after hemolytic or blood loss anemia.
- Perform Coggins test. The Coggins test is an agar gel immunodiffusion test that is used to detect the presence of antibody to EIA virus.
- Perform Coombs test. The direct antiglobulin or Coombs test detects the presence of immunoglobulin and/or complement on the surface of erythrocytes in IMHA.
- Rectal examination for palpation of infectious or neoplastic disorders of the abdomen.
- Urinalysis. A positive result for occult blood without microscopic hematuria or evidence of muscle damage indicates hemoglobinuria (intravascular hemolysis). Urine reagent strips detect only pigmenturia and do not differentiate between whole blood, hemoglobin, and myoglobin.

Suggestions for Additional Diagnostic Tests

- Bone marrow aspirate. Regenerative and nonregenerative anemias can be differentiated by evaluating the myeloid-to-erythroid ratio.
- Thoracic and abdominal ultrasound may aid evaluation of chronic inflammatory or neoplastic diseases that cause inadequate erythropoiesis.
- Abdominocentesis and thoracocentesis. Samples obtained should be submitted for cytologic and bacteriologic examination to confirm infectious or neoplastic causes of inadequate erythropoiesis.
- Gastric endoscopy. Gastric squamous cell carcinoma can be visualized and biopsy samples obtained using this diagnostic test.
- Iron analysis. Low serum iron with normal TIBC suggests anemia of chronic disease.

Additional Notes

- Regenerative and nonregenerative anemias in the horse are best determined by examination of bone marrow aspirates. Performing serial measurements of PCV, hemoglobin, and RCC also may be helpful to evaluate the erythropoietic response (PCV increases by 0.01 or 1% every 3 days). As well, serial determination of TP may aid in the differentiation of acute hemolytic anemias (normal [TP]) from acute blood loss anemias (decreasing [TP] 6-12 hours after blood loss) (see Chapter 12).
- True thrombocytopenia should be confirmed by repeating the count using a manual technique on smears prepared from thoroughly mixed blood samples (no clots) and after ensuring there are no clumps of platelets observed in the tail of the smear.

- Platelet counts of <20 X 10^9/L (i.e., 20,000/ML) can result in signs related to hemorrhage, including epistaxis, petechial and ecchymotic hemorrhages in mucous membranes, bleeding from injection sites, hematomas, and hyphema.
- Care should be taken with interpretation of a negative result for a Coombs test because false-negative results are possible, in particular if there has been prior corticosteroid therapy.
- Analysis for fecal occult blood frequently is unreliable because dietary myoglobin, hemoglobin, or plant peroxidases can give false-positive test results. False-negative results also are possible.
- Edema is abnormal accumulation of extracellular fluid resulting from increased hydrostatic pressure, decreased plasma oncotic pressure, increased capillary permeability, and decreased lymphatic drainage. Inflammation of blood vessels (vasculitis) is a common cause of ventral and limb edema in the horse and usually results from type III hypersensitivity reactions secondary to infections (e.g., *Streptococcus equi* var. *equi,* EIA virus, *Ehrlichia equi),* neoplasia, and occasionally drug administration (see Chapter 12).
- In general, hyperbilirubinemia due to inappetence does not exceed 85 µmol/L (5 mg/dL), although concentrations as high as 137-154 µmol/L (8-9 mg/dL) occur occasionally.
- A bone marrow myeloid:erythroid (M:E) ratio 0.5, in the presence of a decreased PCV and a normal leukogram, and a reticulocyte count >5% (or >50 reticulocytes/1000 erythrocytes), is consistent with erythrocyte regeneration.

Case Resolution

True thrombocytopenia was confirmed, and a Coggins test was positive for EIA.

CASE 5

Signalment: 12-year-old Standardbred mare.

History and Clinical Signs: Chronic weight loss, lethargy, inappetence, intermittent diarrhea, and lower limb edema observed for several months. Pyrexia of unknown origin also noted.

Laboratory Data

Component	SI Units (reference range)	Conventional Units (reference range)	Mild/Moderate/ Marked Change
Hematology			
PCV	0.28 L/L (0.30-0.48)	28% (30-48%)	
RCC	6.32 X 10^{12}/L (7.5-11.0)	6.32 X 10^6/µL (7.5-11.0)	
Hemoglobin	98 g/L (110-160)	9.8 g/dL (11-16)	
Red cell morphology: normal			
WCC	23.8 X 10^9/L (6.0-11.0)	23.8 X 10^3/µL (6.0-11.0)	
Neutrophils	17.8 X 10^9/L (2.5-7.0)	17.8 X 10^3/µL (2.5-7.0)	
Band neutrophils	0.19 X 10^9/L (0-0.24)	0.19 X 10^3/µL (0-0.24)	
Lymphocytes	2.85 X 10^9/L (1.6-5.4)	2.85 X 10^3/µL (1.6-5.4)	
Monocytes	2.96 X 10^9/L (0-0.7)	2.96 X 10^3/µL (0-0.7)	
Eosinophils	0 x 10^9/L (0-0.5)	0 X 10^3/µL (0-0.5)	

White cell morphology: evidence of mild "toxic" changes to neutrophils		
Platelets	800 X 10^9/L (100-300)	800 X 10^3/µL (100-300)
Biochemistry		
BUN	9.5 mmol/L (4.0-8.0)	26.6 mg/dL (11.2-22.4)
Creatinine	180 µmol/L (100-160)	2.04 mg/dL (1.13-1.81)
Plasma total protein	94 g/L (60-75)	9.4 g/dL (6.0-7.5)
Serum total protein	87 g/L (57-73)	8.7 g/dL (5.7-7.3)
Albumin	19 g/L (29-35)	1.9 g/dL (2.9-3.5)
Globulin	68 g/L (28-38)	6.8 g/dL (2.8-3.8)
Fibrinogen	7.0 g/L (1-4)	700 mg/dL (100-400)
CK	120 U/L (60-330)	120 U/L (60-330)
AST	195 U/L (160-412)	195 U/L (160-412)
ALP	167 U/L (138-251)	167 U/L (138-251)
GGT	15 U/L (10-40)	15 U/L (10-40)
Total bilirubin	18.3 µmol/L (10-50)	1.1 mg/dL (0.58-2.92)
Calcium	2.54 mmol/L (2.78-3.32)	10.2 mg/dL (11.1-13.3)
Phosphate	0.42 mmol/L (0.75-1.25)	1.3 mg/dL (2.3-3.9)
Sodium	138 mmol/L (132-142)	138 mEq/L (132-142)
Potassium	4.1 mmol/L (3.2-4.2)	4.1 mEq/L (3.2-4.2)
Chloride	104 mmol/L (94-104)	104 mEq/L (94-104)

Summary of Problems and Interpretation

1. **Severe inflammatory leukogram.** Marked leukocytosis due to marked neutrophilia and monocytosis indicates inflammation or tissue destruction. Absence of band (immature) neutrophils suggests that the condition is chronic and ongoing. Chronic inflammatory disease is reinforced by the presence of marked elevation in fibrinogen and globulin concentrations. Hyperfibrinogenemia generally occurs with infectious, suppurative, traumatic, and neoplastic diseases and remains high as long as inflammation is still active.
2. **Anemia.** Inadequate erythropoiesis due to chronic inflammatory disease likely explains mild decreases in PCV. Immune-mediated hemolysis as a result of bacterial or neoplastic antigens coating erythrocytes also may contribute to anemia.
3. **Thrombocytosis.** Numbers of platelets often increase in response to chronic infectious, inflammatory, and neoplastic diseases. Chronic blood loss also may result in thrombocytosis.
4. **Hyperproteinemia.** Marked hyperproteinemia may be due to a combination of dehydration, hyperglobulinemia, and hyperfibrinogenemia. High concentrations of globulins usually result from an increase in immunoglobulins in response to chronic severe antigenic stimulation (e.g., infection, abscess, neoplasia).
5. **Hypoalbuminemia/hypocalcemia.** Severe decreases in albumin concentrations indicate either metabolic utilization for production of immunoglobulins, gastrointestinal or renal loss, loss into a body cavity, or lack of production by the liver. Pseudohypocalcemia is the result of losses in albumin-bound calcium.
6. **Azotemia.** Mild azotemia in this case probably is prerenal, resulting from dehydration.

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Key Problems

1. The important findings in this case are severe leukocytosis and hyperglobulinemia in response to chronic marked inflammation. This should prompt practitioners to investigate the location of inflammation.

Differential Diagnosis (see also Chapter 7)

- Severe chronic peritonitis or abdominal abscess involving liver, kidney, pancreas, lymph nodes, or gut
- Severe chronic thoracic abscess
- Pneumonia/pleuritis
- Vasculitis
- Bacterial endocarditis
- Neoplasia
- Pericarditis

Suggestions for Further Diagnostic Tests

- Thorough rectal examination and repeat physical examination with emphasis on thoracic and abdominal cavities
- Abdominal and thoracic ultrasound
- Abdominocentesis and thoracocentesis with complete laboratory analysis of samples
- Endoscopy of all structures of upper airway, including guttural pouches
- Serum protein electrophoresis. May aid to determine specific globulin elevations and rule out monoclonal gammopathy as a cause of hyperglobulinemia
- Blood culture

Additional Notes

- Fibrinogen concentration normally increases in response to inflammation and then declines as the condition improves. However, the degree of hyperfibrinogenemia does not always correlate with the severity of disease or inflammation.
- Albumin concentration must generally fall below 15 g/L (1.5 mg/dL) in horses before edema of the distal extremities, ventral body wall, larynx, and face occurs.

Case Resolution

Thoracic ultrasound findings were consistent with the presence of endocarditis. Blood cultures revealed the presence of α-hemolytic streptococci, and based on the severity of the clinical findings and ultrasound examination, euthanasia was recommended.

CASE 6

Signalment: 3-year-old Thoroughbred gelding in race training.
History and Clinical Signs: A "blood screen" requested for a racehorse that has performed poorly. The blood sample was taken soon after exercise, and there was a long delay in sample processing.

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Laboratory Data:

Component	SI Units (reference range)	Conventional Units (reference range)	Mild/Moderate/ Marked Change
Hematology			
PCV	0.50 L/L (0.30-0.48)	50% (30-48%)	
RCC	15.3 X 10^{12}/L (7.5-11.0)	15.3 X 10^{6}/µL (7.5-11.0)	
Hemoglobin	175 g/L (110-160)	17.5 g/dL (11-16)	
Red cell morphology: normal			
WCC	13.5 X 10^{9}/L (6.0-11.0)	13.5 X 10^{3}/µL (6.0-11.0)	
Neutrophils	5.9 x 10^{9}/L (2.5-7.0)	5.9 x 10^{3}/µL (2.5-7.0)	
Lymphocytes	7.3 X 10^{9}/L (1.6-5.4)	7.3 X 10^{3}/ML (1.6-5.4)	
Monocytes	0.3 x 10^{9}/L (0-0.7)	0.3 X 10^{3}/µL (0-0.7)	
Eosinophils	0 X 10^{9}/L (0-0.5)	0 x 10^{3}/µL (0-0.5)	
White cell morphology: poor leukocyte morphology			
Platelets	130 X 10^{9}/L (100-300)	130 X 10^{3}/µL (100-300)	
Biochemistry			
BUN	4.5 mmol/L (4.0-8.0)	12.6 mg/dL (11.2-22.4)	
Creatinine	153 µmol/L (100-160)	1.73 mg/dL (1.13-1.81)	
Plasma total protein	71 g/L (60-75)	7.1 g/dL (6.0-7.5)	
Serum total protein	69 g/L (57-73)	6.9 g/dL (5.7-7.3)	
Albumin	33 g/L (29-35)	3.3 g/dL (2.9-3.5)	
Globulin	36 g/L (28-38)	3.6 g/dL (2.8-3.8)	
Fibrinogen	2 g/L (1-4)	200 mg/dL (100-400)	
CK	75 U/L (60-330)	75 U/L (60-330)	
AST	174 U/L (160-412)	174 U/L (160-412)	
ALP	163 U/L (138-251)	163 U/L (138-251)	
GGT	51 U/L (10-40)	51 U/L (10-40)	
Total bilirubin	43 µmol/L (10-50)	2.5 mg/dL (0.58-2.92)	
Glucose	2.9 mmol/L (4.1-6.4)	52.3 mg/dL (75-115)	
Calcium	2.80 mmol/L (2.78-3.32)	11.2 mg/dL (11.1-13.3)	
Phosphate	1.2 mmol/L (0.75-1.25)	3.7 mg/dL (2.3-3.9)	
Sodium	133 mmol/L (132-142)	133 mEq/L (132-142)	
Potassium	4.7 mmol/L (3.2-4.2)	4.7 mEq/L (3.2-4.2)	
Chloride	98 mmol/L (94-104)	98 mEq/L (94-104)	

Summary of Problems and Interpretation

1. **Polycythemia.** Relative polycythemia is very common in horses as a result of dehydration or catecholamine-induced splenic contraction. Increases of 41, 39, and 50% for RCC, hemoglobin, and PCV, respectively, have been reported in horses after a short intense exercise period, largely due to catecholamine-induced splenic contraction.
2. **Leukocytosis.** In young healthy horses, physiologic leukocytosis, characterized by neutrophilia and/or lymphocytosis, is commonly found in routine hematologic analysis. The response is transient (lasting approximately 20-30 minutes) and results from increased blood pressure, increased lymphatic drainage, and splenic contraction associated with excitement or exercise. Excitement can be induced by stimuli as minimal

as venipuncture and probably varies with the technique of the collector and with the temperament of the individual horse.

3. **Hyperkalemia.** Mild increase in serum potassium concentration frequently is noted in samples that have evidence of hemolysis or in samples that have been stored for lengthy periods without separation of serum or plasma.
4. **Hypoglycemia.** Delayed separation of serum or plasma from blood cells can allow time for leukocytes, erythrocytes, and platelets to consume glucose, thus resulting in a falsely low glucose concentration in the sample.
5. **Elevated GGT.** Increased concentrations of serum GGT frequently are found in racehorses (particularly in Thoroughbreds that are performing below expectation). In all of these cases, other indices of liver damage are normal and no histologic evidence of hepatic disease has been found. Although the stress of training appears to be a factor involved, the reference range for GGT in horses in race training may be higher than that for normal sedentary horses.

Key Problems

1. The key issue is that specific changes in laboratory data frequently can be a result of physiologic, collection, and laboratory variables and environmental influences and are not necessarily due to pathologic conditions.

Differential Diagnosis

- Laboratory error, sample overheating
- Relative polycythemia
- Primary absolute polycythemia (polycythemia vera)
- Secondary absolute polycythemia (increased erythropoietin production).

Suggestions for Further Diagnostic Tests

- Repeat blood collection with appropriate technique, preferably when the horse is relaxed. If necessary, blood samples should be transported chilled and serum or plasma separated from blood cells within 1 hour of collection.

Additional Notes

- The increase in erythrocyte variables resulting from splenic contraction usually does not correct for approximately 60 minutes, depending on the level of stress.
- It is essential that leukocytosis is not overinterpreted. Repeat blood samples should confirm prolonged elevations in specific leukocytes that may indicate a pathologic or inflammatory process. In addition, measurement of fibrinogen concentrations can help differentiate between physiologic and pathologic leukocytosis.
- Chronic hypoxia from severe pulmonary or cardiac disease may induce physiologic increases in serum erythropoietin concentration. Inappropriate increase in erythropoietin concentration may occur as a result of renal, hepatic, or endocrine neoplasia or from administration of recombinant erythropoietin.

CASE 7 **Signalment:** 5-year-old quarter horse gelding.
History and Clinical Signs: Poor recovery after an endurance ride with development of signs of depression, anorexia, sweating, muscle tremor, stiffness, and pain when forced to walk.

Laboratory Data

Component	SI Units (reference range)	Conventional Units (reference range)	Mild/Moderate/ Marked Change
Hematology			
PCV	0.55 L/L (0.30-0.48)	55% (30-48%)	
RCC	18.5 x 10^{12}/L (7.5-11.0)	18.5 X 10^6/µL (7.5-11.0)	
Hemoglobin	215 g/L (110-160)	21.5 g/dL (11-16)	
Red cell morphology: normal			
WCC	14.5 X 10^9/L (6.0-11.0)	14.5 X 10^3/µL (6.0-11.0)	
Neutrophils	9.9 x 10^9/L (2.5-7.0)	9.9 X 10^3/µL (2.5-7.0)	
Band neutrophils	0.3 X 10^9/L (0-0.24)	0.3 X 10^3/ML (0-0.24)	
Lymphocytes	2.7 x 10^9/L (1.6-5.4)	2.7 X 10^3/µL (1.6-5.4)	
Monocytes	1.4 X 10^9/L (0-0.7)	1.4 X 10^3/µL (0-0.7)	
Eosinophils	0.2 X 10^9/L (0-0.5)	0.2 X 10^3/µL (0-0.5)	
White cell morphology: normal			
Platelets	150 X 10^9/L (100-300)	150 X 10^3/µL (100-300)	
Biochemistry			
BUN	21.7 mmol/L (4.0-8.0)	60.8 mg/dL (11.2-22.4)	
Creatinine	250 µmol/L (100-160)	2.83 mg/dL (1.13-1.81)	
Plasma total protein	79 g/L (60-75)	7.9 g/dL (6.0-7.5)	
Serum total protein	76 g/L (57-73)	7.6 g/dL (5.7-7.3)	
Albumin	39 g/L (29-35)	3.9 g/dL (2.9 -3.5)	
Globulin	37 g/L (28-38)	3.7 g/dL (2.8-3.8)	
Fibrinogen	3 g/L (1-4)	300 mg/dL (100-400)	
CK	13,550 U/L (60-330)	13,550 U/L (60-330)	
AST	1685 U/L (160-412)	1685 U/L (160-412)	
ALP	249 U/L (138-251)	249 U/L (138-251)	
GGT	37 U/L (10-40)	37 U/L (10-40)	
Total bilirubin	45 µmol/L (10-50)	2.63 mg/dL (0.58-2.92)	
Calcium	2.55 mmol/L (2.78-3.32)	10.2 mg/dL (11.1-13.3)	
Phosphate	1.6 mmol/L (0.75-1.25)	5.0 mg/dL (2.3-3.9)	
Magnesium	0.45 mmol/L (0.58-0.95)	1.1 mg/dL (1.4-2.3)	
Sodium	123 mmol/L (132-142)	123 mEq/L (132-142)	
Potassium	2.5 mmol/L (3.2-4.2)	2.5 mEq/L (3.2-4.2)	
Chloride	88 mmol/L (94-104)	88 mEq/L (94-104)	
Blood gases and acid-base analysis (venous)			
Pco_2	55 mm Hg (38-48)	55 mm Hg (38-48)	
$HC0_3^-$	35 mmol/L (22-29)	35 mmol/L (22-29)	
pH	7.5 (7.34-7.43)	7.5 (7.34-7.43)	

Summary of Problems and Interpretation

1. **Inflammatory leukogram.** Mild leukocytosis with concomitant left shift and monocytosis suggests tissue (in this case, muscle) inflammation.

2. **Dehydration.** Mild elevations in PCV, TP, and albumin denote, in this case, dehydration due to inadequate water intake and increased losses through sweat.
3. **Azotemia.** Apart from prerenal mechanisms (e.g., dehydration), a significant component of the azotemia may be due to renal damage and reduced GFR secondary to myoglobinuria.
4. **Myopathy.** Marked increases in both CK and AST activity indicate severe muscle damage. However, AST is a nonspecific indicator of tissue necrosis and may be elevated in hepatocellular, cardiac, and kidney disorders. Rarely, CK may be released as a result of cardiac and sometimes intestinal disease. In general, concentrations of CK and then AST increase in response to muscle damage, with CK peaking at 4-6 hours and returning to normal within 12 to 24 hours after muscle damage abates. Concentrations of AST, which peak at 24-48 hours, can remain elevated for up to 10 days.
5. **Hypochloremic metabolic alkalosis.** Loss of large volumes of fluid in sweat high in sodium, potassium, chloride, calcium, and magnesium but without proportionate loss of bicarbonate results in metabolic alkalemia. Renal compensation favors loss of hydrogen ions (low pH) and potassium (hypokalemia) in exchange for sodium, whereas respiratory compensation (hypoventilation) results in increased Pco_2.
6. **Hypocalcemia, Hypomagnesemia, and hyperphosphatemia.** Primary hypocalcemia can be the result of dietary imbalance, lactation, cantharidiasis (blister beetle poisoning), exercise, alkalosis, and, rarely, renal disease. Hyperphosphatemia most commonly is associated with hemolysis secondary to poor sample collection or handling, although mild increases are observed after endurance exercise or due to diets with low calcium/phosphorus ratios. Although little is known about disorders that affect serum magnesium concentrations in the horse, mild decreases have been reported after endurance exercise, cantharidiasis, and primary hypoparathyroidism.

Key Problems

1. The key laboratory abnormalities in this case are marked elevations in muscle enzymes, indicative of severe myopathy. Electrolyte and acid-base derangements are a result of losses in sweat.
2. Hypocalcemia is also a significant abnormality, the clinical manifestations of which ultimately can be life-threatening.
3. Repeat measurements for CK, AST, PCV, TP, and electrolyte and acid-base data are essential to monitor recovery or progression of muscle damage and to monitor response to therapy.

Differential Diagnosis (see also Chapter 4)

- Exhausted horse syndrome
- Exertional rhabdomyolysis
- Polysaccharide storage myopathy
- Trauma

Suggestions for Further Diagnostic Tests

- Urinalysis. In particular, examine urine for myoglobin. To differentiate between hemoglobinuria and myoglobinuria, saturated ammonium sulfate usually precipitates and removes color caused by hemoglobin.

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Suggestions for Additional Diagnostic Tests

- Fractional excretion of urinary electrolytes to evaluate renal status.

Additional Notes

- Persistent increases in CK and AST concentrations over time indicate active continuing muscle damage, and the prognosis tends to be more guarded.
- LDH also can be measured in cases in which muscle damage is suspected.
- Because erythrocytes contain AST and LDH, hemolysis or delayed removal of serum from a clot can give falsely increased serum values of these enzymes. As well, hemolysis and lipemia may interfere with AST and CK measurements using certain assays.
- Other important clinical findings in horses with this syndrome may include synchronous diaphragmatic flutter, ileus, and cardiac arrhythmias. These clinical manifestations mostly are the result of severe decreases in ionized calcium. Release of increased concentrations of myoglobin also can result in oliguric renal failure.
- Unless ionized calcium is determined, measurements for total calcium are influenced by albumin concentration and the acid-base status (ionized calcium increases with acidosis). Therefore, total calcium deficit cannot be accurately calculated, and formulas for adjusting the measured serum calcium have not been described for horses.

Case Resolution

Urinalysis confirmed myoglobinuria as a result of severe myopathy. Appropriate intravenous fluid and electrolyte therapy resulted in improvement in clinical and laboratory measurements.

CASE 8

Signalment: 18-year-old pony mare, retired in paddock.
History and Clinical Signs: Signs of anorexia and profound depression observed for several days. The pony also has had intermittent phenylbutazone administration for recurrent laminitis.

Laboratory Data

Component	SI Units (reference range)	Conventional Units (reference range)	Mild/Moderate/ Marked Change
Hematology			
PCV	0.54 LA (0.30-0.48)	54% (30-48%)	
RCC	12.5 X 10^{12}/µL (7.5-11.0)	12.5 X 10^{6}/ML (7.5-11.0)	
Hemoglobin	195 g/L (110-160)	19.5 g/dL (11-16)	
Red cell morphology: normal			
WCC	10.9 X 10^{9}/L (6.0-11.0)	10.9 X 10^{3}/ML (6.0-11.0)	
Neutrophils	9.2 X 10^{9}/L (2.5-7.0)	9.2 x 10^{3}/µL (2.5-7.0)	
Lymphocytes	1.2 X 10^{9}/L(1.6-5.4)	1.2 X 10^{3}/µL (1.6-5.4)	
Monocytes	0.5 X 10^{9}/L (0-0.7)	0.5 x 10^{3}/µL (0-0.7)	
Eosinophils	0 X 10^{9}/L (0-0.5)	0 X 10^{3}/ML (0-0.5)	

White cell morphology: normal		
Platelets	175 X 10^9/L (100-300)	175 X 10^3/µL (100-300)
Biochemistry		
BUN	15.3 mmol/L (4.0-8.0)	42.9 mg/dL (11.2-22.4)
Creatinine	350 µmol/L (100-160)	3.96 mg/dL (1.13-1.81)
Plasma total protein	53 g/L (60-75)	5.3 g/dL (6.0-7.5)
Serum total protein	50 g/L (57-73)	5.0 g/dL (5.7-7.3)
Albumin	22 g/L (29-35)	2.2 g/dL (2.9-3.5)
Globulin	28 g/L (28-38)	2.8 g/dL (2.8-3.8)
Fibrinogen	3 g/L (1-4)	300 mg/dL (100-400)
CK	256 U/L (60-330)	256 U/L (60-330)
AST	475 U/L (160-412)	475 U/L (160-412)
ALP	305 U/L (138-251)	305 U/L (138-251)
GGT	63 U/L (10-40)	63 U/L (10-40)
Total bilirubin	67.2 µmol/L (10-50)	3.93 mg/dL (0.58-2.92)
Total bile acids	35 µmol/L (5-28)	35 µmol/L (5-28)
Glucose	6.0 mmol/L (4.1-6.4)	108 mg/dL (75-115)
Calcium	2.75 mmol/L (2.78-3.32)	11.0 mg/dL (11.1-13.3)
Phosphate	1.12 mmol/L (0.75-1.25)	3.5 mg/dL (2.3-3.9)
Sodium	135 mmol/L (132-142)	135 mEq/L (132-142)
Potassium	2.9 mmol/L (3.2-4.2)	2.9 mEq/L (3.2-4.2)
Chloride	95 mmol/L (94-104)	95 mEq/L (94-104)
Triglycerides	12 mmol/L (0.06-0.61)	1062 mg/dL (5.3-54)

Summary of Problems and Interpretation

1. **Dehydration.** Moderate elevations in PCV, RCC, and hemoglobin suggest dehydration, probably from lack of water intake. It is important to note that the presence of dehydration means that protein concentrations may be lower than is indicated by these data.
2. **Stress/corticosteroid-induced neutrophilia and lymphopenia.** Endogenous corticosteroids may be released from the adrenal glands as a result of stress-related mechanisms or secondary to excessive adrenocorticotropic hormone (ACTH) production.
3. **Hyperlipemia.** Marked increase in triglycerides (above 5.6 mmol/L, or 500 mg/dL) indicates hyperlipemia, which frequently is observed as opalescent plasma after blood collection. Obese ponies appear to be more prone to hyperlipemia, which almost always is precipitated by a primary disorder including any condition that causes prolonged anorexia (e.g., after surgery), pregnancy, malnutrition, and pituitary disorders.
4. **Hepatopathy.** Moderate elevations in all liver-related enzymes reflect hepatocellular damage and cholestasis, probably the result of fatty infiltration of the liver.
5. **Azotemia.** Dehydration is the likely cause of moderate prerenal azotemia, although lipid accumulation in renal tissue can lead to derangement in renal function.
6. **Hypoproteinemia.** Moderate decreases in albumin and plasma total protein concentrations suggest gastrointestinal or renal protein loss or decreased production by the liver. Commonly encountered causes include parasitism, colitis, and infiltrative or neoplastic diseases of the intestinal tract. In this case, prolonged or excessively high doses of phenylbutazone may have led to a protein-losing enteropathy.
7. **Hypokalemia.** Although uncommon, mild decreases in potassium may

occur in anorexic horses. Other common causes include diarrhea, excessive sweating, and administration of furosemide.

Key Problems

1. The main finding of note is severe hyperlipemia. This condition must be aggressively addressed first, regardless of the cause. However, concentrations of triglycerides of this magnitude generally indicate a guarded prognosis.

Differential Diagnosis (see also Chapters 4, 7, and 15)

- Phenylbutazone toxicity
- Infiltrative disease such as granulomatous enteritis, eosinophilic gastroenteritis, and lymphosarcoma
- Chronic laminitis
- Equine Cushing-like disease
- Other causes of hepatic disease (e.g., chronic active hepatitis, abscessation, or chronic pyrrolizidine alkaloid toxicosis)

Suggestions for Further Diagnostic Tests

- Further diagnostic tests may be superseded in this case by the urgent need for therapy for hyperlipemia. Monitoring blood concentrations of triglycerides, creatinine, BUN, and TP is essential to determine response to therapy and prognosis.

Suggestions for Additional Diagnostic Tests

- Urinalysis to further assess renal function
- Radiography of pedal bone in affected hoof or hooves
- Intestinal biopsy (rectal, at laparotomy, or laparoscopically)
- Urinary Cortisol/creatinine ratio
- Dexamethasone suppression test (clinicians should apply caution with this test in horses already suffering laminitis and may elect to use the thyroid-releasing hormone response test instead)
- Liver biopsy

Additional Notes

- This case illustrates several problems that frequently occur in older ponies, sometimes concurrently (i.e., laminitis, phenylbutazone toxicity, and hyperlipemia).
- Serum triglyceride concentrations begin to increase within 3 days of fasting in horses. A falsely low sodium and chloride concentration can be produced by marked hyperlipemia if ion-specific electrodes for ion determinations are not used.
- Triglyceride concentrations above 22 mmol/L, or 2000 mg/dL, generally suggest a grave prognosis.

Case Resolution

Aggressive therapy for hyperlipemia and laminitis failed to ameliorate the clinical condition of the pony, and euthanasia was elected. Postmortem

examination confirmed extensive fatty infiltration of the liver and kidney, evidence of gastrointestinal ulceration, and moderate enlargement of the pituitary gland.

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CHAPTER 17

Clinical Bacteriology

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Clinical bacteriology is a useful adjunct to other services offered within an equine practice. If samples for bacteriology are collected and handled appropriately and results of culture and sensitivity interpreted correctly, the information obtained can greatly assist appropriate case management. However, if samples are collected or handled inappropriately or the results of culture and sensitivity testing are misinterpreted, the outcome may have detrimental effects.

It is the intent of this chapter to outline in a simple and logical fashion the clinical bacteriologic procedures that can be performed in routine equine practice, not those performed in specialist or referral laboratories. The decision to set up a microbiology laboratory within a practice is necessarily an economic one. However, even if culture and sensitivity testing is not performed within your practice, the correct principles of sample collection, sample processing, and interpretation of results must be adhered to if bacterial infections in horses are to be managed appropriately.

THE PRACTICE LABORATORY

There are five constraints that limit the scope of what can be realistically achieved in the practice laboratory. These limitations are time, knowledge, financial resources, likely remuneration for time and money expended, and turnover.

Practitioners will only have limited time for clinical microbiology. It is not fiscally sensible for a busy practitioner to spend time learning techniques that will be used occasionally or that are so time consuming that reimbursement for client fees never can be expected. Similarly, capital expenditure on equipment should be carefully evaluated. Disposables with limited shelf life, such as identification kits, must be used before the expiration date, so it is important that there is sufficient throughput to warrant their purchase.

KEY POINT

Good advice for practice microbiologists is to keep it simple and cost effective.

With this in mind the following procedures should be possible to perform in a practice laboratory:

- Microscopic examination of stained smears of clinical specimens
- Isolation and identification of *nonfastidious* bacteria from clinical specimens
- Antimicrobial sensitivity testing of nonfastidious bacteria

Many veterinarians are reluctant to perform tests to isolate and identify bacteria. Some believe they are unqualified to do so, and others perceive the process as being too complex. In fact, only about 10 to 12 genera of bacteria are regularly isolated from routine equine specimens. These bacteria can be easily identified using a few simple, inexpensive tests. Because these isolates are found so frequently and are relatively easy to identify, it would pay most veterinary clinics to perform their own routine bacterial cultures.

However, it is also important for the practitioner to recognize cases that may involve organisms that are fastidious or difficult to grow and send these samples to an approriate laboratory.

KEY POINT

Veterinarians should be able to discriminate between samples that may be routinely

cultured in-house and those that need to be sent to a referral laboratory. This decision needs to be made soon after sample collection so that the sample may be processed quickly and appropriately to allow survival of these organisms.

SELECTION OF CASES FOR CULTIVATION OF BACTERIA

It is recognized that in practice, not all horses with suspected bacterial infections would require identification of the causative agent. A number of factors influence the decision to perform culture and sensitivity tests:

- Economic factors related to the case
- Previous antibiotic therapy
- Severity of disease
- Stage of disease (i.e., is the disease process short/long term)
- Results of smears made from sample (i.e., is there evidence of inflammation, presence of bacteria)
- Suspected bacterial agents and likely antimicrobial sensitivity patterns

KEY POINT

If you plan to obtain a sample for cultivation, the appropriate sample must be collected before *placing the animal or antimicrobial agents because they can severely affect the ability to isolate bacteria from samples collected.*

SAMPLE COLLECTION

Localize the Site of Infection

- Once the decision to obtain a sample for bacteriologic cultivation has been made, it is essential to localize the infection as accurately as possible. It is not adequate to merely collect a sample from somewhere near the site of infection (e.g., draining tract, pharyngeal or nasal swabs for lower respiratory tract disease) and submit this sample for culture and sensitivity.

KEY POINT

Specimen collection from the actual site of infection using appropriate techniques is the most important process in practice microbiology. *It is not difficult to culture bacteria; it can be difficult to culture the bacteria that are causing disease if you do not use the correct technique and sample from the correct site.*

- It cannot be overemphasised that if you collect inappropriate specimens, then you will get inappropriate results.
- Specimen collection and subsequent processing of the sample will vary depending on the site to be sampled and the type of sample to be collected.

Preparation of Sample Sites

NORMALLY STERILE SITES

- Sites that are normally sterile include blood, joints, abdominal and thoracic cavities, central nervous system, lower respiratory tract, uterus, bladder, and tissues beneath the skin. Because these sites are normally sterile, sampling technique *must be aseptic* so that organisms are not introduced or surface bacteria do not contaminate the sample obtained.

KEY POINT

It is important to maintain asepsis during collection and processing of samples from normally sterile sites, because growth of any bacteria from these sites usually is considered significant.

- An appropriate technique to disinfect the skin prior to collection of blood, joint fluid, abdominal fluid, thoracic fluid, cerebrospinal fluid (CSF), and subcutaneous tissue is as follows:
  - o Clip hair.
  - o Wash skin thoroughly with soap and water or use a povidone-iodine scrub as you would for a surgical preparation.
  - o Swab with 70% alcohol and then swab with tincture of iodine (2% iodine in 70% alcohol) or povidone-iodine and allow to dry before sampling.

KEY POINT

It is preferable to wear a surgical mask when sampling CSF and joint fluid and to wear sterile gloves if the area is to be palpated to facilitate collection. This is to prevent introduction of the collector's pharyngeal bacteria (such as Staphylococcus *spp.) and skin flora into the sterile site during sample collection.*

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SITES WITH NORMAL FLORA

KEY POINT

Normal flora will be present in these samples, and culture results have to be interpreted in the light of knowledge of the bacteria normally found at these sites. The presence of inflammation in smears stained with Diff Quik is an important indicator of disease at these sites and must be present for significance to be attached to any isolates obtained.

- When collecting samples from these sites, try to avoid or eliminate as much of the normal flora as possible. For example, if sampling skin lesions, select an intact pustule rather than ulcers or erosions. The surface of the pustule should be swabbed with alcohol, and then the pustule can be incised with a sterile scalpel blade and underlying tissues sampled using moistened swabs. Alternatively, if the lesion is large enough, a fine-needle aspiration of infected tissue may be obtained. For sites where avoidance of normal flora is impossible (e.g., lesions within mucous membranes of the mouth or vagina), use a dry swab to remove as much of the secretions and surface flora as possible before sampling with a moistened swab.

Collection Techniques

A number of different techniques are available to sample for bacterial cultivation. The choice of technique will depend on the site to be sampled and the type of bacteria suspected.

SWABS

- Swabs are a useful and simple method for collecting samples for bacteriologic cultivation. However, there are a number of problems associated with the use of swabs, and one should be aware of these before their use.

KEY POINT

It can be difficult to interpret the significance of bacteria isolated from a sample collected and transported on a dry swab. Dry swabs may only yield the hardier organism(s), whereas a more delicate organism(s) may have been the etiologic agent.

- If dry swabs are used, they should be moistened before collection with either sterile saline or an appropriate transport media (e.g., Stuart's medium). A swab system that incorporates a transport media, for example, Marion Scientific Culturette specimen transport system (Marion Scientific, Kansas City, MO), is a simple alternative.
- If the contagious equine metritis organism *(Taylorella equigenitalis)* is suspected, Amies medium should be used to moisten and transport swabs after collection.

KEY POINT

Swabs are definitely inferior *for collection of specimens where there are suspected anaerobic bacteria. They should only be used when the sample cannot be obtained by another means.*

- If swabs have to be used for culture of anaerobic bacteria, the swab should be placed immediately into a commercial transport system that is suitable for these organisms (e.g., Port-A-Cul Tube, BBL Division of Becton-Dickinson and Co., Cockeysville, MD; BD Vacutainer Anaerobic Specimen Collector, BD Division of Becton-Dickinson and Co., Rutherford, NJ; or the Scott Two-Tube System, Scott Laboratories, Inc., Fiskeville, RI).
- Regardless of the type of swab used, all swabs should be processed as soon as possible after collection.

NEEDLE AND SYRINGE

- Another simple collection technique is a needle and syringe. Samples commonly collected with this technique include pus, tracheal washes, samples from subcutaneous or soft tissue infections, and fluid samples (e.g., joint, abdominal, thoracic and CSF). In addition, this technique may be used for collection of samples suspected to contain anaerobic bacteria.
- After collection of the sample, expel as much air as possible from the syringe, remove the needle, and cap it effectively to prevent leakage. These samples may then be transported to the laboratory. If strictly anaerobic bacteria are suspected, draw the sample into syringe through a wide-bore needle, expel any air, and insert the needle into a rubber stopper to exclude air or replace the needle with a plastic cap.

KEY POINT

If a long delay is anticipated between collection and processing, it is preferable to place the sample in a suitable transport medium rather than leaving it in the syringe. This is absolutely essential for fluid samples collected (e.g., blood, joint, abdominal, thoracic, and CSF).

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STERILE COLLECTION VIALS

- A variety of sterile, empty, screw-capped collection vials should be kept within your practice for the collection of fluids or tissues. These samples would include urine, milk, pus, and solid tissues. In addition, fastidious bacteria and anaerobes may survive for several days in pieces of tissues kept at cool temperatures.
- Select a container that will provide minimal airspace once the sample is included, and tape the lid to prevent leakage. Again, if delay between collection and processing is anticipated, add a small amount of sterile saline or transport medium to prevent the tissue from drying out.

Samples with Special Consideration for Collection

BLOOD

Blood cultures are an essential part of a diagnostic workup in suspected cases of septicemia. However, there are a number of special considerations that must be taken into account when culturing blood.

Time of Sampling

To obtain the most reliable results, a blood culture should be performed either before the commencement of antimicrobial therapy or when antibiotic therapy has been withdrawn for 24 hours. Additional blood cultures (two or three) may be collected when trough antimicrobial concentrations are anticipated (i.e., just before the next required treatment), and this will increase the likelihood of isolating the causative agent.

Fever clears the blood of organisms, so if blood is cultured during a febrile period, the yield will be low. Therefore, if the patient has a history of fever spikes or some predictable pattern of fever peaks, it is best to take the sample approximately 1 hour before the next fever peak.

KEY POINT

Often, blood is free from significant numbers of organisms at the time of peak fever. However, it may be difficult in practice to select the sampling time 1 hour before a fever spike. In these cases a number of samples should be taken to obtain a representative sample during a period of peak bacteremia.

In septicemic foals, multiple cultures may not be required, because numbers of circulating bacteria are frequently quite high. However, in some septicemic states (e.g., *Rhodococcus equi* infections), where it may be difficult to sample alternate sites, the numbers of organisms in the blood may be disappointingly low, and multiple samples may be required.

KEY POINT

It is important to remember that small bacterial numbers can occur even when bacteremia is significant. This fact will play a role when interpreting blood smears and when judging time for incubation before a negative result is reported.

Collection of Blood for Cultures

It is preferable to sample from a previously unused vein to ensure that an aseptic sample is obtained. The area over the selected vein should be prepared as described for sampling of sterile sites using a surgical preparation. Ten to 20 mL of blood should be obtained from the patient using a *new* needle and syringe and only minimum negative pressure to collect the sample.

KEY POINT

Vacutainer tubes (Becton-Dickinson and Co., Rutherford, NJ) should not be used to collect blood for culture because the excessive vacuum may result in contamination of the sample.

BODY FLUIDS (JOINT, ABDOMINAL, THORACIC, AND CEREBROSPINAL)

- As with blood cultures, samples from these sites should not contain bacteria. Thus, maintenance of absolute sterility when collecting samples from these sites is of utmost importance. In addition, numbers of bacteria in these samples may be low. Therefore, obtaining up to 10 mL of sample is preferable.

TRACHEAL ASPIRATES

- Tracheal aspirates are the preferred sample for cultivation if bacterial pneumonia or pleuropneumonia is suspected. Nasal or pharangeal swabs or samples collected by bronchoalveolar lavage (BAL) are *not* an appropriate sample in these cases.

KEY POINT

Samples collected via BAL tubes are not appropriate for cultivation of samples from lower airways due to oropharyngeal contamination and because only caudodorsal

(and not cranioventral) lung lobes are sampled.

- Tracheal aspirates may be collected using either the percutaneous transtracheal aspiration technique or via an endoscope using a plugged, guarded catheter (Darien Microbiological Aspiration Catheter; Mill Rose Laboratories, Mentor, OH). If pleuropneumonia is suspected, samples from both the left and right thoracic cavities also should be collected for cultivation.
- The sample is best submitted to the laboratory in the syringe into which it has been aspirated at the time of collection.

URINE

- Cultivation of urine in equine practice usually is not required. However, it should be remembered that urethritis, cystitis, and pyelonephritis have been reported in horses. Urine should be collected to avoid contamination with normal urethral/vaginal/preputial flora. Cystocentesis (the preferred technique for collection of urine for cultivation) is not practical in horses, but midstream voided or samples collected by sterile catheter are appropriate if the vulva/prepuce/penis has been cleaned and dried thoroughly before voiding. If urine is to be collected for cultivation, the sample should be collected in a sterile screw-capped container and kept upright to make sure that it does not leak during transportation.

SAMPLES IN WHICH STRICTLY ANAEROBIC BACTERIA ARE SUSPECTED TO BE PRESENT

- Routine isolation and identification of anaerobes may be unwarranted. Such work is time consuming and expensive, and anaerobes are part of the normal flora in many sites. It is important, therefore, to decide when it is necessary to attempt isolation of anaerobic bacteria.

KEY POINT

Anaerobic bacteria may be the sole etiologic agents in an infectious process or part of mixed infections (e.g., abscesses, osteomyelitis, postoperative infection of the gastrointestinal and female genital tracts, pleuritis, pneumonia, and peritonitis).

- There are a number of criteria that may alert the practitioner that anaerobic bacteria may be playing a role in an infectious process:
  - o Infection sites located close to areas where anaerobes are a component of the normal flora (e.g., submandibular abscess, osteomyelitis after a compound fracture, pleuritis, injection abscess)
  - o Presence of gas at the site of infection
  - o Black discoloration at the site of infection
  - ° Foul odor
  - o Morphology of bacteria seen in the initial Gram stain (e.g., gram-negative rods and filaments *[Bacteroides* spp., *Fusobacterium* spp.]; branching gram-positive filaments [possibly *Actinomyces* spp.]; or large gram-positive, gram-variable, or gram-negative rods that may or may not be sporing *[Clostridium* spp.])
  - o Infections secondary to an animal bite
  - o Prior therapy with aminoglycoside antibiotics (e.g., kanamycin, neomycin, gentamicin, amikacin)

KEY POINT

Anaerobes are resistant to aminoglycosides (as are facultative bacteria multiplying anaerobically) because these antibiotics require oxygen for transport into cells; the aminoglycosides may kill the normal bacterial flora at the site of infection and thus allow unchecked multiplication of anaerobes.

- If anaerobic bacteria are thought to be playing a significant role in an infectious process, the method of sample collection must allow survival of these bacteria. The preferred techniques were described earlier under Collection Techniques.

INITIAL PROCESSING AND EXAMINATION OF SAMPLES

Regardless of whether you decide to culture a sample within your practice laboratory or send it to a specialist microbiology laboratory, there are a number of procedures that should be performed within your practice that will greatly enhance your understanding of the disease process under investigation. These will allow you to make rational decisions regarding therapy and management of your case. These procedures include criteria for sample rejection and making, staining, and examining smears.

Criteria for Sample Rejection

There are a number of samples that *should not be processed* because results will be misleading:

- Dried swabs (see below)
- Swab samples obtained from draining tracts
- Samples of tissue with gross external contami-

nation should be rejected if an *appropriate* representative sample cannot be taken from a central portion of the lesion.
- Samples from an autopsy when the organ/tissue has been subjected to room temperature for greater than 4 hours, or when samples arrive at room temperature, should not be processed without noting and using guarded interpretation.
- Urine samples should be rejected if not refrigerated (see below).

Smears

Smears of the original sample are essential to the interpretation of any bacteriologic cultivation. These may be performed on the sample once it reaches the laboratory, or if there is a delay anticipated between sample collection and processing, they should be made as soon after collection as possible. These smears may be then stained at a later time.

KEY POINT

Two smears should be made from each sample collected and stained with Diff Quik and Gram stain, respectively. These smears should be evaluated before a decision is made to proceed with cultivation of the sample.

To make a smear of a fluid sample (e.g., pus, joint fluid), place one drop of the sample onto a clean microscope slide and smear the material as you would a blood smear. If the sample is a piece of solid tissue, place the sample onto a slide and press the tissue onto the slide, as for an impression smear. If a swab was used to collect the sample, make a smear by rolling the swab onto the surface of a glass slide using mild pressure. It is preferable, if using a swab, to either collect two swabs at the time of collection (one for culture and one for smears) or if only one swab was collected to culture the sample before making the smears. Allow smears to dry thoroughly *in air* before staining.

KEY POINT

Examination of smears made from the original sample is essential for assessment of the original numbers of bacteria in a sample and assessment of the ratio of the different morphologic types of bacteria. They are especially important when interpreting cultures of samples placed in transport medium and for samples suspected to contain anaerobic bacteria.

Stains

DIFF QUIK

The first smear should be stained with a cytologic stain (e.g., Diff Quik) and evaluated for the presence of inflammatory cells (neutrophils, macrophages, lymphocytes). The presence of intra- or extracellular bacteria may also be observed in this preparation, although the Gram reaction cannot be determined because all bacteria will stain purple. The Diff Quik stain is as sensitive, if not more so, as Gram-stained preparations for detecting bacteria (especially gram-negative rods). Furthermore, it is far better for assessment of cellular responses to bacterial infection and for assessment of toxic changes to neutrophils.

KEY POINT

If there is no evidence of an inflammatory process in your smears but bacteria are present, it is more likely that you sampled from a site with a normal flora rather than one with an infectious process. In these cases the bacteria that you culture are unlikely to be significant and cultivation is not *recommended.*

GRAM STAIN

If inflammatory cells and bacteria are observed in the Diff Quik-stained preparation, a second smear may be made from body tissues or fluids and should be stained with Burke's Gram stain (see Appendix in this chapter for details). Record the relative numbers of each morphologic type of bacteria (it may be helpful to draw, using colored pens, the morphology and Gram reaction of all organisms).

SPECIAL STAINS

There are a number of special stains and different cytologic preparations available that may be used to help identify certain bacteria. These include acid-fast stain, modified acid-fast stain, wet films, saline wet films, India ink preparations, and 10% KOH to name a few. These stains are occasionally used to help identify more uncommon bacteria (e.g., *Mycobacterium* spp., *Brucella*, *Nocardia*, *Rickettsia*, yeasts) and are probably not within the realm of most routine equine practices. If unusual bacteria or fungi are observed within the original stained smears or suspected based on history and physical examination, these samples should be submitted to a specialist laboratory for culture and identification of the organism.

REFERRAL OF SAMPLES TO A SPECIALIST LABORATORY

Once smears have been examined, a decision must be made as to whether the sample can be processed within your own practice laboratory or whether it requires specialist attention. Types of samples to be sent to a specialist laboratory would vary between practices and would depend on the cost effectiveness to process the samples in your own laboratory. In general, it is not cost effective for practices to keep specialized media that are required for fastidious and uncommon organisms. Examples of this situation include

- Samples that have been previously culture "negative" but in which the disease process is ongoing or in which bacteria were (and still are) observed on a Gram-stained smear.
- Samples in which organisms of unusual morphology or staining pattern were observed in stained smear preparations:

  Example 1: Gram-positive spore-forming rods obtained from an aspirate of muscle tissue in suspected cases of myonecrosis. The organisms will probably be *Clostridium* spp. and will require a system for anaerobic cultures. If you do not keep these in your practice laboratory, you will need to send these samples to a specialist laboratory or, alternatively, treat and manage this case based on a morphologic diagnosis.

  Example 2: Gram-positive branching rods that may be either *Actinomyces* spp. or *Nocardia* spp. Both bacteria are relatively unusual causes of infections in horses and may be difficult to culture. Consequently, you may wish to send away these samples for cultivation.

  Example 3: Samples in which fungi (either yeasts or filaments) are observed, especially involving systemic infections. This would not include ringworm infections that may be easily diagnosed in practice, e.g., with Fungassays.

  Example 4: Cases in which history or clinical signs make you suspect an unusual organism that is fastidious (difficult to grow; e.g., *Mycobacterium* spp., *Brucella* spp., *Taylorella equigenitalis).*

"Specialist" procedures for cultivation of fastidious and unusual bacteria are not discussed in this chapter.

TRANSPORTATION OF SAMPLES TO THE LABORATORY

If samples are to be submitted to a referral laboratory or if one anticipates a delay between collection and processing of samples, then appropriate measures must be taken to ensure maintenance of viability of organisms during this delay. In addition, samples must not be allowed to spill or discharge into the environment.

KEY POINT

The two basic aims during transportation of samples to the laboratory are preservation of the viability of organisms and preservation of the original numbers and proportion of species.

Preservation of Viability of Organisms During Transportation: Commercially Available Transport Media

The best way to preserve the viability of microorganisms present in samples is by processing the sample *as soon as possible* and *not allowing the sample to dry out.* Careful attention must be paid to swabs, which are particularly vulnerable to drying. A number of different transport media are available to help prevent samples drying out and are necessary if a delay is anticipated between collection and processing:

- For transportation of samples collected with a swab, a variety of systems are available that contain media to prevent the swab from drying and allow preservation of aerobic or facultatively anaerobic bacteria during transportation (see earlier under Swabs).
- If solid tissue is to be transported, the BBL Port-A-Cul Transport System Vial (BBL Division of Becton-Dickinson and Co., Cockeysville, MD) may also be used.
- If strictly anaerobic bacteria are suspected, place sample into a commercially available medium that is capable of supporting the growth of strictly anaerobic bacteria (e.g., Port-A-Cul Vial or BD Vacutainer Anaerobic Specimen Collector, Becton-Dickinson and Co., Rutherford, NJ).
- If the contagious equine metritis organism *(T. equigenitalis)* is suspected, Amies medium should be used to moisten and transport swabs after collection.

KEY POINT

Body fluids such as blood, CSF, joint fluids, abdominal fluids, and thoracic fluids contain factors that inhibit or kill bacteria and must be processed immediately after collection by inoculation into an appropriate medium (e.g., Oxoid Blood Culture Bottles; Oxoid USA, Columbia, MD).

- Transportation of urine samples differs from other specimens collected. Transport the sample to the laboratory as quickly as possible and keep the temperature less than 15°C. If a delay of greater than 20 minutes is anticipated, place at 5°C. Even 20 minutes at room temperature can change results significantly. Bacteria can grow readily at room temperature and white blood cells lyse in this environment. Release of inhibitory substances from lysed white blood cells will interfere with cultivation of the urine.

Preservation of Original Numbers and Proportion of Bacteria

The second aim of preservation of original numbers and proportion of bacteria is more difficult to achieve. The reason for this is that different genera of bacteria can multiply at different rates, whereas others may die out completely during the processing of the sample. Furthermore, although transport media prevent drying of the sample, they do not inhibit multiplication of bacteria. Thus, the number and proportion of bacteria present within the sample at the time of collection may be greatly altered after transportation. One way to prevent unchecked multiplication of bacteria is to keep the sample cool but preferably not frozen.

KEY POINT

Uncontrolled freezing kills a large percentage of bacteria. Cooling (to approximately 15°C) keeps most organisms alive but retards their multiplication.

In addition, a good way to determine the numbers and proportion of bacteria in your original sample is to make a thin smear at the time of collection that may be later stained and examined (see above). The smear will indicate the numbers, morphology, and Gram reaction of the different bacteria in your sample at the time of collection.

KEY POINT

Thin smears should be made at the time of collection regardless of the method of collection. These will give you some idea of the numbers and proportion of bacterial or fungal species present at the time of sampling and allow you to evaluate the cytologic response.

CULTIVATION OF SAMPLES WITHIN A PRACTICE LABORATORY

If a decision to culture a sample is made after examination of smear preparations, the appropriate primary isolation medium must be chosen for optimal recovery of microorganisms. Selection of the appropriate medium is done on the basis of the sample obtained and the presence or otherwise of possible swarming bacteria (e.g., *Proteus* spp.). In addition, it is essential to inoculate the primary medium with the specimen as soon after collection as possible.

Media for Primary Cultures

Most (if not all) media listed below are available as prepoured plates or broth cultures from bacteriologic suppliers and are a relatively inexpensive alternative to pouring your own plates. It is necessary, however, to ensure the quality of basal medium. To get growth of *Streptococcus* spp. and delicate gram-negative organisms, there is a need to ensure that a yeast or other extract is incorporated into the medium. Many blood agar bases and nutrient broths and agar do not contain yeast extract and thus may limit the range of bacteria grown. Check with the manufacturer/bacteriologic supplier to ensure that these supplements are added.

The media listed below are basic requirements that would be useful for cultivation of nonfastidious bacteria within a practice laboratory. It should be noted that the media listed are recommendations only, and a number of other media may be substituted as long as the underlying principles remain. More specialized media are also available, if required, for isolation of more fastidious bacteria (e.g., *Mycoplasma).* However, because these organisms are relatively uncommon causes of infections in horses, these media are rarely required in routine practice laboratories and will frequently reach their expiry date before use. Consequently, most practices choose to send samples containing suspected fastidious or unusual bacteria to a referral laboratory.

KEY POINT

The basic goal in a practice laboratory is to keep as few different types of media in your practice as possible but allowing the cultivation of bacteria that are commonly encountered. This will reduce waste and cost of processing.

SOLID CULTURE MEDIA

Two Percent Horse or Sheep Blood Agar Plates

Blood agar plates are the most common plates used for primary isolation of bacteria from sam-

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ples because this medium will support the growth of most fastidious bacteria (e.g., *Streptococcus* spp.) and less fastidious bacteria (e.g., *Staphylococcus* spp., Enterobacteriaceae, *P seudomonas aeruginosa).*

If swarming bacteria (e.g., *Proteus* spp.) are suspected, the sample should be placed onto a 4% agar blood plate as well because the increased agar concentration retards swarming of *Proteus* spp. However, it is worth remembering that on 4% agar, the typical colonial morphology of bacteria may not be seen because of the surface-restricting properties of the medium.

MacConkey's Agar Plates

If the sample collected is thought to contain Enterobacteriaceae, it may also be desirable to place the sample onto a MacConkey's agar plate (e.g., endometrial swabs, aspirates or swabs from wound infections). MacConkey's agar contains bile salts that inhibit growth of most gram-positive bacteria and nonenteric gram-negative bacteria. In addition, it contains lactose and a neutral red indicator, allowing differentiation between lactose fermenters (e.g., *Escherichia coli)* and nonlactose fermenters (e.g., *Salmonella, Proteus).* Finally, *Proteus* spp. will not swarm on MacConkey's agar plates because of the higher surface tension associated with the bile salts present in the medium.

Chocolate Agar

This medium is required for growth of the fastidious bacterium *T. equigenitalis* (the causative agent of contagious equine metritis). If sampling for this organism is routinely conducted within your practice, you may wish to include this agar within your inventory. Samples should be inoculated onto a Columbia base chocolate horse agar (Oxoid USA, Columbia, MD) and incubated in 5 to 10% CO, in humidified air for 48 to 72 hours at 37°C. Some authors also recommend addition of L-cysteine HCl (100 mg/L).

Sabouraud's and Mycosel Agar Plates and Fungassays

Isolation of yeasts and/or filamentous fungi (other than dermatophytes) is not performed frequently in equine practice. If you wish to culture these organisms (e.g., corneal swabs in cases of suspected fungal keratitis), inoculate the sample onto a Sabouraud's agar plate. If dermatophytes (ringworm) are suspected, Mycosel (BBL Division of Becton-Dickinson Co., Cockeysville, MD), which has chloramphenicol and cycloheximidine added to inhibit growth of insignificant fungal contaminants, should be inoculated with your sample. Alternatively, a Fungassay test may be used for differentiation of dermatophyte infections. These are cheap, simple to use, and easy to interpret and are recommended for practices in which dermatophyte infections are diagnosed frequently. Plates and Fungassays should be incubated at room temperature (25-30°C) for up to 10 days before discarding and interpreting the result as negative.

LIQUID CULTURE MEDIA

KEY POINT

Blood and inflammatory cells contain substances that are inhibitory to bacterial growth. Hence, if large amounts of these fluids or cells are present in the sample collected, the sample should be placed into liquid culture media so that these substances are diluted out. As a general rule, fluid from enclosed body cavities (i.e., blood, abdominal, thoracic, joint, and CSF) should be cultured in liquid media.

Commercially Available Liquid Culture Media

Special media are available for cultivation of blood and are also recommended for cultivation of other fluids (e.g., abdominal, thoracic, joint fluid, CSF, etc.). Care must be taken in the use of commercial media for joint fluid and CSF so that you do not dilute organisms so much that growth does not occur. Up to 10 mL of fluid should be inoculated into the medium, but smaller volumes are acceptable. Do not exceed this volume, however, because inhibitory substances present within these fluid samples may inhibit growth. An additional advantage of these fluids is that they will *support growth of many strictly anaerobic and microaerophilic bacteria* without needing to be placed in an anaerobic or microaerophilic environment. An example of this type of medium is the Signal Blood Culture Bottle (Oxoid USA, Columbia, MD). These media are discussed further under *Special Laboratory Processing for Selected Tissues-Blood Cultures.*

Brain Heart Infusion Broth

An alternative to liquid culture medium obtained from a commercial source is one that is prepared within the practice. A suitable medium in this instance is brain heart infusion broth (Oxoid USA) and is prepared according to the manufacturer's instructions. In this instance, the volume of sample inoculated may vary from 2 to 10 mL depending on the numbers of organisms seen and the amount of blood (generally a dilution of 1:5 to 1:10 is required).

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Cooked Meat Medium and Prereduced Medium (e.g., Thioglycolate Broth)

These media may be used if strictly anaerobic bacteria are suspected to be involved in the disease. These media have reducing agents that support the growth of anaerobes.

Inoculation of Primary Media with Sample

SWABS

Swabs usually are inoculated directly onto agar plates. The swab should be rolled onto one corner of the agar plate repeatedly to inoculate the organisms. A platinum wire loop then should be used to streak the remainder of the plate with the inoculum. It should be remembered to heat the loop until it is red (with a flame from a Bunsen burner) between inoculation streaks so that single colonies may be obtained. The loop should be allowed to cool after flaming and before streaking so that the bacteria will not be splattered into the environment or killed due to an excessively hot loop.

TISSUES

If the sample obtained for cultivation is a piece of tissue, it may be directly inoculated onto a blood agar plate or placed within a liquid culture medium. However, frequently the bacteria are situated within the tissue and will not be released onto/into the medium, resulting in a false-negative culture. Consequently, it is recommended that pieces of tissues are first disrupted before inoculation into primary cultivation media. This may be achieved by simply chopping the tissue into small pieces with a sterile scalpel blade, adding them to a sterile mortar together with a small volume of sterile medium (e.g., BHIB, Oxoid USA) or sterile saline, and grinding the tissue with a sterile pestle. With a flamed platinum loop, obtain a loopful of the ground tissue and inoculate an appropriate agar plate and streak for single colonies as described above. Alternatively, the tissue may be placed into a sterile plastic bag along with a small amount of sterile medium and processed in a Stomacher 80-lab blender (Tekmar Co., Cincinnati, OH). This machine gently extracts the bacteria or fungi from tissues and so greatly enhances culture of bacteria from tissue samples. A loopful of the extracted fluid is processed as above.

FLUIDS

Abominal, Thoracic, Joint, Blood, and CSF

Fluids from enclosed body spaces (e.g., blood, peritoneal/thoracic fluid, joint fluid, CSF) *must* be inoculated into a liquid culture medium (e.g., Blood Culture Bottle, Oxoid USA). In addition, they may be inoculated directly onto solid medium (agar plate) if time is a consideration in the diagnosis of these infections.

Pus, Tracheal Wash, Milk, and Urine

Unlike samples from enclosed body spaces, these fluids do not need to be inoculated into a liquid culture medium; rather it is preferable to inoculate these samples onto a solid medium (e.g., *2%* blood agar). The solid medium will allow better differentiation between pathogens and contaminants, which are frequently encountered in these samples (see Interpretation of Primary Culture Results). However, liquid media may be used in addition to solid medium if large numbers of inflammatory cells are observed in smear preparations. To inoculate a solid medium (e.g., blood agar plate) with these samples, first eject a few drops of fluid sample from the syringe into an appropriate disinfectant, then place one to two drops of fluid onto a corner of the plate and use the platinum wire loop to spread the drop over this section. The wire loop should then be used to streak the remainder of the plate with the inoculum.

Atmospheric Conditions and Temperature for Incubation

All specimens should be placed into the appropriate atmospheric conditions, depending on the clinical history and appearance of the Gram stain. It usually is possible to determine if strictly anaerobic and/or microaerophilic bacteria are suspected using these two criteria.

AEROBIC CONDITIONS

- Aerobic conditions can be achieved by incubation under normal atmospheric conditions, usually at 37°C. Room temperature (25-30°C) should be used if molds are suspected.

MICROAEROPHILIC CONDITIONS

- Microaerophilic conditions should be used *only* if required for primary isolation of organisms, and the history and clinical signs determine this. Organisms that require microaerophilic conditions include *T. equigenitalis*, *Brucella* spp., and *Campylobacter* spp. However, because these organisms are infrequently cultured in routine equine practice, you may wish to send samples that you suspect contain these bacteria to a specialist laboratory.

- Alternatively, microaerophilic conditions can be achieved either by the addition of a commercially available gas-generating system similar to that used for anaerobic bacteria ($C0_2$-generating kits, Oxoid USA) or with the use of a Candle Jar.

ANAEROBIC CONDITIONS

- Anaerobic conditions should be used only if required for primary isolation of strictly anaerobic organisms. Remember that it is not recommended to perform anaerobic culture on specimens that have a significant population of normal floral anaerobes.
- The simplest method for achieving anaerobic conditions in a clinical setting is to use one of the many commercially available systems. This includes the AnaeroGen system (Oxoid USA), BBL GasPak Jar (Becton-Dickinson Microbiology Systems, Cockeysville, MD), the Anaerobic system (Difco Laboratories, Detroit, MI), and the GasGendicator system (Adams Scientific Inc., West Warwick, RI).
- Most systems listed above use disposable gas-generating envelopes that remove oxygen from the chamber by reaction of hydrogen with a palladium catalyst and result in the liberation of H_2 and CO_2. The use of a catalyst in these systems is necessary, an example of which is the "cold" catalysts available from Oxoid USA. All catalysts should be rejuvenated after each use by heating to 160°C for 1 to 3 hours to drive off moisture and H_2S. While still hot, they should be placed into a desiccator.
- An alternative system is the AnaeroGen system (Oxoid USA), which has an internal generator and does not require a catalyst or hydrogen to generate anaerobic conditions. Furthermore, this system generates anaerobic conditions faster than those using the palladium catalyst. This may result in increased ability to culture the more O_2-sensitive strictly anaerobic bacteria. The AnaeroGen system also comes in a small disposable pouch system that can hold four plates or 1 microtiter plate and that would replace the large "jars," which can be expensive and take up considerable space within an incubator.
- All anaerobic systems require the use of a disposable "resazurin" indicator strip (which also can be bought from the above companies) as an indicator that anaerobic conditions have been reached. Anaerobic conditions should occur within 3 hours after incubation, and the jars should be checked at that time to ensure that the indicator strip is white (indicating anaerobic conditions).

Storage of Samples

The residual component of samples subjected to culture should be stored in either a sterile container or in a syringe from which air has been excluded. Specimens may be stored at 5°C for up to 3 days after primary setup. If you are unable to culture bacteria in your practice laboratory, you may wish to send these samples to a specialist laboratory for more specialized culture conditions.

SPECIAL LABORATORY PROCESSING FOR SELECTED TISSUES

Blood Cultures

INITIAL PROCESSING AND BLOOD SMEARS

- Blood samples must be submitted immediately to the laboratory so that processing can commence before clotting of the sample. Alternatively, the sample may be inoculated directly into a liquid culture medium (see above) at the time of collection.

KEY POINT

All procedures must be done rapidly before the blood clots (usually within 2 minutes).

- A second sample of blood preserved in EDTA is required for making of blood smears. Slides for blood smears should first be prepared by swabbing the slides with 70% alcohol and allowing them to dry. These slides should then be handled such that fingerprints and squames and dust do not contaminate the slide. The tube containing the blood sample should be rotated to mix the blood and a small drop of blood should be placed onto a slide using a microhematocrit tube. The edge of another prepared slide should be dipped into the drop of blood so that approximately one quarter of the drop adheres to the edge of slide. This slide is then used to smear a third slide so that a thin blood film is achieved. It is best to prepare several thin slides for Gram staining.
- Stain the blood films with a Gram stain but cut down the staining cycles to 10 seconds each.

KEY POINT

Remember to blot the slide dry after decolorization and before application of

safranin. This procedure is necessary for accurate staining of blood-containing smears of any origin.

- Examination of blood smears may be immediately rewarding because bacteria may be observed directly and in high numbers. However, frequently, low numbers of bacteria are present and examination may require a search of up to 20 to 30 minutes. In these cases the low number of bacteria makes it easier to mistake bacteria for stain debris or other contaminants. Consequently, you may wish to send these slides to a referral laboratory for examination and confirmation of findings.

KEY POINT

Accurate observation and interpretation of the Gram-stained original smear are of major diagnostic significance. If you are unsure of your interpretation of these smears, they should be sent to a referral laboratory.

CULTIVATION OF BLOOD SAMPLES: BLOOD CULTURE MEDIA

- Blood contains bacteriostatic agents (e.g., acidic polypeptides released from neutrophils) that will inhibit growth of significant numbers of bacteria if these substances are present in high concentrations (e.g., if cultured on blood plates). Blood also clots, preventing the growth of bacteria. Therefore, all blood samples should be inoculated into a broth medium at a dilution of 5:1 (i.e., 5 parts broth to 1 part blood). This dilution will overcome the inhibitory phenomena and allow bacteria to grow within the culture medium.

KEY POINT

Commercial blood culture bottles are available that contain sodium polyanetholesulfonate (SPS), a substance that "neutralizes" the neutrophils and antimicrobial agents in the sample and prevents clotting.

- SPS is a polyanionic anticoagulant that is supplemented at the rate of 0.025% to 0.05% in most commercially available blood culture media. It acts to inhibit complement and lysozyme activity, interferes with phagocytosis, and inactivates residual clinically achievable concentrations of aminoglycosides not neutralized by dilution.
- An example of a blood culture medium is the Signal Blood Culture System (Oxoid USA), but there are a number of other similar systems available. Many of these systems will also support the growth of strictly anaerobic and microaerophilic bacteria and so can be used when these organisms are suspected as the cause of septicemia.
- Alternatively, prereduced anaerobically sterilized brain heart infusion broth (Oxoid USA) may be prepared in your own laboratory and used for blood cultures. This medium, dispensed in aliquots of 10 mL, will sustain the growth of most bacteria-causing septicemia in the horse and can be used routinely to culture blood samples.

INOCULATION OF MEDIA AND ASSESSMENT OF POSITIVE CULTURES

- Ten milliliters of blood should be inoculated into a blood culture bottle as per manufacturer's instructions or 2 mL of blood can be added to each of four to five brain heart infusion broth bottles. Cultures should be incubated for 24 hours at 37°C.
- If commercially available media bottles are used, it is recommended to use at least two bottles per animal (10 mL of blood per bottle). This is to check for contamination, which can occur at the time of sampling or when sampling broth cultures for bacterial growth. In this way, if bacteria grow in one culture bottle but not in another, the bacteria that are growing are *probably* a contaminant.
- After 24 hours, blood cultures should be observed for evidence of bacterial growth (either turbidity within the medium or by small white colonies on top of the settled red blood cells). If growth is evident, blood cultures can be sampled and Gram-stained and subcultured onto a blood agar plate.

KEY POINT

It is important to use aseptic procedures when sampling broths to ensure that false-positive results do not occur. Always leave at least one broth undisturbed to check the relevance of any isolate.

- It may take 4 to 7 days for a positive sample to show growth; therefore, do not discard your cultures before this. Incubation time will depend on the number of organisms present and the species of organism growing. Once organisms have been subcultured to a blood agar plate, identification and sensitivity testing can proceed as with any organism.

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Fluids from Enclosed Body Cavities (Abdominal, Thoracic, Joint, CSF)

- These body fluids, like blood, contain factors that inhibit or kill microorganisms. They also may contain high numbers of neutrophils that will also inhibit growth of bacteria, especially if they deteriorate/disintegrate and release the cell contents into the fluid. It therefore is imperative to process these samples without delay.
- As for blood cultures, the most important thing to remember when sampling these sites is that the sample must be inoculated into a liquid culture medium to dilute out these inhibitory substances. The liquid culture media available for blood cultures (see above) are also suitable for use with joint, thoracic, abdominal, and CSF.
- However, on occasions, large numbers of bacteria may be present in these samples, which will facilitate growth of the bacteria. Consequently, you may choose to inoculate a blood agar plate at the same time as inoculating a liquid culture medium. This will allow the saving of 12 hours for identification of the organism and in cases of acute disease, when time is vital, this is worth considering.
- In cases where low numbers of bacteria are suspected or when bacteria may be sequestered, for instance in the synovial lining, it is recommended that the sample also is centrifuged, the supernatant fluid removed, and the pellet resuspended in a small amount of liquid culture medium before inoculation into liquid culture medium.

KEY POINT

It is very important to remember that strictly aseptic techniques *must be used when using liquid culture media. This is due to the fact that contaminant bacteria can multiply in these media and can be present in large numbers after incubation. These bacteria will interfere with the interpretation of any significant isolates present in your sample.*

Urine

KEY POINT

Urine samples should be processed and results interpreted differently from most other fluid samples.

- There is a strict set of criteria drawn up for cultivation and interpretation of human urine samples. Similar work has not been done in veterinary medicine, but there are some guidelines (based on these studies) that are generally followed.
- Urine should be collected to avoid contamination with normal urethral/vaginal flora (e.g., a midstream-voided sample is useful only if the vulva/prepuce/penis has been cleaned and dried thoroughly before voiding). Samples collected by catheterization are preferable to decrease the contamination from normal flora of the lower urinary tract.

KEY POINT

All urine samples should be refrigerated immediately after collection. Even 20 minutes at room temperature can change results significantly.

INITIAL PROCESSING

- A full urinalysis should be carried out on urine samples submitted for bacteriology. This is necessary to allow interpretation of findings. The urinalysis should include examination of a wet preparation under high dry magnification (40 X objective) to look for bacteria and inflammatory cells. It is usual to consider a possible urinary tract infection if greater than five white blood cells and greater than 10 bacteria are seen per 40 X field.
- When using a direct cell count of the urine sample with a disposable chamber (e.g., Kova Glasstic Slide 10 with grids, Hycor Biomedical, Inc., Irvine, CA), if greater than 10^4 white blood cells/mL of urine are present, then the possibility of a urinary tract infection should be considered.
- If there is evidence of a bacterial cystitis, a smear and Gram stain should be performed. The Gram reaction and morphology of bacteria present and the relative proportions of each type of bacteria should be noted. The number and type of inflammatory and other cells seen in the smear also *must* be noted, because these will help in the interpretation of culture results.

CULTURE OF URINE SAMPLES

- For valid interpretation of urine cultures, quantitative urine cultures should be performed. Because cystitis is relatively uncommon in the horse, most practices will have little or no experience with urine cultures in this species. Therefore, if there is evidence of a urinary tract infection after urinalysis and evaluation of a Gram-stained smear of the urine, the samples can be submitted to a referral laboratory for quantitation and identification of bacteria.

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- A sensitivity test also should be requested, because the bacteria that commonly cause cystitis in the horse do not have predictable sensitivities (e.g., *E. coli, P. mirabilis, Klebsiella* spp., *Enterobacter* spp.).
- It is important to remember when submitting urine samples for culture that urine must be kept cold (4°C) until processed because bacteria can multiply rapidly in urine at room temperature and so will invalidate quantitative tests performed on the urine sample. Urine may be stored at 4°C for 24 hours without any significant change in the numbers of bacteria. However, leukocytes will disintegrate after several hours, and the viability of bacteria will decrease on extended storage. Both factors adversely affect interpretation of results.
- After results of the culture and sensitivity are received and the animal is placed on the appropriate antimicrobial agent, the urine should be reexamined after 5 to 7 days of therapy to check that the therapy has been effective. It may be sufficient to obtain a *fresh* midstream urine sample for this examination. Examination will consist of a check of pH, presence of inflammatory cells, and presence of bacteria. It may be necessary to centrifuge the sample and examine the deposit. If bacteria are seen, withdraw antibiotics for 24 to 48 hours and reculture the urine.
- Anaerobic bacteria rarely cause cystitis, and samples are not routinely cultured anaerobically.

Feces

KEY POINT

Feces should only *be cultured for specific organisms.*

Because feces can contain a multitude of organisms, many of which are not pathogenic, the search should be directed to the question, Does this fecal sample contain a specific bacterium, e.g., *Salmonellal*

SALMONELLA

KEY POINT

Because Salmonella *may be shed in very low numbers in feces of horses with salmonellosis, a total of three to six fecal samples (approximately 5 g of feces per sample) should be collected at 12-hour intervals to increase the possibility of diagnosis.*

- Because of the presence of large numbers of normal flora of the gastrointestinal tract in fecal samples, feces should be inoculated into enrichment media that retard the growth of many of the normal flora and selectively enrich the growth of *Salmonella.* Hence, feces should be inoculated into tetrathionate or selenite broth cultures (Oxoid USA; Difco, BBL Division of Becton-Dickinson and Co.). Inoculate between 1 and 5 g of feces into approximately 20 mL of enrichment media. Direct plating onto xylose lysine deoxycholate (XLD) or MacConkey's agar (Oxoid USA) also may be useful at this stage on the chance that *Salmonella* are present in high numbers.
- The tetrathionate or selenite broth cultures should be subcultured to XLD or MacConkey's agar at 12 and 24 hours and these plates then incubated a further 12 to 24 hours at 37°C.
- Suspect colonies (black colonies if XLD agar is used or white colonies if MacConkey's agar is used) may be subcultured to obtain pure growth or tested directly to see whether they are smooth using fresh acriflavine solution. Smooth colonies should then be tested biochemically to verify that *Salmonella* are present (see below under Biochemical Tests), and if this is the case they should be tested against poly-O and poly-H *Salmonella* typing sera (e.g., BBL Division of Becton-Dickinson and Co.) or sent to a referral laboratory for serotyping.

INTERPRETATION OF PRIMARY CULTURE RESULTS

KEY POINT

Unless the sample has been collected thoughtfully and carefully, obeying the principles set out earlier, meaningful interpretations of laboratory results often are impossible.

It is important to interpret the likely significance of colonies present on primary cultures so that a decision can be made to continue with identification of the organism(s) isolated, and possibly sensitivity testing, or to discard the culture as probable contamination. Even with a well-taken specimen, interpretations can be difficult.

KEY POINT

The site from which the sample was collected, the type of medium on which the sample was cultured, the number of colonies observed, and the number of different types of bacteria observed will influence your decision to proceed. In addition, this must be related to

the morphology, Gram reaction, and ratios of bacteria in the original smears.

Interpretation of Isolation of Bacteria from Normally Sterile Sites

SOLID MEDIA: PURE CULTURE

KEY POINT

If the primary blood agar plate yields a pure culture, do not immediately assume that it is the organism responsible for the infection.

- If you are sure that you collected your sample in an appropriate fashion from a normally sterile site, then you can be reasonably confident that the organisms you are growing may be contributing to the infectious process and continue with identification. This is particularly true if a heavy growth of one type of bacteria is obtained. Low numbers of one type of bacteria may still be significant, if the sample has been collected appropriately, but *must be interpreted with more caution.*
- In addition, the possibility still exists that the real causative organism of the infectious process has not grown, and the organism isolated is simply multiplying in a favorable environment. Gram stains of the original sample will aid the interpretation in this case.

SOLID MEDIA: MIXED CULTURE

- If more than one organism is obtained from a normally sterile site, a mixed infection may be causing the inflammatory process. This is frequently true in sites with access to or close to a site with normal bacterial flora (e.g., tooth root abscess, hoof abscess). These infections usually will involve aerobic and anaerobic species of bacteria. Identification of all bacteria involved in these infections is frequently not required; rather the predominant species may be selected and identified.
- If two or three different bacterial colonies are obtained in large numbers from a primary culture, each type of bacteria should be identified and sensitivity testing performed if indicated. Colonies that are present in low numbers in these cultures are usually not investigated, unless there is a specific need.
- If, on the other hand, three or four different types of bacterial colonies are observed and all of the colony types are *present in low numbers,* then this indicates *probable contamination of the sample,* and this culture should be discarded without further identification of the bacteria.

KEY POINT

Mixed and scanty growth is common in samples contaminated by normal flora at the time of collection. Do not waste time in trying to evaluate these plates, because the growth of bacteria in such samples is not significant.

FLUID CULTURE MEDIA

- If there is positive growth (turbidity) in fluid culture media, a smear should be made of the medium for Gram staining and examined for presence of bacteria. If only one or two types of bacteria are identified in the Gram-stained preparation, the fluid culture should be subcultured onto a 2% blood agar plate.
- Interpretation of the subcultures are then as for solid media *except* that numbers of colonies are related only to the rate of growth of the bacteria isolated and *not to the number of bacteria present in the original sample.* Consequently, large numbers of colonies present on subcultures of samples from fluid culture medium do not equate to large numbers of bacteria in the original sample and cannot be used to help the interpretation of culture results.
- The presence of many different types of bacteria (three or more) from fluid cultures indicates that either the infection was mixed (and likely involved spread from a site with normal bacterial flora) or that there was contamination of the sample from a site with normal flora. In either case it is not usual to continue with identification of the bacteria involved.

Interpretation of Isolation of Bacteria from Sites with Normal Flora

If you have cultured bacteria from samples obtained from a site with normal flora, three investigations are possible:

1. Full investigation with identification of all organisms found. This is time consuming and only appropriate for a research laboratory.
2. Exclusion of known pathogens (an organism that has previously been shown to cause disease in this site). This should be done thoroughly.
3. Exclusion of known pathogens plus a partial investigation of the normal flora. This is the preferred approach for referral laboratories.

KEY POINT

In many cases, the pathogenic organism(s) belongs to the normal flora, and it may only be present in excess numbers in clinical cases.

On the other hand, the predominance of one species of the normal flora does not necessarily mean it is causing disease. It simply may reflect an imbalance in the flora due to another factor(s). The real pathogen may be suppressed for some reason (e.g., antibiotics, host responses, inappropriate laboratory media, or inappropriate sampling and transport). It is important in these cases to try to determine what is going on. The other laboratory data that you have collected may be of some value in these cases (see below).

IDENTIFICATION OF BACTERIA

If there is positive growth in primary cultures, and a decision has been made that the isolate is probably significant, the next step may include identification of the bacteria to the genus or species level. Identification of bacteria can aid in the interpretation of the likely significance of isolates and provide information that can alter treatment, prognosis, and control of these infections.

Traditionally, bacteria have been identified using "conventional" procedures, including observation of physical characteristics such as colony morphology and odor, results of Gram stain, biochemical reactions, agglutination tests, and sensitivity profiles. These procedures eventually defined the genera and species of bacteria and yeasts and became the reference method by which we confirm and identify isolates. More recently, however, a number of commonly used biochemical reactions have been miniaturized into a more convenient format. This approach is the basis of most current commercial substrate profile systems and has enabled a much more "user-friendly" system to be adopted for identification of bacteria in practice.

In equine practice, identification of bacteria using a combination of conventional methods and substrate profile systems (commercial identification kits) is probably most convenient and is discussed below. It is also useful to have a prior knowledge of the organisms frequently encountered within your practice and to prepare a *flow chart* to enable identification of these frequently encountered bacteria. The flow chart should indicate the appropriate next biochemical test or procedure, allowing you to eventually arrive at identification of the bacteria at either the genus or species level. Isolates that cannot be identified by these flow charts could be sent to a referral laboratory for identification. A simple example of a series of flow charts that may be used in routine equine practice is outlined in Figures 17-1 through 17-5. However, *it must be stressed* that these flow charts are a gross simplification of routine identification of bacteria and have been designed for use in horses and not for other species. In addition, the flow chart has been devised for identification of organisms that are commonly encountered in equine practice and not for identification of more fastidious or unusual organisms. Finally, different bacteria may cause disease in different areas in a country or around the world. These must be taken into account when interpreting this flow chart, and additional bacteria may need to be incorporated into a chart used in your practice.

Time of Subculture

KEY POINT

Before biochemical tests can be performed on bacteria isolated in primary cultures, subculture of colonies is usually required and is aimed at keeping organisms in log phase of growth. This is imperative to ensure optimal enzymatic activity for biochemical testing and also optimal growth for antibiotic sensitivity testing.

The time and frequency of subculturing will depend on the growth cycle of the organism isolated.

RAPIDLY GROWING BACTERIA

- These bacteria are commonly encountered and will grow to maximum colonial size in 16 to 18 hours. Colony size will be 2 to 4 mm. Examples include Enterobacteriaceae, *Staphylococcus* spp., *Pseudomonas aeruginosa, Bacteroides fragilis* types, *Fusobacterium necrophorum,* some *Clostridium* spp., and *Bacillus* spp. These organisms can be handled rapidly (i.e., after overnight growth).
- If the colony obtained in a primary culture is 2 to 4 mm or greater in diameter, subculture a portion of one colony to another blood agar plate. The colonies in this pure culture then can be used to identify the organism involved. Alternatively, if the primary culture is pure (one colony type only) and especially if there are sufficient numbers of colonies (i.e., >5), then colonies on the primary culture plate may be used for biochemical testing.
- The rapidly growing organisms usually can be mishandled and still allow all their biochemical tests to be performed correctly because they have great biochemical reserves.

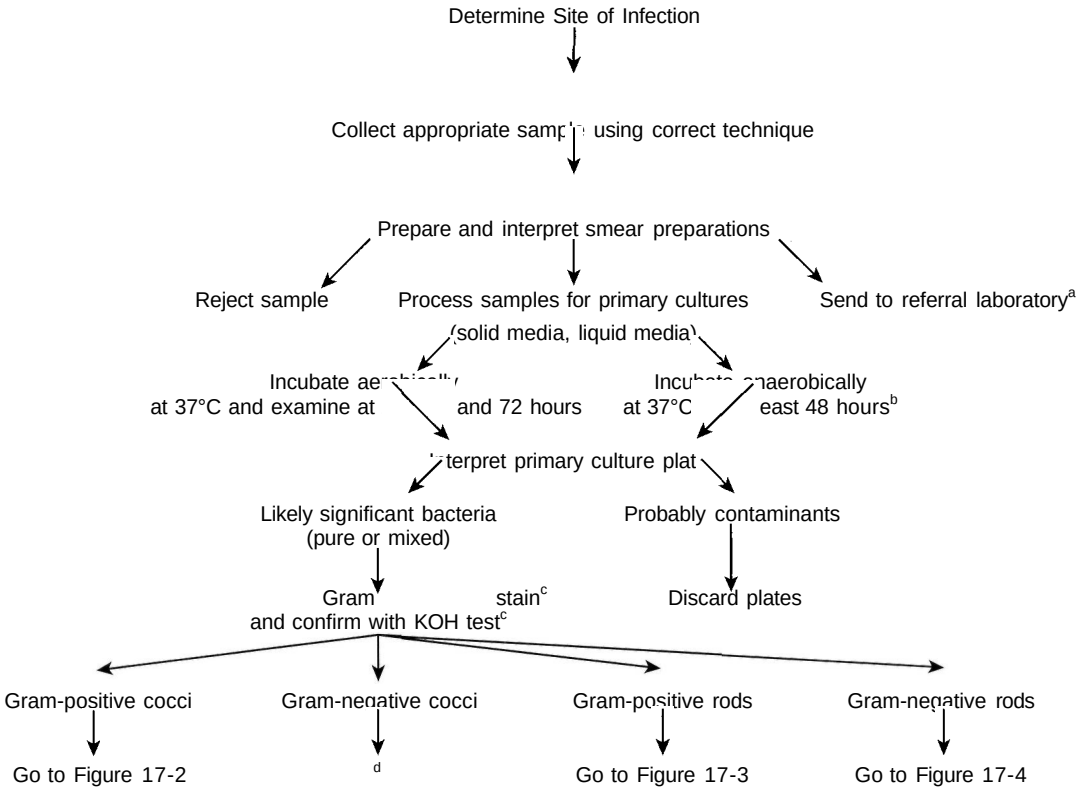


Figure 17-1. Initial procedures for identification of bacteria from equine samples.

NOTES:

[a]Refer to text for times when samples should be sent to a referral laboratory.

[b]Anaerobic cultures should only be performed if indicated (see text).

[c]Descriptions of Gram stains and KOH tests are given in the Appendix in this chapter.

[d]Gram-negative cocci (e.g., *Neisseria* spp.) are unusual causes of disease in horses. Consequently, a flow chart for this group of bacteria has not been prepared. If gram-negative cocci are observed in original smears of the sample, it is more likely that they are gram-positive cocci (e.g., *Staphylococcus* spp., *Streptococcus* spp.) that are staining gram negatively. *Streptococcus* spp. in particular are easy to decolorize and often stain gram negatively or have mixed Gram reactions. These samples should be cultured with this in mind. If, however, gram-negative cocci are detected in smears of colonies grown in primary cultures (and you are sure that you have performed the Gram stain correctly on a young colony), these cultures may be sent to a referral laboratory for identification and interpretation.

ORGANISMS THAT GROW WELL

These bacteria grow to maximum colony size in 24 to 36 hours and colony size is 2 to 4 mm. Examples include *Pusteurella multocida, Actinobacillus* spp., *Streptococcus equi* var. *equi,* and *R. equi.* These organisms usually will require nutrient-rich media for sustained growth (e.g., blood agar), and the original colonies may have limited viability on the surfaces of agar plates. Consequently, organisms in the "good grower" category may require 24-hour subculturing, for example, two to three times, before growth is sufficiently strong to perform biochemical testing.

ORGANISMS THAT GROW POORLY

These bacteria take 48 hours to reach maximum size, and colonies are small (1-2 mm diameter). Examples include *Brucella* spp., *Actinomyces* spp., *Corynebacterium* spp., and some capsulated *S. equi* var. *equi.*

The "poor growers" will require subculturing at 48-hour intervals before placing into appropriate media for biochemical testing. Because of the difficulty in isolation and testing of these organisms, it is usually judicious to send samples in which these bacteria are suspected to a referral laboratory for isolation and identification.

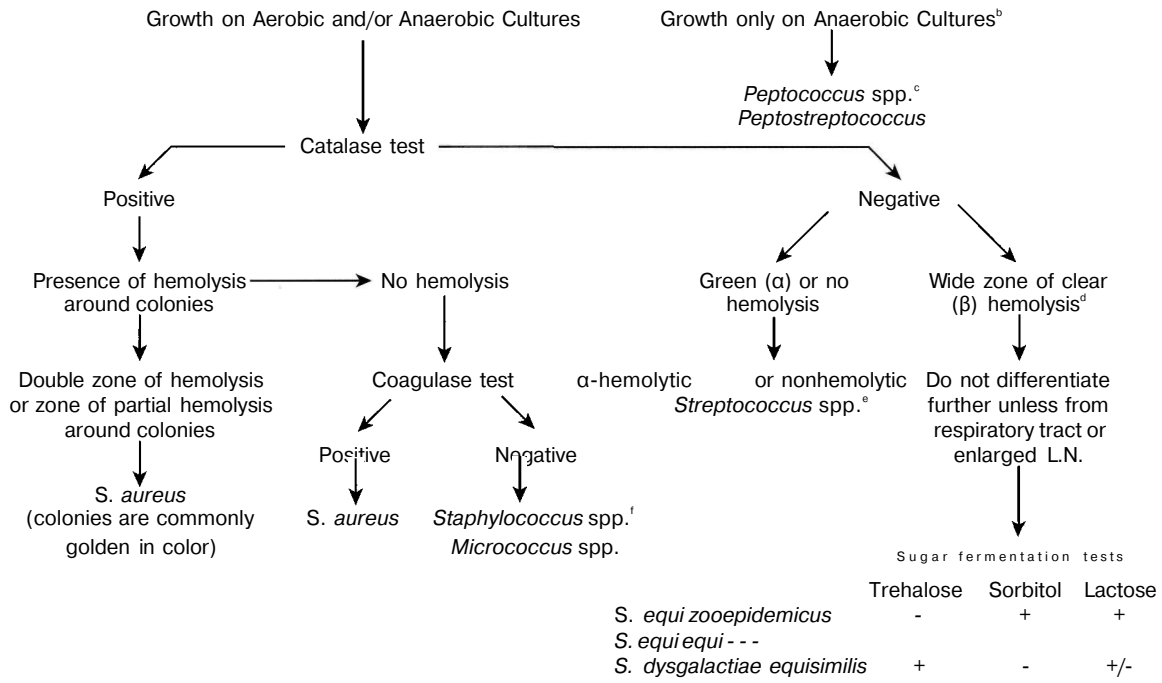


Figure 17-2. Gram-positive cocci.[3]

NOTES:

[a]Gram-positive cocci are important causes of infection in horses and are very commonly encountered in equine practice. It is important to differentiate between these organisms (at times to both the genus and species level) as identification will influence treatment, control, and prognosis in many cases.

[b]Anaerobic cultures should only be performed if indicated (see notes).

[c]It is not necessary to differentiate between *Peptococcus* and *Peptostreptococcus* spp. as this will not influence treatment, control, or prognosis. They are usually found as part of mixed infections involving strictly anaerobic and facultatively anaerobic bacteria.

[d]β-Hemolytic *Streptococcus* spp. are more likely to be pathogenic in horses and may on occasions need identification to the species level.

[e]Care must be taken in the interpretation of isolation of this group of bacteria. α-Hemolytic and nonhemolytic *Streptococcus* spp. are frequently nonpathogenic and may be found as contaminants in cultures. Group D *Streptococcus* spp. are occasionally pathogenic (e.g., bacterial endocarditis, respiratory tract infections, urinary tract infections).

[f]Coagulase-negative *Staphylococcus* spp. and *Micrococcus* spp. are usually nonpathogenic and are frequently encountered as contaminants. Colonies of *Micrococcus* spp. are frequently bright yellow, which may help to differentiate this group of bacteria.

Media for Biochemical Testing

CONVENTIONAL BIOCHEMICAL TESTING

- There are a variety of individual biochemical tests available commercially as tubed media, reagent strips, or agar plates. These simple tests may be sufficient for identification of bacteria without the use of more expensive packaged microbial identification systems. The biochemical tests required to identify the bacteria commonly encountered in your practice will dictate the media purchased. Examples of biochemical tests that may be useful in identifying organisms encountered in equine practice are given in Figures 17-1 through 17-5, and instructions for the performance and interpretations of these tests are in the Appendix at the end of this chapter.

COMMERCIALLY AVAILABLE KITS

- The introduction of packaged microbial identification systems by a number of microbiologic supply companies has made the biochemical identification of bacteria easier to perform for the veterinarian in clinical practice. These system-dependent methods use a set of carefully selected substrates to allow a positive- and negative-reaction pattern to emerge and thus create a metabolic profile to be compared with an established database profile. In these systems,

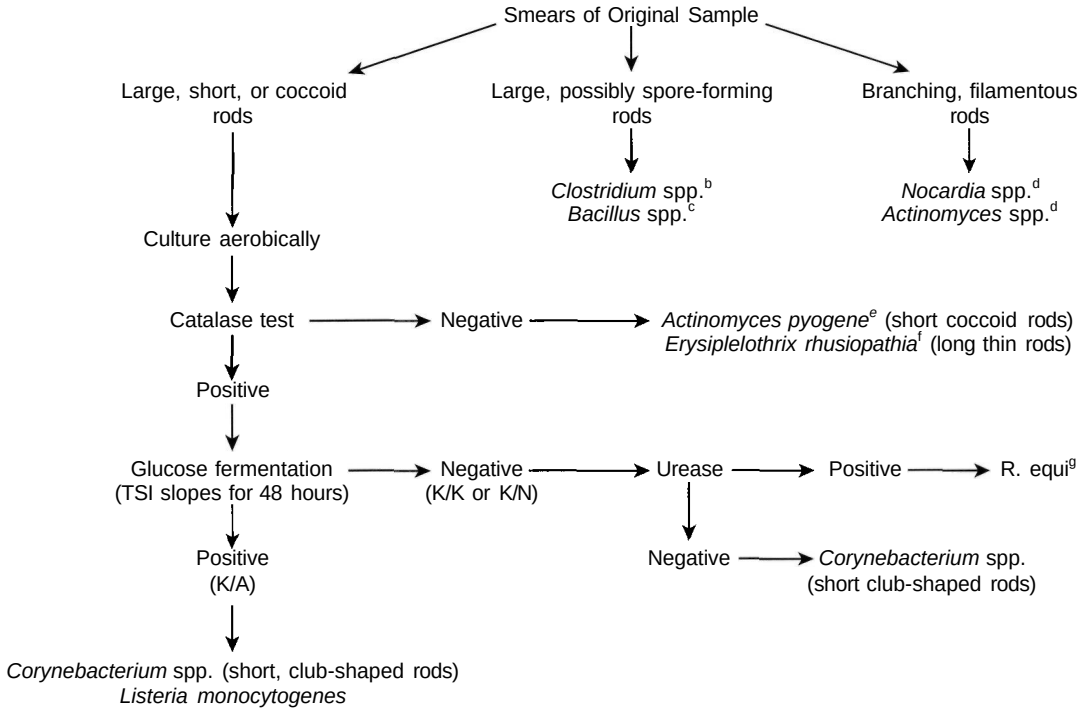


Figure 17-3. Gram-positive rods.[a]

NOTES:

[a]Gram-positive rods are encountered less frequently in equine practice than gram-positive cocci or gram-negative rods. History and clinical signs are *very important* in making a presumptive identification of gram-positive rods as most of these organisms are associated with distinctive disease conditions (e.g., *Rhodococcus equi* infections in foals, *Dermatophilus* [rainscald] infection). In addition, smear preparations of the original sample may aid diagnosis. For example, if large spore-forming rods are observed, *Clostridium* spp. are the probable cause of infection (a far less likely alternative is *Bacillus* spp.) Filamentous branching rods are likely to be either *Nocardia* spp. or *Actinomyces* spp.

[b]*Clostridium* spp. (or their toxins) infrequently cause infections in horses. They may be associated with myonecrosis, diarrhea, wound infections, and other syndromes (e.g., tetanus, botulism, Black disease). The presence of large, spore-forming, gram-variable rods in original smears, particularly in animals with history and clinical signs consistent with the above syndromes, may aid in their diagnosis. *Clostridium* spp. are strict anaerobes, and if positive identification of these bacteria is required, samples should be cultured anaerobically or sent to a referral laboratory.

[c]*Bacillus* spp. are catalase positive, usually hemolytic, and produce large colonies. The Gram stain shows large box car-like rods with spores. They are seldom pathogenic (except *Bacillus anthracis* but anthrax is rare in horses). They are *very common* plate contaminants.

[d]*Nocardia* spp. (strict aerobes) and *Actinomyces* spp. (usually strict anaerobes on initial culture) are usually associated with granulomatous conditions. They are both branching filamentous rods. These organisms may be difficult to culture, and if branching rods are seen in original smears, these samples may be sent to a referral laboratory.

[e]*Actinomyces pyogenes* are very short coccoid rods. They may be mistaken for *Streptococcus* spp. unless careful evaluation of morphology is made. Evidence of chains in broth cultures may help to differentiate.

[f] *Listeria monocytogenes* and *Erysipelothrix rhusiopathiae* rarely cause infections in horses.

[g] *Rhodococcus equi* colonies are characteristic pink mucoid colonies that flow together ("pink spit").

different sets of substrates are necessary to identify the different groups of bacteria encountered (e.g., the rapidly growing members of the family *Entetobacteriaceae,* slower growing gram-negative *non-Enterobacteriaceae* rods, gram-positive cocci, gram-positive rods, anaerobes, and yeasts. A summary list of identification systems currently available and used in equine practice is given in Table 17-1.

KEY POINT

Packaged bacterial identification kits have a "use-by" date. Consequently, kits should only

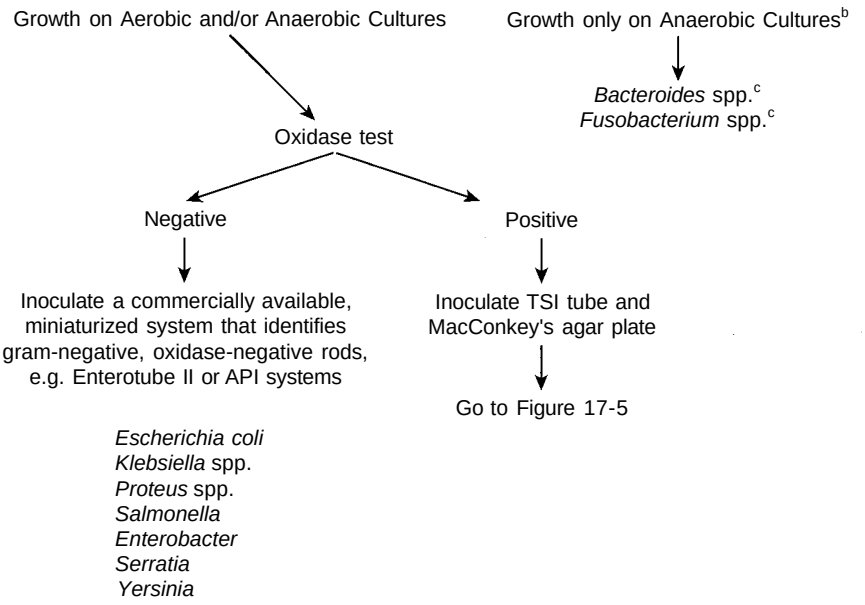


Figure 17-4. Gram-negative rods.[a]

NOTES:

[a]Gram-negative rods are important causes of infection in horses and are very commonly encountered in equine practice. It is important to differentiate between these organisms (at times to both the genus and species level) as identification will influence treatment, control, and prognosis in many cases.

[b]Anaerobic cultures should only be performed if indicated (see text).

[c]Gram-negative, oxidase-negative rods are commonly encountered in equine practice and are important causes of disease. Most gram-negative, oxidase-negative rods belong to the family *Enterobacteriaceae.* However, it is important to identify these organisms to the genus level as not all members of this family are considered to be pathogenic. In addition, identification of some of these organisms (e.g., *Salmonella, Klebsiella)* may have implications for control programs. The packaged miniaturized commercial systems that are available are easy to use and identify this group of organisms very accurately.

be purchased that identify bacteria that are commonly encountered in routine equine practice.

- The types of bacteria commonly encountered within the practice dictate the choice of bacterial identification systems for use in a clinical situation. Because these systems have a predetermined shelf life, it would not be economically rational to buy a kit that was rarely, if ever, used. We have found that identification kits for the Enterobacteriaceae and for the gram-negative non-Enterobacteriaceae rods are most useful in equine practice. Identification kits for gram-positive rods may also be of value but will depend on how frequently these organisms are encountered within your practice. Systems for the identification of gram-positive cocci often are not required because these organisms usually can be identified quickly, easily, and more cheaply using conventional biochemical testing. Finally, systems for the identification of more unusually encountered bacteria, or bacteria that are more difficult to handle (e.g., gram-negative cocci, enterococci, *Neisseria, Haemophilus* and *Moraxella,* anaerobes, and yeasts), would rarely be required in routine equine practice, and samples suspected of containing these bacteria should be sent to a referral laboratory.
- Considerable care must be taken in the use of these identification systems and the manufacturer's instructions diligently followed. Only a single colony should be used to inoculate media to avoid mixed results associated with a number of different organisms. These mixed results are likely to result in incorrect identification of the organism(s) involved. Some experience with bacterial cultures also is preferred to enable accurate and reliable use of these tests and especially to ensure that aberrant and inconsistent

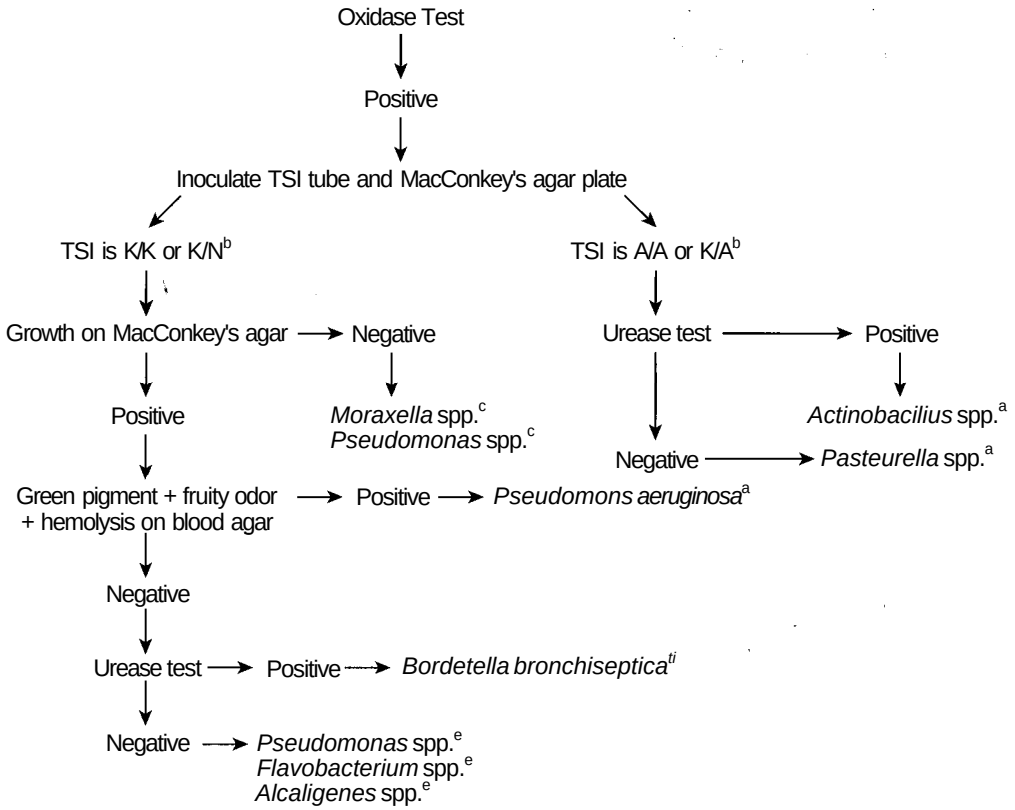


Figure 17-5. Oxidase positive, gram-negative rods.[a]

NOTES:

[a]There are three gram-negative, oxidase-positive rods that are relatively frequently encountered in equine bacterial infections: *Pseudomonas aeruginosa, Pasteurella multocida,* and *Actinobacilius* spp. These organisms may be identified with the commercially available miniaturized systems or by using more conventional methods (see above). The remaining nonfastidious organisms in this group are more rarely encountered, and if they are suspected, the simple chart above may be used or alternatively samples may be submitted to a referral laboratory for isolation and identification (e.g., *Bordetella* spp. and *Moraxella* spp.). Samples thought to contain bacteria that require special media and growth conditions (e.g., *Taylorella equigenitalis. Brucella* spp.) should be sent to a referral laboratory.

[b]Reactions of Triple Sugar Iron Media are described in the Appendix in this chapter.

[c]*Moraxella* spp. (including *M. bovis)* and *Pseudomonas* spp. (except *P. aeruginosa) are* less freqeuntly associated with infections in horses. In addition, care must be taken with interpretation of these isolates as they may be common contaminants, particularly the *Pseudomonas* spp.

[d]*Bordetella bronchiseptica* may cause lower respiratory tract infections in horses.

[e]*Pseudomonas* spp., *Flavobacterium* spp., and *Alcaligenes* spp. are very unusual causes of infection in the horse, and contamination must be suspected if these species are isolated.

reactions can be detected. Commercial media can give incorrect naming of organisms, which may go undetected by inexperienced personnel.

INTERPRETATION OF ISOLATES IDENTIFIED

- Once an organism has been identified, it must not be assumed that this bacterium is necessarily significant. Merely identifying a bacterial species does not make it meaningful.

KEY POINT

After identification of cultured bacteria you must always first ask, Does this organism cause this disease, in this site, in the horse?

If the answer to this question is that you do not know or it has not been reported, then it is likely

TABLE 17-1. Packaged Systems that Identify Bacteria by Biochemical Testing and that Could Be of Use for Identifying Common Equine Pathogens

System Manufacturer	Name	Organisms Identified
Baxter Diagnostics	Neg ID type 2	Enterobacteriaceae, and other fermenting and nonfermenting bacteria
	Pos ID	Gram-positive cocci and *Listeria* spp.
Becton-Dickinson Microbiology Systems (BDMS)	Entero Tube-II	Enterobacteriaceae
	Oxi/Ferm Tube	Nonfermentative gram-negative rods
	Minitek	Anaerobes, Enterobacteriaceae, gram-positive organisms, *Neisseria* spp., nonfermenters, and yeasts
BioMerieux Vitek	API 20 Strep	Streptococci and enterococci
	API Staph	Staphylococci and micrococci
	API Coryne	Corynebacteria
	API 20A	Anaerobes
	API 20C	Yeast
	API 3600S	MIC susceptibility testing
Biolog	GN Microplate	Aerobic gram-negative bacteria
	GP Microplate	Most gram-positive cocci and rods
Difco/Pasco	ID Tri-Panel	Gram-negative and gram-positive bacteria
Flow Laboratories	Uni-N/F Tek	Nonfermentative and other gram-negative bacteria
	Uni-Yeast Tek	Yeast
	Enteric-Tek	Enterobacteriaceae
	Anaerobe-Tek	Anaerobic bacteria
Radiometer America	Sensititre AP 80	Enterobacteriaceae, and nonfermenting gram-negative bacteria
	Sensititre Microbact	Enterobacteriaceae and selected gram-negative bacteria

that the organism isolated is a contaminant and is not contributing to the disease process. If, however, the answer to this question is yes, and especially if you have grown the bacteria in pure culture and in moderate to large numbers, then you can be more confident of the significance of the isolation.

For example, if you isolate and identify *Staphylococcus aureus* from the joint fluid of a horse with clinical signs consistent with septic arthritis, the joint aspirate is consistent with an inflammatory exudate, and the sample had been collected, stored, and processed in an appropriate fashion, then you can be relatively confident that this organism is contributing to this disease process observed. This decision is reached because you would be aware that *S. aureus* has been reported to cause septic arthritis in horse. In this case you may wish to perform a sensitivity test on the *S. aureus* isolated and place the horse on appropriate antimicrobial agents once results of the sensitivity test are obtained. If, on the other hand, you identified a *Micrococcus* spp., you would be far less confident that the organism isolated was significant because *Micrococcus* spp. have a low pathogenicity and are common contaminants. You would review your collection and processing protocols to determine if there was a possibility of contamination and remember that it would take very unusual circumstances to allow this organism to grow, multiply, and cause pathology in this site. In addition, you should not perforin a sensitivity test on this isolate unless you are very sure that these unusual circumstances have been met.

When interpreting results of identification of bacteria, you should also remember that common pathogens "commonly cause disease." Table 17-2 lists the bacteria that are frequently isolated from cases of infection at various body sites in the horse. These organisms may vary in different parts of the world and you should be aware of the organisms found in the area in

TABLE 17-2. Organisms that May Cause Disease at Various Sites in the Horse

Site	Common Isolates	Uncommon Isolates
Lower respiratory tract	*S. equi* ss. *zooepidemicus* *Pasteurella* spp., *Actinobacillus* spp., Obligate anaerobes, Enterobacteriaceae, *Bordetella* spp. *R. equi* (foals)	*S. pneumoniae* (foals) 5. *equi* ss. *equi* (foals) *Salmonella* (foals) *Mycoplasma* *Pseudomonas aeruginosa*
Uterus	*S. equi* ss. *zooepidemicus* *E. coli, Klebsiella pneumoniae* *P. aeruginosa*	*Actinobacillus equuli* *Salmonella abortus equi* *T. equigenitalis* *S. aureus*
Thoracic/peritoneal cavities	Obligate anaerobes, e.g., *Bacteroides* Enterobacteriaceae, e.g., *E. coli* *S. equi* ss. *zooepidemicus* *A. equuli*	*Corynebacterium* spp. *Actinomyces pyogenes*
Abscesses	*S. equi* ss. *zooepidemicus* *C. pseudotuberculosis* Obligate anaerobes *E. coli* *S. aureus*	*P. aeruginosa* *A. pyogenes*
Bone/joints	*S. aureus* *E. coil* *S. equi* ss. *zooepidemicus* *A. equuli* (foals) *Salmonella* (foals)	*Fusobacterium necrophorum* *R. equi* (foals) Other coliforms
Gastrointestinal tract	*Salmonella* *Clostridium perfringens* *Ehrlichia risticii* *R. equi* (foals)	*Clostridium difficile* (foals) *Clostridium cadaveris*
Skin	*S. aureus* *Dermatophilus congolensis*	*C. pseudotuberculosis* *Streptococcus* spp.
Septicemia	*A. equuli* (foals) *E. coli* *Salmonella* (foals)	*Enterobacter* spp. (foals) *Serratia* spp. (foals)
Eye	*S. equi* ss. *zooepidemicus* *S. aureus* *Corynebacterium* spp. *P. aeruginosa*	*Bacillus cereus* *Klebsiella* spp.
Nervous system	—	*Listeria monocytogenes* *S. equi* ss. *zooepidemicus*
Bladder (urine)	*E. coli* *Proteus mirabilis* *Klebsiella* spp. *Staphylococcus* spp.	*Corynebacterium* spp. *Enterobacter* spp.

which you practice. Identification of unusual or bizarre bacteria from samples (or one that is identified by a referral laboratory) must be questioned. You should review the collection and processing of the sample rather than assume the organism is significant and to ask for a sensitivity test to be performed.

- Finally, the task of determining the significance of the bacteria identified will be easier if you also consider:

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- o A knowledge of the normal flora (species and approximate distribution) in various sites
- o A knowledge of the likely pathogens in various sites (see Table 17-2)
- o A realization that the pathogenic organism(s) can originate from outside the horse (e.g., wound contamination), the horse's own normal flora (e.g., fecal contamination of a wound, abscess communicating with the oral cavity), or overgrowth of one species in the gastrointestinal tract due to unwise antibiotic usage.
- o A realization that culture results must be correlated with the clinical signs in the horse and evidence of inflammation in the cytologic preparations collected and examined
- o The use of other laboratory data is also essential and may help in the interpretation of the significance of your isolate.

Cytology. There should be cytologic evidence of inflammation in the original smear of the sample obtained. In acute bacterial infections in horses, the most common cells observed should be neutrophils, together with macrophages and occasional lymphocytes. The presence of phagocytosis of bacteria is an additional clue that the organisms present are likely to be contributing to the disease process. If there is an absence of inflammatory cells in the original smear, it is more likely that there is not a bacterial infection occurring, unless there is a valid reason for their absence (e.g., toxins produced by the bacteria lysed the inflammatory cells in the infected area).

Hematology. Infections *may* be accompanied by a neutrophilia and possibly a leukocytosis. The presence of a left shift may also be noted (although this is less common in horses than in dogs or cats). Alternatively, leukopenia/neutropenia may be observed in horses, especially in the acute stage of severe infections involving gram-negative bacteria and endotoxemia. Toxic changes observed in neutrophils are a strong indication that a bacterial infection (and toxin) is present.

Histopathology. Evidence of inflammation in the tissue sample thought to be infected will confirm the significance of the bacteria isolated. The bacteria (rods or cocci) may also be observed in these sections. Furthermore, to diagnose systemic fungal infection, invasion of tissue must be demonstrated.

Urinalysis. An elevated white cell count in urine may indicate urinary tract infection.

Serology. Evidence of a high antibody titer or rising antibody titer to the organism isolated or identified may help confirm its significance.

ANTIBIOTIC SENSITIVITY TESTING

A number of different types of antimicrobial sensitivity testing are available to the veterinary practitioner. However, there are a few basic principles that are true for all bacterial isolates and all the types of sensitivity testing:

- Attempt to collect a sample for isolation of the causative organism before treatment begins.
- Only perform antibiotic sensitivity tests on an organism that you have isolated and consider pathogenic in the given situation.
- If you believe that more than one organism is significant, test each one separately.

KEY POINT

Some bacteria, which may be recognizable in a Gram stain, by culture, or by subsequent identification, have predictable sensitivity patterns, and susceptibility testing is therefore not necessary. Other bacteria do not have a predictable sensitivity pattern and require sensitivity testing before therapy is instituted.

- Predictable and unpredictable antibiotic sensitivities of bacteria are provided in Table 17-3.

TABLE 17-3. Indications of Bacteria with Predictable and Nonpredictable Antibiotic Sensitivity Patterns

Predictable	Not Predictable
β-Hemolytic *Streptococcus* spp.	*Staphylococcus* spp.
Clostridium spp.	α-Hemolytic *Streptococcus* spp.
Corynebacterium spp.	*Nocardia asteroides*
Most *Bacteroides* spp.	*Bacteroides fragilis*
Erysipelothrix rhusopathiae	Most gram-negative facultatively anaerobic bacteria, e.g., *Pseudomonas* spp., *E. coti, Klebsiella pneumoniae,*
Bacillus anthracis	*Salmonella* spp.,
Pasteurella multocida (in horses)	*Enterobacter* spp.,
Listeria monocytogenes	*Actinobacillus equuli,*
Actinomyces spp.	*Bordetella bronchiseptica*
Dermatophilus congolensis	
Fusobacterium spp.	
All gram-positive strict anaerobes	
All *Rickettsia* (e.g., *Ehrlichia)*	

KEY POINT

Most bacteria isolated commonly from horses that have predictable sensitivities are sensitive to benzylpenicillin, except Rickettsia, *which are sensitive to tetracylines.*

KEY POINT

In life-threatening situations, where therapy must be initiated immediately, use clinical judgment based on experience and likely causes of infection to select an appropriate agent.

- Two things may be of value in assisting your choice of antimicrobial agents in these situations:
  - o A Gram stain, which shows the morphology and Gram reaction of the bacteria involved.
  - o Previously published reports or previously accumulated data from your own practice of the sensitivity patterns of the bacteria frequently involved in the same clinical situation as your current sample.

KEY POINT

Different sensitivity patterns may exist in different areas. Thus, knowledge of sensitivity patterns for bacteria from your practice area is important.

Antimicrobial Sensitivity Tests

The range of sensitivity tests available for use in routine equine practice include the following.

DIRECT ANTIBIOTIC SENSITIVITY TESTING

- Direct antibiotic sensitivity testing is the placement of antibiotic disks onto the surface of a plate that has been seeded over its surface with an original clinical sample. *This procedure is not recommended for use in clinical practice.* The original sample may yield insufficient growth on the sensitivity plate or there may be a mixture of organisms. In either case, a meaningful interpretation of the result is impossible. In addition, if there is a mixture of organisms, it is impossible to interpret the importance of each bacterial species without identification of each type. For example, one of the bacteria present may be a contaminant yet may be resistant to a preferred antimicrobial agent. Thus, the result of the direct sensitivity testing would not provide guidelines as to the antibiotic of choice. Alternatively, one of the bacterial species may be producing antibiotics or bacteriocins that will affect the result of the sensitivity test but may not be contributing to disease.

KEY POINT

Direct *antibiotic sensitivity testing of the original sample is rarely,* if ever, *warranted.*

- It is argued that direct sensitivity testing gives a quick result. However, a quick wrong result is worse than useless. Often, it is possible to achieve an equally rapid result using appropriate techniques.

MODIFIED KIRBY-BAUER DISK DIFFUSION METHOD

- The Kirby-Bauer technique is simple, quick, and gives a much more reliable indication of the best antibiotic to use than uncontrolled sensitivity testing.

KEY POINT

Testing should be done with the standardized technique as outlined in microbiologic manuals. Without a standardized test, it is not possible to determine zone size for "zone size interpretation."

- It should also be remembered that this test is only valid for organisms in log phase at the time of disk application and organisms that grow rapidly (i.e., 16 hours to achieve full colony size, e.g., *Staphylococcus* spp., Enterobacteriaceae, *Pseudomonas* spp.).
- One criticism of this test in veterinary medicine is that interpretation of zone sizes (and hence whether a bacterial isolate is sensitive or resistant) is determined by achievable concentrations of antibiotics within the blood of *humans* (and not domestic species) after a standard dose of antibiotic. However, if the test is performed in a meticulous and standardized manner and if the antibiotics used are bactericidal agents (e.g., penicillins, aminoglycosides), then interpretation of this test appears to be valid for most antibiotics used commonly in equine practice.
- If this test is performed in your practice, strict adherence to the guidelines set out for conducting this assay should be practiced. These guidelines have been recently revised and are available from bacteriologic texts or the National Committee for Clinical Laboratory Standards.
- An alternate disk diffusion technique is the calibrated dichotomous sensitivity (CDS) method. This method differs from other disk diffusion

techniques inasmuch as the amount of drug contained in the disks has been chosen so that sensitivity to most antibiotics is indicated by a uniform zone size of inhibition. Interpretative charts are therefore not required, and the organisms are graded as sensitive if the annular radius of the zone of inhibition is >6 mm and resistant if it is <6 mm. A description of this method can be obtained in Bell (1988).

BROTH DILUTION TESTS (MINIMUM INHIBITORY CONCENTRATIONS)

KEY POINT

This method is used in more accurate work when the pharmacokinetics of an antibiotic is to be determined. It is not usual to use this method for routine sensitivity testing in practice.

- In broth dilution tests, doubling dilutions of antibiotic in broth are inoculated with small numbers of the bacterium being tested, incubated usually for 18 hours, and examined to find the smallest concentration that has completely inhibited macroscopic growth of the test organisms (i.e., turbidity). This concentration of antibiotic is called the *minimum inhibitory concentration* (MIC).

KEY POINT

A variation of this theme is presented in the form of commercially available microliter plates containing antibiotics at concentrations that should cover the range of sensitivities of most of the organisms encountered in veterinary practice.

- The organisms to be tested are grown on agar plates, picked off, and emulsified in a broth culture to a specific concentration. Drops of the organism are placed into the wells and incubated along with the appropriate controls. With this assay, it is possible to determine the MIC of the organism under test in as few as 12 hours.
- This test may be of value if the MIC can be interpreted in light of the dose schedules given to horses, which is a major limitation of the current disk test system. However, these tests are more difficult to perform and are infrequently used in clinical practice.

AGAR DILUTION TESTS

KEY POINT

Agar dilution tests are the most practical form of test when moderate to large volumes of work are to be undertaken.

- They are used more commonly in larger laboratories to obtain the same accurate information as the broth dilution tests yet without the consumption of large amounts of time and materials.
- Agar dilution tests also may be used when it is desired to determine the MICs of a number of strains of bacteria, using doubling dilutions of antibiotic over a range, as in broth dilution tests.

Appendix

SMEAR PREPARATIONS

Thin Smears

- To make a thin smear, first label a slide with a diamond pen to determine which side of the slide has been stained and to mark the center of the smear area. Then make a thin smear of your sample by either
  1. Rolling a swab onto the surface of the slide if the sample is presented on a swab.
  2. Placing a small loopful of pus/fluid onto the slide and spreading with the wire loop into a thin smear.
  3. Solid material may be emulsified in saline or broth and smeared as in 2.
  4. Impression smears may be made from solid tissue.
- Allow smears to dry thoroughly in air thereby allowing the protein in body fluid to "fix" the cells and bacteria to the slide.

STAINS

Gram Stains and KOH Test

- The Gram stain is used to differentiate between gram-positive and gram-negative bacteria.

KEY POINT

For best results when staining smears from body tissues and fluids, the Burke's modification of the Gram stain should be used (i.e., Burke's Gram stain). When staining organisms from cultures (colonies from agar plates or broth cultures), a Hucker 's Gram stain may be used.

- Occasionally, questionable results are obtained after performing a Gram stain on cultured bacteria. In these cases, the Gram reaction may be verified by the KOH test, which correlates with the Gram reaction.

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BURKE'S GRAM STAIN

1. Cover the slide with Burke's crystal violet, add three drops of Burke's solution B (sodium bicarbonate buffer), and wait 3 minutes.
2. Wash off the crystal violet with tap water, cover the slide with Burke's iodine, and wait 3 minutes.
3. Wash off the iodine with tap water.
4. Hold the slide between the thumb and forefinger and flood the surface with a few drops of ether-acetone decolorizer until no violet color washes off. This usually requires about 10 seconds or less.
5. Cover the slide with safranin counterstain and wait for 1 minute. Wash the slide with tap water.
6. Place the smear in an upright position, allow the excess water to drain off, and let the smear dry or, preferably, blot dry.
7. Examine the stained smear under 100x (oil) immersion objective of the microscope.

Results

- Gram-positive bacteria stain—dark blue/purple
- Gram-negative bacteria stain—pink/red

Record the relative numbers and morphology of each bacterial type observed. Although you may note numbers and types of inflammatory and other cells (e.g., squames) present, these cells will be much easier to identify and enumerate using a smear stained with a cytologic stain (e.g., Diff Quik).

HUCKER'S GRAM STAIN

1. Cover slide with Hucker's crystal violet and leave for 1 minute.
2. Wash in tap water by holding under running tap (faucet). Shake off surplus water.
3. Flood slide with Gram's iodine and leave 1 minute.
4. Wash slide in tap water and allow surplus water to be drained off.
5. Decolorize in 95% ethanol (ethyl alcohol). Hold the slide at an angle of about 45 degrees and drip on alcohol, letting it run over the film until no more blue comes out. Run ethanol down both sides of the slide to ensure all blue is removed. Do this slowly over a period of 1 to 2 minutes or as required to allow adequate penetration of the smear. Ethanol has poor penetration powers and thick smears or thick sections of smears may not decolorize fully with ethanol no matter how long decolorizing is performed.
6. Counterstain slide with safranin solution for 10 seconds.
7. Wash in tap water.
8. Blot dry and examine slide.

Results

- Gram-positive organisms—dark blue
- Gram-negative organisms—orange-red

KOH TEST

1. Transfer two loopfuls of 3% KOH to a slide.
2. With a loop, pick up a young (24 hours growth) colony of the organism to be tested and mix with the KOH on the slide.
3. Observe for viscosity by drawing the loop slowly out of the mixture.

Results

- Gram-negative bacteria—If the mixture becomes markedly viscid or gels within 5 to 60 seconds. The viscosity is due to the release of DNA from the thinner walled gram-negative bacteria.
- Gram-positive bacteria—The mixture will not become viscid, and the bacteria will mix smoothly into solution.

Diff Quik Stain

Diff Quik is a quick and easy-to-use stain that yields smear preparations suitable for cytologic evaluation. It is important to regularly renew the stains used in practice or the staining quality becomes poor and there will be increased amounts of sediment that make evaluation of smears for bacteria more difficult.

1. Prepare a smear preparation from the sample as described above.
2. Immerse the slide in the fixative for at least 1 minute (the slide may be left in the fixative for longer periods of time but should not be fixed for any shorter period).
3. Immerse the slide in solution 1 (red-colored solution) for approximately six 1-seconds dips.
4. Immerse the slide in solution 2 (purple-colored solution) for approximately six 1-second dips.
5. Wash the slide thoroughly in tap water and dry rapidly (e.g., with a hair dryer).
6. Cytologic examination of slides usually involves evaluation of fields using the 10 X, possibly the 20 X , and the 40 X objectives. If the slide is to be examined using the 40 X objective (high dry), first place a drop of oil on the section to be examined, then place a coverslip over the oil and allow the oil to disperse under the coverslip. This will greatly improve the clarity of the field to be

examined. However, it must be remembered that bacteria are usually not readily visible (except for large rods, e.g., *Clostridium* spp.) unless the oil objective (100x) is used. A drop of oil may be placed directly on top of the coverslip and the section examined for the presence of bacteria if their presence is suspected.

BIOCHEMICAL TESTS

There are a few simple biochemical tests that can be easily performed and will aid in the identification of bacteria commonly encountered in equine practice. These biochemical tests *should be performed on young (24-hour) cultures* to ensure optimal biochemical reactions. Media or reagents for most of these tests are available commercially and would be purchased if the turnover of these reagents in your practice warranted their use. The tests purchased from commercial companies will usually be accompanied by instructions, but a simple set of instructions is outlined below. The use of positive and negative controls for each test is recommended. Organisms used as controls should be rapidly growing, nonfastidious, and maintain viability and biochemical activity for prolonged periods. These organisms may be subcultured onto blood agar plates, incubated overnight at 37°C, then stored in the refrigerator for up to 1 month. The organism most frequently used as controls include *S. aureus, P. aeruginosa,* and *E. coli.*

Catalase Test

- This test detects the presence of catalase enzyme activity. You should note that red blood cells may give a positive test; therefore, care should be exercised when testing colonies from a blood agar plate.
  - o Place a loopful of 3% hydrogen peroxide (H_2O_2) onto a clean glass slide.
  - o Using your loop, select a single colony of bacteria to be tested and mix the colony in with the loopful of H_2O_2.
- Positive test—Bubbles will be liberated immediately (e.g., *Staphylococcus* spp.).
- Negative test—There are no bubbles, and the colony mixes smoothly into solution (e.g., *Streptococcus* spp.).

Coagulase Test

- The enzyme coagulase is produced by some *Staphylococcus* spp. and will cause plasma to clot. There are two types of coagulase: bound and free. Bound coagulase is detected by the *slide coagulase test,* whereas both bound and free coagulase are detected by *tube coagulase test.* We routinely perform the slide coagulase test first and if these results are negative will then perform the tube coagulase test.

SLIDE COAGULASE TEST

1. Mix two to three colonies in a loopful of water to form a uniformly thick smooth emulsion.
2. Add one loopful of undiluted sterile rabbit plasma.

- Positive test—Immediate clumping or formation of white precipitate (e.g., *S. aureus)*
- Negative test—Nil or delayed clumping (e.g., *S. epidermidis, S. saprophyticus, Micrococcus* spp.).

TUBE COAGULASE TEST

1. Emulsify two colonies in 0.5 mL of undiluted sterile plasma in a small tube.
2. Incubate at 37°C.

- Positive test—A solid gel is formed, generally within 2 to 4 hours, and persists.
- Negative test—No gel formation.

Oxidase Test

- This reaction depends on the presence of ion-containing enzymes, oxidases, within cells.
  - o Place an oxidase strip (filter paper impregnated with tetramethyl-*p*-phenylene diamine HCl) onto a microscope slide and moisten with tap water.
  - o A colony of the test organism is rubbed onto the moistened oxidase strip.
  - o Note length of time for the color change to develop and intensity of the color achieved.
  - o On the same test strip you should also test a known positive control (usually *P. aeruginosa)* and a known negative control (usually *E. coli)* and interpret the results with reference to the controls.
- Positive test—*Strong,* a deep purple color develops within 5 seconds (e.g. *Pseudomonas* spp.); *Weak,* a weak purple/pink color develops within 4 minutes (e.g. *Pasteurella* spp., *Actinobacillus* spp.).
- Negative test—No change or change consistent with control negative organisms rubbed onto the same paper (e.g., members of the *Enterobacteriaceae).*

Sugar Fermentation Tests

- Acid or acid and gas production in a basal medium of 1% peptone measures metabolism of

carbohydrates (sugars). To this medium is added 1 % of the required carbohydrate, a dye indicator for acid, and an inverted glass tube (Durham tube) to indicate if gas is produced. In addition, *Streptococcus* spp. will require addition of 10% horse serum to the basal medium for growth. These tests are usually conducted in small glass bottles, but the medium may also be added to an agar base, and plates of the sugars to be tested poured.

- Sugar fermentation tests are most commonly used in equine practice for the identification of β-hemolytic *Streptococcus* spp. The sugars most commonly tested are trehalose, sorbitol, and lactose.
  - o Add several colonies of test organism to a liquid culture medium that will support the growth of *Streptococcus* spp. Incubate until the medium is slightly turbid, then use two to three drops of this culture to inoculate the tubes of prepared medium with added sugars (these tubes may be bought from a commercial company or made within your practice).
  - o Incubate overnight at 37°C.
- Positive test—Yellow color (if Phenol Red is indicator that has been incorporated into the medium)
- Negative test—Red color (if Phenol Red is the indicator that has been incorporated into the medium)

Urease Test

- The urease test detects the splitting of urea by ureases produced by bacteria. The splitting of urea results in ammonia formation and increase in pH.
  - ° Inoculate a Christensens urea slope.
  - o Incubate overnight at 37°C.
- Positive test—Bright pink (e.g., *Proteus* spp., *Actinobacillus* spp.).
- Negative Test—No color changes from basal medium—pale orange (e.g., *Salmonella, Pasteurella* spp.).

Triple Sugar Iron Test

- The triple sugar iron (TSI) test is used to detect the ability of an organism to ferment specific carbohydrates that are incorporated into a basal growth medium, with or without the production of gas and with or without the production of hydrogen sulfide. For the flow charts used in this chapter, the fermentation of sugars with the production of acid and the utilization of peptones with the production of alkali are the key biochemical changes noted.

1. Pick up a single colony with a straight inoculating wire and inoculate the slanted tube medium by first "fish-tailing" the slant, then stabbing the butt with the straight wire.

2. Incubate the tubes for 18 to 24 hours *(no earlier and no later)* at 37°C.

3. For interpretation of color reactions, the tubes should always be compared with uninoculated media. In TSI tubes, alkaline reactions cause formation of a red color, and acid production will be a yellow color. If there is no change in the medium it will be the same color as uninoculated medium. Symbols used for interpretation of TSIs include

K/K = alkaline slant/alkaline butt

K/N = alkaline slant/no change in butt

K/A = alkaline slant/acid butt

A/A = acid slant/acid butt

where K/K or K/N is *Pseudomonas aeruginosa, Pseudomonas* spp., *Moraxella* spp., *Bordetella* spp., etc. and K/A or A/A is *Pasteurella* spp., *Actinobacillus* spp.

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CHAPTER 18

Clinical Nutrition

John R. Kohnke

- Horses must be provided with an adequate supply of carbohydrates, protein, fats, vitamins, minerals, and electrolytes, balanced with fiber for digestive function, and must have access to clean fresh water at all times.
- Horses are able to adapt to a large variety of grains, protein meals, and hay, although they are generally very selective in plant preferences and grazing patterns when given access to pasture.

KEY POINT

The ration provided must meet a horse's specific work or purpose needs; must be economical, palatable, well accepted, and utilized; and maintain the animal in good health and suitable condition relative to its age or performance requirements.

- The selection and blend of feeds become most important when horses are confined to stalls, dry lots, or yards and have little free choice or are provided with bulky, less palatable feeds that reduce their ability to consume sufficient amounts to meet daily exercise or other needs.
- There is often a *limit to the horse "feeding budget,"* and not all owners are able to purchase, or select, the best-quality feed, particularly when seasonal conditions influence the *availability, quality,* and *cost of feed.*
- Many horses are overfed relative to their growth and exercise demand. Equine practitioners are often required to evaluate and provide sound advice on the adequacy and feeding management of rations.
- The National Research Council (NRC) and other specialized groups provide scientific guidelines for nutrient intake for all types of horses relative to their age, body weight, exercise, growth, and reproduction demand.
- The dietary guidelines provided for each class of horse in this chapter are formulated to meet the needs of an average horse maturing to or maintaining a body weight of 450 kg (1000 lb) based on NRC (1989) recommendations. Other horses should be fed in proportion to their body weight.

KEY POINT

To avoid variations in nutrient supply caused by differing quality of feedstuff's, feeds should be measured by weight rather than volume. Most horse owners relate intake to volume of feed measured out, and the ration guidelines provided include both weight and volume measurements of average-quality feeds. Volume measures are given in liters (1 L = 1000 mL 2 pints) or US pints (1 pint 500 mL = 16 fluid oz; 1 cup 250 mL [½ pint = 8 oz.]).

PRACTICAL FEEDING RULES AND NUTRITIONAL MANAGEMENT

These are summarized in Tables 18-1 and 18-2. A degree of common sense applies to feeding horses. Certain rules for feeding should be strictly followed, especially when providing concentrate rations with limited or no access to pasture. Proper feeding management will ensure that a horse will stay in sound health and receive maximum benefit from its ration. Horses are always hungry. Certain "do's" and "don'ts" should be observed when feeding them to avoid digestive upset and other metabolic conditions (see Tables 18-1 and 18-2).

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TABLE 18-1. Practical Feeding Rules: The Do's

Always Ensure That:	Practical Guidelines
1. The diet should contain a balance between roughages (hay, cubes, pasture) and concentrates (grains, protein meals, fats, etc.) relative to the horse's requirements.	Always *measure feed by weight* rather than volume to maintain a uniform nutrient intake. All rations should be based on quality roughage (pasture, long-stem hay, cubes, chopped hay), with grains and meals added in direct proportion to exercise, reproduction, or growth needs. Minimum roughage intake is 1 kg/100 kg (1 lb/100 lb) or 1% of body weight to maintain digestive function and store water in the large intestine.
2. The ration should be modified to suit individual horses and must be palatable and economical and supply the horse's requirements.	Feed to maintain a suitable body condition score. Feed each horse as an individual to cater for likes and dislikes. Change the ingredient blend to meet appetite limits and acceptance. Rations need not be complicated mixtures. Substitute ingredients if necessary. Lonely unfed horses are more likely to develop chewing vices, weaving, stall walking, etc. All horses should have an opportunity to exercise in a paddock or be exercised each day to gain the best benefit from their rations.
3. Meals should be fed at regular times and at least twice daily to confined horses or those with limited access to grazing.	Horses are creatures of habit and come to expect to be fed at the same time every day. Racing horses in training should be fed at least four times a day. Stabled horses in work should be fed at approximately equal intervals to ensure a continuous digestion pattern. Slow eaters should be fed little and often, and "nervy" horses allowed adequate undisturbed time to eat. Provide most of the bulk overnight as hay for stabled horses to keep them occupied and relieve boredom.
4. The feed should be regularly evaluated for quality.	Provide the best-quality feed available. If feed is measured by volume, weigh new batches of grain occasionally and adjust volume to ensure a more constant intake of nutrients. Dampen dusty hay or feeds to improve utilization and minimize dust and wastage.
5. The ration should be well mixed.	Mix ingredients carefully to prevent a horse selecting only the feed it likes, especially in high-grain rations. Ensure that there are no lumps of minerals. Dampen with water or a molasses and water mix to reduce dust and sifting out of supplements.
6. The ration should be freshly mixed for each feed.	Dry mixes can be stored for 1-2 days, but do not store dampened feed for more than 1 hour. Remove leftovers before each new feed is given.
7. The ration should also be complemented by good husbandry.	Give careful attention to general health, teeth care, regular worming, and daily exercise. Check the amount, color, odor, and consistency of the feces to monitor digestive function and relative hydration state.
8. The ration should include adequate clean water at all times.	Horses should have free access to water during hot weather and periods of hard work and when electrolytes are added to the ration. Check water flow; clean troughs/bowls regularly.

TABLE 18-2. Practical Feeding Rules: The Don'ts

Take Care To:	Practical Guidelines
1. Limit sudden changes in ration proportions or ingredients.	Introduce new feeds over 4-5 days or major changes over 7-10 days or longer. Do not change feeds within a few days of an important race, competition, or show.
2. Avoid sudden increases in grain content or too rapid introduction to highly concentrated rations.	Always keep the work level ahead of the feed. Increase grain gradually in proportion to the amount of work performed rather than planned for each day.
3. Do not feed dusty, moldy, or contaminated ingredients, spoiled "leftover" feed, or poor quality feeds.	Dusty and moldy feeds can cause digestive and respiratory problems. Dampen before feeding. *Do not* feed moldy feeds, feed containing mice or rat droppings, or feed mixes made for other animals with growth promotants. Regularly clean feed tubs and water bowls and troughs. Clean out any damp feed a horse does not eat each day. Poor-quality roughage (hay) will be wasted and lead to excessive digestive bulking ("hay belly") or compaction. Do not disguise poor-quality feed with sweeteners, such as molasses or yucca.
4. Restrict the grain intake on planned rest or other idle days.	Reduce grain or concentrate feeds to *one third* on the night before planned rest days—replace with hay or cubes or chopped hay (chaff). If a horse is not worked on a day because of lameness, sickness, or wet weather etc., reduce grain in the next feed. Reintroduce grain gradually—/or *one day off work, take two days to return to full grain intake* so as to avoid "tying up" in working horses, particularly in nervy young fillies.
5. Do not allow horses to gorge concentrate feeds or allow ponies unlimited grazing on lush spring pasture.	Greedy horses are likely to choke or develop digestive upsets. The maximum intake of grain should be limited to 0.5 kg (1 lb) per 100 kg (220 lb) body weight to avoid risk of starch overload and hindgut acidosis in all working horses. Carefully mix grains with chopped hay or cubes. Avoid free access to concentrates during cold weather. Restrict pasture access to ponies and horses prone to laminitis, and do not allow them to graze overnight.
6. Do not feed from dirty feeders or waterers or feed concentrates or hay on the ground.	Regularly clean out feeders to avoid caking of residues—separate water troughs and feed tubs to reduce grain build-up in troughs and eat-drink feeding habits. Feed on the ground is wasted and often contaminated with sand and parasite eggs and larvae. Provide an adequate-sized feed tub with safe edges and enough weight to prevent a horse from tipping it over.
7. Control the intake of hay just before working a horse and avoid working a horse on a full stomach.	A large feed just before working can cause discomfort because of a distended gut. A small feed containing 60% grain and 40% hay or cubes is less bulky. Feed most of the long-stem hay overnight. A dampened feed after exercise helps to encourage appetite and provide moisture to replenish fluid levels.
8. Limit access to large volumes of cold water after strenuous exercise.	It is unwise to allow a hot, sweaty horse to drink large quantities of cold water immediately after exercise. Allow a few swallows of water initially, then more water in 10 minutes. Take the chill off very cold water by mixing in a small quantity of hot water, before offering it to a horse at the completion of strenuous exercise.

TABLE 18-3. Feed Intake and Optimum Roughage-to-Grain Ratios

Type and Duration of Exercise, Classes of Horses, Reproduction and Growth Needs	Total Daily Feed Intake to Meet Energy Requirements. As Fed Hay and Grains (90% Dry Matter)	Balance of Roughage and Grain/Concentrate Required for Different Activities	
		Approx. % Roughage by Weight—Long-Stem Hay, Roughage Cubes, Chopped Hay (chaff) Haylage, Wheat Bran	Approx. % Grain/ Concentrate by Weight—Grains, Protein Meals, Fats (Oils), Raw or Cooked (extruded) Grains
Idle/resting horses			
• Maintenance diet • Early pregnancy • Stallions off season	1.5-2.0% body weight 1.5-2 kg/100 kg (1.5-2 lb/ 100 lb)	80-100%	0-20% maximum
Light work			
30-60 min daily walking, trotting, some cantering • Pleasure, basic dressage, show horses, working ponies • Mares, late pregnancy: 9-11 months • Early race training • Stallions at stud • Horses 18-30 months of age	2.0% body weight 2 kg/100 kg (2 lb/100 lb)	65-75% (average 70%) Good pasture with hay to maintain body weight	25-35% (average 30%) Feed in proportion to exercise, reduce grain to one-third on idle/rest days, replace weight with hay. Nervy horses—feed minimum grain, or extruded feeds or 5% fat for energy.
Moderate work			
60-120 minutes walking, trotting, canter, some galloping • Advanced dressage, jumping, hunters, polocrosse, one-day horse trials (eventers) • Late pregnancy (bulk of intake limited—provide maximum concentrate intake) • Late lactation after 3 months • Yearling 12 months	2.5% body weight 2.5 kg/100 kg (2.5 lb/100 lb)	45-55% (average 50%) Roughage bulk may limit intake to meet energy needs in small-framed horses.	45-55% (average 50%) Feed in proportion to exercise, reduce grain to one-third on idle/rest days, replace weight with hay.
Intense (heavy) work			
30-60 minutes sustained cantering, galloping, or intense stressful exercise • Advanced race training, polo, upper level horse trials (eventers) • Endurance riding • Early lactation 0-3 months—may be able to consume up to 3% of body weight, provide extra roughage • Weanlings	2.5-3.0% body weight 2.5-3 kg/100 kg (2.5-3 lb/100 lb) Horses in heavy work may not be able to consume more than 2.5% body weight of dry feed.	35-45% (average 40%) Maintain at least 50-60% roughage in weanlings and lactating mares as bulk can be normally consumed.	55-65% (average 60%) Racing horses up to 60% grain—limit bulk by substituting high-energy-density grains (corn, barley, sorghum) with 5% fat. Up to 60% grain as oats, corn or barley mix. If low-fiber grains such as corn replace more than 50% of oats in a ration, limit grainrroughage to 50:50 ratio by weight. Feed in proportion to exercise, reduce grain to one-third on idle/rest days, replace weight with hay.

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PRACTICAL GUIDELINES FOR DAILY INTAKE

Many owners seek advice on how much a horse should be able to consume and what proportion of concentrate or grain mix to roughage is suitable to meet a horse's needs. The guidelines in Table 18-3 are provided for horses with limited access to pasture or when pasture is low quality, short, or sparse. Roughage, as hay or pasture, must form the basis of any diet, with concentrates provided relevant to exercise or other needs. Horses in training or those confined to stalls require a blend of hay and concentrate to provide the major nutrient intake in an adequate bulk they can consume. A grazing horse on green pasture needs to consume approximately 5 to 7% body weight, or 5 to 7 kg/100 kg (5-7 lb/100 lb), each day to maintain itself.

KEY POINT

A horse stabled and lightly worked under conditions of moderate temperature (60—75°F, or 15-25°C) requires water at a weight approximately three times that of dry food consumed each day and up to six times more water than weight of feed consumed under hot conditions or when there is prolonged regular exercise.

Condition Scoring

- Condition scoring is a practical system used to evaluate body condition and subcutaneous fat distribution in show, working, and breeding horses.

KEY POINT

Condition scoring provides a standardized and repeatable method of monitoring variation in condition and can be used to adjust feed intake and exercise to maintain optimum condition of a horse relative to its use or purpose.

A horse's condition can be scored from very poor to extremely fat or obese by visual appraisal and palpation of the prominence of bones and fat distribution over the neck, shoulders, ribs, back, loins, hindquarters, and tail head areas.

The American system ranges from 1 (very poor) to 9 (extremely fat) condition score, and the Australian evaluation method is based on a 0 to 5 condition score. These two scoring systems have been combined in Table 18-4 to provide a universally accepted evaluation system for condition scoring.

KEY POINT

A large horse may have a 10% change in body weight before it will show a visible change in condition score.

- Guidelines for optimum condition scores for various classes and purposes of horses are provided in Table 18-5, including comments related to the standard of appearance required for each type of horse. Adjustment to the ration and exercise program may be necessary to maintain condition within the optimum score range for a particular class of horse. As a veterinarian, you may be required to condition score a horse relative to an optimum score, in a case in which the owner is being prosecuted for neglect in feeding and care of an animal.
- Score each area of the body and then add the scores and average them to obtain the relative condition score for an individual horse.
- In the ration guidelines, the optimum condition score is included for each class of horse to equate condition to accepted standards.

FURTHER READING

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Growing Horses

In an attempt to produce well-grown and well-developed young horses, breeders often become overzealous in trying to achieve maximum growth in the formative years. Limb abnormalities associated with developmental orthopedic disease (DOD) have been linked to overfeeding, vitamin and mineral imbalances, and lack of adequate exercise. These are widely considered as the underlying factors that lead to skeletal weakness and

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TABLE 18-4. Evaluation of Condition Score

Condition Score (Visual Condition)					
USA	Australian	**Neck**	**Withers and Shoulders**	**Ribs, Back, Loin**	**Pelvis, Rump/Croup/ Tailhead**
1 Very poor	0 (Very Poor)	Individual bone structure visible Marked "ewe neck" narrow and slack at base	Bone structure very visible. Spinous process sharp and pointed. No fat.	Ribs very visible and skin furrows between ribs. Spinous processes visible and pointed. No fat.	Tailhead and hip bones very visible, angular pelvis, skin tight. Deep cavity under tail and each side of croup.
			Animal extremely emaciated, no fatty tissue can be felt.		
2 Very thin	0.5	Bones just visible "Ewe neck"	Withers obvious, very minimal fat covering shoulder. Bone structure outlined	Ribs easily seen. Slight depression between ribs.	Tailhead and hip bone obvious. Deep depression under tail.
3 Thin	1 (Poor)	Thin, flat muscle covering	Withers, accentuated shoulder bones, and spinous processes well defined, some fat cover	Rib outline obvious. Flat spinous processes not felt. Fat build-up on vertical spines.	Tailhead prominent, hip bones rounded but visible. Rump sunken but skin supple, deep depression under tail. Pin bones covered.
4 Moderately thin	1.5	Neck covered with fat, not obviously thin	Withers and shoulder not obviously thin, smooth edges	Ribs faint outline. Slight ridge along back.	Fat can be felt. Rump flat either side of backbone.

5 Moderate	2 (Moderate)	Narrow but firm, blends smoothly into body	Withers rounded over top, spinous processes just felt.	Ribs just visible. Back level and backbone well covered.	Fat around tailhead, beginning to feel spongy. Croup well defined, some fat. Slight cavity under tail.
6 Moderately fleshy	2.5	Some fat can be felt.	Fat layer can be felt over shoulder.	Thin layer of even fat over ribs—slightly spongy crease	Fat around tailhead feels soft, can be seen easily.
7 Fleshy	3 (Good)	No crest—visible fat deposits along neck.	Fat build-up behind shoulder.	Ribs covered—individual ribs can still be felt. Spinous processes can be felt. Midline crease but no gutter along back.	Fat around tail obvious, soft and rounded off.
8 Fat	4 (Fat)	Noticeable thickening of neck. Slight crest, wide and firm.	Area along withers filled with fat. Area behind shoulder filled in with fat.	Ribs well covered, need firm pressure to feel. Crease down back evident. Gutter to base of tail. Pelvis covered with soft fat—felt only with firm pressure.	Tailhead fat, very soft and flabby. Fat deposited on inner buttocks.
9 Extremely fat	5 (Very fat)	Marked crest—bulging fat. Very wide and firm.	Ribs—patchy fat and buried under layer of fat. Shoulder bulging fat.	Deep midline gutter along back—broad and flat. Flank filled in flush.	Deep gutter to base of tail. Skin tight and distended. Pelvis buried, cannot feel. Fat along inner buttocks may rub together.

Data from Henneke, D. R., Potter, G.D., Kreider, J.L., and Yeates, B.F.: Relationship between condition score, physical measurement, and body fat percentage in mares. *Equine Vet .1* 15:371-372, 1983; Carroll, C.L., Huntington, P.J.: Body condition scoring and weight estimation of horses. *Equine Vet J* 20:41-45, 1988.

TABLE 18-5. Optimum Condition Scores for Various Classes of Horses

	Optimum Condition Score	
Class of Horse	Condition Score Range	Practical Guidelines
Breeding horses		
Nonpregnant (dry) mares for breeding	USA 4-5 increasing Aust 1.5-2 increasing	Do not starve initially. Increase energy intake during last 6 weeks before breeding. Maintain fat mares in fat condition.
Pregnant mares (midterm)	USA 5-6 Aust 2-2.5	Maintain in a trim to moderate condition.
Pregnant mares last 3 months	USA 6-7 Aust 2.5-3	Ensure some reserves; maintain in a moderate to good condition.
Lactating mares	USA 6-7 Aust 2.5-3	Maintain in good condition. Avoid weight loss, as fertility will be adversely affected.
Stallions (breeding)	USA 6-7 Aust 2.5-3	Maintain in moderate to good condition. Avoid excessive exercise
Stallions (off season)	USA 5-6 Aust 2-2.5	Maintain in moderate condition. Avoid excess condition.
Equestrian horses		
Dressage	USA 6-7 Aust 2.5-3	Muscle bulk with rounded appearance relative to breed and build, in moderate to good condition.
Endurance	USA 3-4 Aust 1.5-2	Some reserve, trim and fit appearance.
Eventing (horse trials)	USA 4-5 Aust 1.5-2	Require fitness, muscle bulk, and moderate condition for all phases.
Showjumpers	USA 5-6 Aust 2-2.5	Fit, some reserve, not thin.
Hunters	USA 5-6 Aust 2-2.5	Not fat, moderate with some reserve.
Polo	USA 3-4 Aust 1.5-2	Some reserve, but not thin.
Polocrosse	USA 4-5 Aust 1.5-2.5	Maintain in trim to moderate condition.
Show horses ridden Led in classes Western pleasure	USA 6-7 Aust 2.5-3	Well rounded and covered, moderate to good but not excessively fat.
Working western horses (rodeo, cutting, etc.)	USA 5-6 Aust 2-2.5	Maintain in a trim to moderate fit condition.
Leisure/pleasure horses		
Horses and ponies ridden on weekends	USA 5-7 Aust 2-3	Moderate to good condition. Avoid heavy condition—feed to exercise level.
Ponies—grazing spring pasture	USA 5-6 Aust 2-2.5	Avoid cresty neck, maintain moderate condition by restricted grazing and exercise to avoid laminitis.
Resting horses	USA 5-7 Aust 2-3	Monitor regularly—feed accordingly, especially aged horses.

TABLE 18-5. Optimum Condition Scores for Various Classes of Horses *Continued*

Class of Horse	Optimum Condition Score: Condition Score Range	Optimum Condition Score: Practical Guidelines
Racing horses—racetraining		
Arabians	USA 4-6 Aust 1.5-2.5	Maintain fit, trim to moderate appearance.
Appaloosas	USA 4-6 Aust 1.5-2.5	Some bulk, maintain in moderate condition.
Quarter horses	USA 5-7 Aust 2-3	Muscle bulk, some reserve, moderate condition.
Standardbreds	USA 4-6 Aust 1.5-2.5	Trim to moderate—not too thin but minimum gut weight.
Thoroughbreds	USA 4-6 Aust 1.5-2.5	Should have some reserve, sprinters bulkier than longer distance horses.
Growing horses		
Weanling 6-12 mo	USA 5-6 Aust 2-2.5	No lower than moderately thin—last few ribs outlined. Avoid overcondition in a growing horse.
Yearling 12-18 mo	USA 5-6 Aust 2-2.5	Yearling can have more reserves in lead up to sale.

Data from Kohnke, J.R.: *Feeding and Nutrition of Horses: The Making of a Champion.* Sydney. Australia. Vetsearch International, 1998.

breakdown in racing and equestrian horses. More emphasis is now placed on achieving an optimum rather than maximum rate of growth, maintained by a combination of adequate and balanced nutrition, feeding management, regular observation, and daily opportunity for exercise.

NEWBORN FOALS

All foals need careful feeding and management, particularly if they are orphaned at or soon after birth. Guidelines on feeding and raising an orphan foal are commonly sought from veterinarians. It is most important that all newborn foals receive sufficient colostrum to provide immunoglobin defense against environmental pathogens. Foals are most susceptible to gut and other pathogens at the low point in immunity, which occurs at 5 to 7 weeks of age (see Failure of Passive Transfer, Chapter 9).

KEY POINT

A newly foaled mare produces colostrum only for the first 2 to 3 days of lactation, and the highest uptake of immunoglobulins IgG and IgM occurs during the first 6 to 12 hours of a foal's life. Gut-protective IgA levels are secreted in milk for 2 to 3 weeks after foaling.

For orphan, weak, sick, or colostrum-deficient nursing foals, extra colostrum can be collected within 12 hours of foaling, preferably from mares that have a dead foal or have lost their foals soon after birth or from newly foaled mares that are productive milkers. Up to 250 mL (1 cupful, or ½ pint) can be taken from a newly foaled mare at hourly intervals immediately after her own foal has nursed. This volume and frequency of colostrum removal will not significantly drain or affect the quality of colostrum available to her own foal at its next drink during its first 6 hours of life. If mare colostrum is not readily available, colostrum from a newly calved cow will provide protective IgG to an orphan foal, but mare colostrum is preferred if available.

KEY POINT

Foals orphaned at birth should receive approximately 500 mL (1 pint) per 50 kg (110 lb) of body weight of high-quality colostrum each hour by nasogastric tube, as many times as possible within 12 hours of birth, to establish protective immunoglobulin levels.

Up to 1 L (2 pints) per 50 kg (110 lb) body weight of colostrum may be given via tube at hourly intervals, but it is important to check for gastric reflux before administering each feed. If gastric reflux is present, then delay the feed for 30 to 60 minutes. A foal's gut is unable to absorb antibodies approximately 12 to 16 hours after birth.

KEY POINT

Colostrum given by nasogastric tube at regular intervals has been shown to establish more rapid and higher uptake of immunoglobulins than can be achieved by bottle feeding.

A check of immunoglobulin levels can be carried out just before the next scheduled feed once a minimum of 3 L (6 pints) has been administered (see Failure of Passive Transfer, Chapter 9).

Note: In foals less than 16 hours of age, *do not* feed milk or water until colostrum is given, because the immunoglobulin transfer cells in the gut lining will cease antibody uptake.

Orphan Foals

FOSTER MOTHERING

In newly born orphan foals, foster mothering is usually successful, provided a suitable nurse mare is available. Larger breeds, such as draft mares and their crossbreds, are popular as nurse mares because they are quiet and maternal and produce more milk than light horse breed mares. However, excess milk intake from 2 to 5 months of age in a foal may increase the risk of DOD.

KEY POINT

Regular monitoring of growth rate and an opportunity for adequate exercise is paramount to avoid limb abnormalities in foals fostered to a heavy breed mare.

Most mares that have a foal themselves will accept a newly orphaned young foal within 2 to 3 days, although some may take up to 7 days to fully adopt a foal. Where a foster mother, a nurse mare, or even a milking goat elevated on a raised platform to enable a foal to suck is not available, the foal will need to be hand reared.

DIETARY GUIDELINES: FIRST WEEK OF AGE

Specialized commercial foal milk replacers are available that are convenient and provide adequate nutrition with low risk of digestive upset when fed as directed. Bottle feeds can be given for the first 2 to 3 days using a commercial milk replacer formulated for foals. Most foals can be taught to drink from a bucket by 3 days of age.

PREPARATION OF MILK

If a milk replacer is unavailable, then *low-fat whole fresh milk,* fat-reduced powdered milk, or a calf skim milk replacer can be used. Cow's milk contains more fat and less lactose and calcium than mare's milk. Therefore, it should be fortified with 6 to 7% additional lactose or glucose (dextrose) and 0.25% calcium to make it up to the approximate composition of mare's milk.

KEY POINT

Although lactose and glucose are well digested, do not add disaccharide sugars such as sucrose (table sugar), corn syrup, or honey to fortify cow's milk. Young foals do not have specific enzymes in their gut lining to digest these sugars, and diarrhea and colic may result.

Milk for young foals can be made up by dissolving approximately 20 to 30 g (-l oz or 1-1.5 tablespoons) lactose (preferably) or, alternatively, glucose (dextrose) *(not* sucrose [table sugar]) into 1000 mL (approximately 2 pints) of low-fat (2%) cow's milk or accurately reconstituted cow's skim milk powder mixture.

KEY POINT

In each liter (2 pints), dissolve two level teaspoons (10 g) of dicalcium phosphate powder, because cow's milk is lower in calcium than mare's milk.

Make up fresh milk as required. Acidified milk replacers formulated for calves are suitable for foals and are well accepted. They may present less risk of digestive disturbance and keep longer when reconstituted than standard milk replacers.

Goat's milk is well tolerated, but if digestive upset occurs, then add extra dextrose and calcium as for cow's milk. When possible, it is best to house orphan foals in a loose box, preferably with a companion, such as a sheep or goat, for the first 1 to 2 weeks, especially during cold or wet weather.

BOTTLE FEEDING

A standard baby's bottle (e.g., a plastic bottle with screw-down teat, with a hole large enough for a

drip to collect when the full bottle is held teat downward) is suitable for foals up to 2 to 3 days of age, or alternatively, a soda drink bottle fitted with a small lamb's teat can be used. Feed at blood temperature (98.5°F, or 37°C) (drip some on inside of wrist; it should feel just warm) for foals 1 to 2 days of age, shaking the bottle occasionally during feeding.

KEY POINT

Ensure the bottle is held so that the foal's nose is lower than its forehead to reduce the risk of milk aspiration when drinking. Milk temperature can be lowered to room temperature (70-80°F, or 20-25°C) from day 2 of bottle feeding, in preparation for the introduction of bucket feeding.

FEEDING VOLUMES

Traditionally, orphan foals were given small volumes of milk up to 10 to 15% of their body weight daily to avoid overconsumption and digestive upset. Recent studies indicate that larger volumes of milk are well tolerated and will help achieve early growth rates comparable with nursing foals. A nursing foal will ingest up to 20% of its body weight in milk each day. Feeding volumes and management guidelines are provided in Table 18-6.

TABLE 18-6. Feeding Guidelines: Orphan Foal, Light Horse Breed 45 kg (100 lb) Body Weight. Always Provide Fresh Water Ad Libitum Each Day

Age	Volume/Day	Suggested Feeding Intervals
1-7 days Bottle change to bucket feeding by day 3-4.	First 12-16 hours 3-5 L (6-10 pints) colostrum. Then 1 L (2 pints) milk every 3 hours for first 2 days of age. 10 L (20 pints) daily. Approx. 2 L (4 pints) per feed.	4 hourly from day 3 6:00 AM-10:00 PM (5 feeds). A 2:00 AM feed may be given for the first 3-4 days to a weak or dehydrated foal.
7-14 days Bucket feeding. Provide 1 kg (2 lb) of 16-18% crude protein concentrate mix (see Concentrate Feeds in text) in a shallow dish near feeding bucket for foal to nibble between milk feeds.	12.5 L (25 pints) daily. Approx. 2.5 L (5 pints) per feed. Place milk in bucket and allow foal to drink ad lib between feeds.	4 hourly 6:00 AM-10:00 PM (5 feeds). If diarrhea or colic results from milk overload, reduce to half volume for 2 feeds, gradually increasing to full volume over 24-36 hours. Evaluate dehydration state before each feed if low-grade diarrhea persists.
14-21 days Provide 1.5 kg (3.5 lb) of 16-18% crude protein concentrate mix (see Concentrate Feeds in text) for ad lib consumption each day.	16 L (32 pints) daily Approx. 4 L (8 pints) per feed. Place milk in bucket and allow foal to drink ad lib between feeds.	6 hourly (4 feeds) 6:00 AM-10:00 PM (or later bedtime). (Refer to comments regarding diarrhea or colic above.)
21 days-8 weeks Provide ad lib 16-18% crude protein concentrate mix (see Concentrate Feeds in text) for consumption each day.	Increase total volume by 1 L (2 pints) per week to 18-20 L (36-40 pints) by 8 weeks of age. Approx. 6 L (12 pints) per feed. Place milk in bucket and allow foal to drink ad lib between feeds.	8 hourly (3 feeds) 6:00 AM, 2 PM, 10 PM.
8 weeks—weaning Provide ad lib 16-18% crude protein concentrate mix and access to grazing if possible each day.	Total volume 18-20 L (36-40 pints) per day. Divide volume between 2 and 3 feeds for ad lib consumption.	8-12 hourly (2-3 feeds) Once foal is eating concentrates, it may leave milk—reduce amount by 1 L (2 pints) per feed each week until milk is no longer taken.

KEY POINT

The volume of milk fed in liters (pints) should be approximately 20% of the foal's body weight in kilograms (pounds) each day. The volume should be adjusted at weekly intervals relative to body weight gain.

KEY POINT

If the foal is hungry and seeks more milk, an extra half volume of milk can be provided in between the 2-hour feeds. Monitor the amount and consistency of feces between feeds, and reduce the volume of milk if a white, pasty form of diarrhea develops in the presence of normal body temperature due to milk overload.

BOTTLE TO BUCKET CHANGE

Most foals can be taught to drink from a bucket from 2 to 3 days of age. Bucket feeding should be commenced within the first week to save time. In most cases, ad libitum milk in a bucket can be provided for foals after 2 weeks of age without risk of overload and associated diarrhea.

KEY POINT

A foal is more likely to drink from a bucket if it has not been fed for 4 to 6 hours because it will be hungry.

In a foal less than 1 week of age or one that will not drink, stimulate the sucking reflex by letting the foal suck on a finger (dip the finger in milk; moving it against the palate and tongue often helps). Once the foal starts to suck, lift the dish of milk until the foal drinks the milk, then remove the finger. Do not immerse its nostrils in the milk. Forcing the foal's head down into the milk is counterproductive. Repeat the process until the foal learns to drink.

Generally, young foals will nurse on a finger and will readily learn to drink once the milk has been tasted after one or two attempts. Foals that have been bottle fed for 1 to 2 days will be accustomed to drinking the milk mixture. Initially, offer the milk in a wide shallow container in a small enclosure with the foal. Show the foal where the milk is and splash milk on its nose. A foal will usually be tempted to drink once it becomes hungry.

KEY POINT

Once the foal learns to drink from the dish, place the milk in a light-colored shallow plastic bucket 300 mm (12 inches) in diameter, 200 to 250 mm (8-10 inches) deep, secured 600 mm (24 inches) above the ground (just below chest height) for subsequent feeds. A foal will not willingly put its head into a dark bucket space and, in this case, may be reluctant to take the milk. Raising the bucket will prevent the foal from standing in it, tipping it over, fouling it, and aspirating milk when drinking. Always provide a bucket of fresh clean water next to the milk feeding bucket for the foal to drink, particularly during warm weather.

CONCENTRATE FEEDS

Most young foals will nibble supplementary feeds and milk-based pellets from 7 to 10 days of age. Many commercial feeds formulated for foals containing 16 to 18% crude protein are available as creep or supplementary feeds. Alternatively, a palatable high-quality protein, low-fiber ration suitable for foals can be mixed as outlined in Table 18-7.

The dry feed base of a home-prepared ration can be mixed and stored for a few days. The

TABLE 18-7. Dietary Guidelines: Foal 1 to 6 Weeks of Age

Ingredient	Weight	Approx. Volume
Crimped or crushed oats	2 kg (4.5 lb)	4 L (8 pints)
Finely cracked, or preferably extruded corn	1 kg (2 lb)	1.5 L (3 pints)
Full cream milk powder*	300 g (10 oz)	~ 1.5 cups
Soybean meal	200 g (7 oz)	1 cup
Alfalfa cubes, crushed	500 g (1 lb)	1 L (2 pints)
Dicalcium phosphate	30 g (1 oz)	1.5 tablespoons
Vitamin E	250 IU	—
Salt	15 g (0.5 oz)	3 teaspoons
Molasses	1 cup in 1 cup warm water mixed into feed	
Commercial vitamin/mineral supplement at foal dosage		

*In a foal over 6 weeks of age, reduce milk powder by 100 g (3 oz) per week and substitute with 100 g (3 oz) soybean or 135 g (4.5 oz) canola meal, because these meals provide equivalent protein and lysine.

molasses and water should be added and mixed fresh each day to prevent fermentation and mold growth. If a commercial feed is used, additional vitamin E at 250 IU daily for each foal, as recommended in Table 18-7, and the molasses sweetener may be mixed into the feed.

After 2 to 3 weeks, most orphans (and nursing foals) will begin to graze to supplement their milk intake if green pasture is available. Alternatively, leafy good-quality alfalfa and grass hay mix, or chopped hay (chaff) dampened and sweetened with a 50:50 molasses and water mix, can be provided in a shallow safe trough. Dampening the hay or cubes will soften them for chewing and will help ensure better acceptance and utilization.

FOALS 6 TO 16 WEEKS

After about 6 weeks of age, reduce milk powder by 100 g (3 oz) per week and substitute with 100 g (3 oz) soybean meal. Milk powder in the ration may give slightly higher growth rates (because of its better digestibility in young foals) than comparable amounts of soybean meal. Soybean meal is generally less expensive and is a well-balanced protein source for older foals. When molasses is used as an appetizer, feeds should be made fresh each day.

KEY POINT

A nursing foal or orphan foal of a light horse breed with access to daily exercise can be provided with a 16 to 18% crude protein concentrate feed at the rate of 1.0 to 1.5% body weight (1.0-1.5 kg/100 kg [1-1.5 lb/100 lb]), increasing at the rate of 500 g (1 lb) per month of age in addition to good-quality pasture or alfalfa hay.

GUIDE TO HAND REARING

Feeding Rules

Certain rules of feeding should be observed to prevent gastric upsets, diarrhea, or food refusal:

- Changes in diet must be made gradually.
- Milk should be offered at blood heat (98.5°F or 37°C) to newborn foals during the first 1 to 2 days of age, then gradually lower the temperature to room temperature (70-80°F or 20-25°C).
- Change milk mixtures, if necessary, in a stepwise manner over at least 2 or 3 days.
- Provide palatable fresh concentrate feed from 7 to 10 days of age.
- Do not overfeed—it is best to feed small feeds more frequently to demand rather than large milk feeds during the first 1 to 2 weeks of age. Diarrhea induced by excess milk intake can reduce the digestive tract's immunity to bacterial pathogens, such as *Salmonella* spp.
- Provide an ad libitum supply of clean, fresh water in a bucket next to the milk feed bucket.
- Gradually wean the foal off milk at 2 to 2.5 months of age, relative to its rate of development and acceptance of concentrate food, and available grazing to supplement its diet.

Prevent Diarrhea

Increased intake of fibrous or high-moisture feeds, such as green grass, can cause mild diarrhea from 7 to 10 days of age. Control of intestinal threadworms *(Strongyloides westeri)* may be necessary if the foal develops a persistent "brown bubbly" type of diarrhea despite careful feeding and a normal body temperature. In foals from 10 to 14 days of age, a sample of fresh feces should be examined (within 2 hours of collection) for the characteristic embryonated strongyle-type eggs. *Note:* Infections are not patent for 8 to 14 days.

Exercise and Sunlight

Ensure that the foal has sufficient area to exercise and receives at least 2 to 3 hours of sunlight per day, preferably more. The foal should be taught to lead as soon as possible.

Parasite Control

The foal can be treated for internal parasites routinely at 4 to 6 weeks of age with an anthelmintic paste formulation (see Anthelmintics, Chapter 19). Repeat worming every 4 to 6 weeks, shifting to a new pasture after each worming, if possible.

General Observation

Instruct your clients to observe for signs of depression, diarrhea, dehydration, colic, and reluctance to drink or eat. Foals pass feces two to three times each day on a low-residue milk diet during the first 10 to 14 days of life and then up to five times daily once they start eating solid foods. Any change in consistency, volume, color, or odor and signs of discomfort on defecation should be noted.

KEY POINT

It is also good practice to give the newborn foal 1500 units of tetanus antitoxin. In weaker foals, consider an additional immunoglobulin

boost during the first week and at 5 weeks of age with a commercial immunoglobulin preparation just before the low point in immune protection in foals at 5 to 7 weeks of age.

Discipline

An orphan foal can become difficult to manage because it does not receive its mother's discipline and has no herd position. Care and strictness in handling the foal are therefore important. It is best not to allow a foal to play games with its handlers.

KEY POINT

An orphan foal should be weaned into a group of other weanlings at 3 to 4 months of age, provided it is independent and growing well, to ensure normal psychologic development.

NURSING FOALS

The most rapid period of growth and development of the young foal occurs during the first 3 months of age, with a well-nourished foal doubling its birthweight at 1 month of age and trebling it by 3 months of age. The peak of lactation occurs from 4 to 10 weeks after foaling, which corresponds to a foal's most active growth period. In most cases, the nutritional needs of the growing foal for the first 2 months will be provided by milk intake and access to good-quality pasture or sharing of supplementary concentrates (cofeeding) provided for the mare.

However, where the contribution from grazing falls short of requirements because of seasonal availability or heavy stocking rates, then supplementary concentrate feeds may be necessary to maintain optimal, but not excessive, growth rates.

KEY POINT

Foals that are supplemented with a good-quality, palatable ration from 2 to 3 months of age will suffer less nutritional setback at weaning.

When a mare's milk production dries up because of mastitis, injury, sickness, or malnutrition or in an aged mare with poor milk supply, it is best to wean a foal that is less than 4 weeks of age and rear it as an orphan.

Generally, concentrate feeds in a separate "creep" feed area are of no advantage to foals growing at an average rate. Studies indicate that average Thoroughbred-sized foals (450-500 kg [1000-1100 lb] mature weight) gain approximately 1 to 1.4 kg (2-3 lb) daily for the first 90 days after birth. Weight gain drops to 700 g (1.5 lb) daily at 6 months of age and to 500 g (1 lb) daily after weaning.

In poor seasons or when access to grazing is restricted, creep feeding may be beneficial. However, uncontrolled feeding can result in excess energy intake, with risk of DOD. Studies have shown that excess energy intake relative to exercise and growth results in a higher incidence of DOD. Most younger foals prefer to cofeed with their mothers, sharing a 14 to 16% crude protein lactating mare concentrate ration, because this provides security and develops eating patterns. Regular daily paddock exercise for at least 2 hours is important to ensure optimum musculoskeletal development. If the opportunity for the foal to exercise is restricted, then the mare and the foal ideally should be walked together for 15 to 20 minutes daily.

KEY POINT

Young nursing foals can be supplemented until weaning with a 16 to 18% crude protein, 1.5% calcium, 1.2% phosphorus, 70% grain mix as outlined in Table 18-8 at the rate of 1 to 1.5% body weight (1-1.5 kg/100 kg, or 1-1.5 lb/100 lb), complemented by unrestricted paddock exercise, without risk of overfeeding and DOD problems. If a 100% crushed grain and protein meal mix is used to supplement grazing, maximum feeding rate is 0.75 to 1.0% body weight per day in addition to milk intake.

Foals that develop evidence of DOD, with epiphysitis or contracted tendons, can be restricted in growth by reducing supplementary feed intake by 10 to 15% for 3 to 4 weeks and ensuring regular exercise. This is most easily done by teaching affected foals to lead, walking them for 10 to 15 minutes daily, or leading the mare with the foal at foot.

KEY POINT

If the concentrate intake is reduced to slow growth to avoid DOD, ensure the mineral and vitamin intake, particularly calcium, copper, phosphorus, zinc, manganese, and vitamin D, are maintained relative to the foal's body weight.

Many good-quality commercial creep feeds are

TABLE 18-8. Dietary Guidelines: Young Foal

Age: 8 weeks to weaning
Frequency. Ad libitum in creep area

Ingredient	Weight	Approx. Volume
Steam rolled barley	1 kg (2 lb)	2 L (4 pints)
Finely cracked, or preferably extruded corn	500 g (1 lb)	1 L (2 pints)
Skim milk powder*	100 g (3 oz)	~0.5 cup
Soybean meal	200 g (7 oz)	1 cup
Alfalfa cubes, crushed	300 g (10 oz)	700 mL (1.5 pints)
Dicalcium phosphate	20 g (0.6 oz)	1 tablespoon
Salt	15 g(0.5 oz)	3 teaspoons
Molasses	Half cup in half cup of warm water mixed into feed	
Commercial vitamin/mineral supplement at foal dosage		

* 100 g (3 oz) milk powder can be replaced by 100 g (3 oz) soybean or 135 g (4.5 oz) canola meal in a stepwise manner over 2 weeks for equivalent protein and lysine.

available. These are convenient and can be provided on an ad libitum basis from a self-feeder in a creep area. However, when owners wish to mix their own feeds, a suitable palatable creep feed ration can be made up as outlined in Table 18-8.

The ration can be offered at the rate of 1 to 1.5 kg/100 kg (1-1.5 lb/100 lb) body weight, adjusted to body weight as the foal develops, which at normal growth rates requires an increase of approximately 250 g (8 oz) each fortnight, or 500 g (1 lb) per month of age. Body development should be monitored weekly and the creep feed limited in foals that are developing too quickly owing to excessive energy intake. Where pasture is limited, good-quality alfafa and grass hay mix can be provided to satisfy appetite.

WEANLINGS

At weaning at around 6 months of age, the young horse should have reached 85% of its adult height and 50% of its mature body weight. A well-managed weaning process minimizes any psychologic, nutritional, or health stress on the young horse. Nutritional or disease-related setbacks at weaning can affect the subsequent age of maturity of the horse. Most foals by 5 to 6 months of age have little nutritional reliance on nursing, and if they are provided with good pasture or are accustomed to consuming concentrate feed, they will suffer no significant setback at weaning.

KEY POINT

Foals should be weaned before 7 months of age. Early weaning at less than 4 months of age has no significant benefit, except where a mare has inadequate milk to feed her foal.

Fretting is common in younger weanlings for the first 5 to 7 days, and they may lose their appetite. It is important that a palatable concentrate feed and good, clean pasture be available.

Dietery guidelines for weanlings to achieve a moderate growth rate are given in Table 18-9. Weanlings of light horse breeds should gain about 500 g (1 lb) daily until 12 months of age. Provision of concentrate (14-16% CP, 3 Mcal [13 MJ]/kg) at a rate of 1.25 to 1.5 kg/100 kg (1.25-1.5 lb/100 lb) body weight daily, supplemented ad libitum by good-quality alfalfa and grass hay mix or plentiful paddock grazing and free exercise will maintain an adequate rate of growth and development. Placement of feeders well apart from water troughs will also encourage exercise.

KEY POINT

The ration must be adjusted in proportion to body weight. The quality and availability of the pasture should be monitored regularly, particularly during autumn and winter periods.

Commercial rations such as sweet feed blends, extruded (cooked) grain mix, or pelleted feeds are popular because of convenience; uniformity of energy, protein, and calcium/phosphorus levels; and savings in time of mixing. Home-mixed rations must be palatable to tempt young weanlings to feed without encouraging overconsumption, particularly in cold weather. If the ration is offered ad libitum from automatic feeders, then care should be taken to restrict access to two or three feeding periods of 1 to 2 hours daily to avoid the risk of overeating under cold conditions.

Increase the amount fed and the hay or roughage in the ration by approximately 250 g (8 oz) per fortnight in proportion to growth rate. Regular weekly assessments of growth and development should be made to achieve a steady daily weight gain to 9 to 12 months of age. Feed adjustments should be made relative to quality of pasture,

TABLE 18-9. Dietary Guidelines: Weanling

Body weight: 200-250 kg (450-550 lb) weanling
Age: 6 months of age, maturing to 450-500 kg (1000-1100 lb), medium growth rate
Condition score: 5-6 (USA) 2-2.5 (Aust)
Frequency of feeding: Access to good-quality mixed pasture, one feed per day

Ingredient	Weight	Approx. Volume
Steam rolled barley or crushed oats	2 kg (4 lb)	4 L (8 pints)
Cracked or preferably extruded corn	1 kg (2 lb)	1.5 L (3 pints)
Soybean meal	400 g (13.5 oz)	~2 cups
Alfalfa cubes, broken for mixing, or chopped hay (chaff by weight)	1.5 kg (3 lb)	3 L (6.5 pints)
Dicalcium phosphate	60 g (2 oz)	3 tablespoons
Calcium carbonate	30 g (1 oz)	1.5 tablespoons
Salt	20 g (0.7 oz)	1 tablespoon
Vitamin E (optional)	500 IU	
Molasses	1/2-1 cup (125-250 mL [4-8 oz]) in 1 cup of warm water mixed into feed	

A commercial vitamin/trace mineral supplement containing vitamin A, vitamin D, copper, zinc, manganese, iodine, and selenium should be provided to meet growth needs of a weanling. Mixed legume and grass hay may be provided on an ad libitum basis, with the intake monitored and adjusted relative to the growth rate, appetite, and condition score of the weanlings.

opportunity for exercise, weather conditions, and the appetite and metabolic efficiency of the horse.

If the grazing pasture becomes dominant in either grass or legume, additional sources of phosphorus (to balance calcium intake from legumes and alfalfa hay) and calcium (to balance low calcium intake from grasses) may be necessary to maintain an optimal calcium-to-phosphorus ratio of 1.2 to 2.0 Ca:1.0 P in growing horses.

KEY POINT

The addition of 0.8% calcium (20 g calcium carbonate [1 tbsp]/kg of feed, or oz [½ tbsp)/lb of feed) to cereal-based rations and the addition of 0.5% phosphorus (30 g dicalcium phosphate [1½ tbsp]/kg of feed, or 1 oz/2 lb) or, preferably, a similar amount of sodium triphosphate, as a phosphorus source, to alfalfa-based rations is a useful guideline to balance the calcium and phosphorus ratio for growing foals.

Weanlings grazing tropical grass species containing a high oxalate content, which interferes with calcium absorption and is an underlying cause of nutritional secondary hyperparathyroidism (NSH), may develop clinical signs of DOD between 2 and 3 months after weaning. Concentrate feeds should contain 1.0% calcium powder (30 g/kg of feed, or 1 oz/2 lb) as calcium carbonate or a commercial supplement. It is best to feed the weanlings in a small yard or fenced corner area to ensure that the concentrate is consumed.

KEY POINT

The level of both elemental copper and zinc in a ration is recommended to be 20 to 50 mg/kg (10-25 mg/lb) of concentrate feed.

Additional copper and zinc can be provided by a commercial supplement containing these minerals at higher than NRC (1989) levels. Alternatively, thoroughly blending 1 part copper sulfate pentahydrate ($CuS0_4 \cdot 5H_20$; bluestone) with 9 parts glucose, castor sugar, or fine middlings (pollard), mixed into the ration at the rate of 10 g (½ tbsp)/20 kg (44 lb) of grain mix will provide 50 mg/kg in the concentrate, or approximately 25 to 30 mg/kg elemental copper in the total concentrate and hay ration. A similar mixing rate for zinc sulfate heptahydrate ($ZnS0_4 \cdot 7H_20$) will also provide approximately 30 mg/kg elemental zinc in the total ration.

YEARLINGS

KEY POINT

Most well-grown young horses should have achieved 90% of their mature height and 70% of their adult body weight at 12 to 15 months of age.

The yearling age is ideal to evaluate the growth and development of the young horse so that nutritional management can be modified to achieve the standards desired. Preparation of a yearling for sale requires careful attention to nutrition and ex-

ercise, particularly during the final 3 to 4 months before sale.

Each yearling for sale must be assessed individually, so adjustment in feeding and overall preparation can be tailored to achieve the standards required.

A commercial concentrate feed containing 3 Mcals (13 MJ)/kg of energy, 12 to 14% CP, at an approximate rate of 1.25 kg (2.5 lb) per 100 kg (220 lb) of body weight, will allow adequate growth and development. This ration should increase to 1.5 kg (3 lb) per 100 kg body weight when pasture is limited, with regular adjustment for body weight gain. However, because individual yearlings may differ in metabolic efficiency, each animal should be assessed weekly and fed to maintain an even growth rate. It is essential to avoid high or restricted energy intake, which may increase the risk of developmental orthopedic limb abnormalities.

When predominantly grass-based pastures are grazed, which often contain phosphorus in excess of calcium intake, provision of ad libitum alfalfa hay and the concentrate outlined in Table 18-10 is recommended to help maintain a positive calcium-to-phosphorus ratio. Good-quality grass hay (1.5-1.8 kg [3-4 lb]) may be provided with the meal for semiconfined horses on poor grazing. A commercial vitamin/mineral supplement containing both zinc and copper (40-mg/kg ration) and 750 to 1000 IU vitamin E may be mixed into the concentrate ration for performance horses or potential sale animals. Young horses grazing tropical grass pasture with high oxalate content should be provided with ad libitum alfalfa hay and brought into a yard to consume the concentrate feed containing 1% added calcium (i.e., 30 g/kg [1 oz/2 lb] of calcium carbonate) and 30 to 50 mg/kg copper and zinc if signs of DOD become apparent.

If pastures are legume-based, containing alfalfa or clover species, then total phosphorus intake relative to calcium may not meet the requirements of the growing horse. In this case, the concentrate feed should contain only half the amount of dicalcium phosphate, and extra phosphorus can be provided by mixing 360 g (12 oz) or 2 L (4 pints) of wheat bran into the guideline ration in Table 18-10.

TABLE 18-10. Dietary Guidelines: Yearling

Body weight: 325 kg (700 lb) yearling maturing to 450 kg (1000 lb)
Condition score: 5-6 (USA), 2-2.5 (Aust)
Frequency of feeding: Access to pasture, one feed daily
Provision: 75% NRC (1989) plus grazing to maintain growth and condition

Ingredient	Weight	Approx. Volume
Steam rolled barley or whole oats	3 kg (6.5 lb)	5.5 L (11 pints)
Soybean meal	400 g (13.5 oz)	~2 cups
Alfalfa cubes or lucerne chaff by weight	2 kg (4.5 lb)	4 L (8 pints)
Dicalcium phosphate	60 g (2 oz)	3 tablespoons
Calcium carbonate	30g (l oz)	1.5 tablespoons
Vitamin E (optional)	750-1000 IU	—
Salt	20 g (0.6 oz)	1 tablespoon
Molasses	1 cup in 1 cup warm water mixed into feed	

A commercial vitamin/trace mineral supplement containing vitamin A, vitamin D, copper, zinc, manganese, iodine, and selenium should be provided to meet needs of a yearling. Mixed legume and grass hay may be provided on an ad libitum basis, with the intake monitored and adjusted relative to the growth rate, appetite, and condition score of the yearlings.

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Performance Horses

GENERAL GUIDELINES

The rations for racing, showjumping, eventing, polo, and endurance horses should be formulated to meet the needs of the individual horse. This will be related to the horse's age and build; duration, type, distance, and intensity of exercise; stage of training; temperament; appetite and eating habits; and the climatic conditions.

KEY POINT

A performance horse will consume from 2.0 to 2.5% of its body weight per day in dry feeds as fed. A minimum content of 35 to 40% of the feed weight should be good-quality roughage in horses on high-grain rations to meet exercise needs. Smaller framed horses may eat less, whereas hard-working horses may consume feed at up to 3.0% of their body weight per day.

The feeding routine must be regular (designed to provide a natural feeding pattern), with three to four meals per day for stabled horses, providing a nutritionally balanced palatable diet. Rations need not be complicated mixtures. A balanced diet can be formulated using one or two sources of energy, a protein source if required, and adequate roughage consisting of either long-stemmed hay, chopped hay, or cubed hay. The ratio of grain to roughage relative to the needs of various classes of horses is provided in Table 18-3.

Supplementary calcium, phosphorus, electrolytes, or trace mineral/vitamin additives may be required to balance the ration or meet elevated needs relative to exercise intensity and duration. It is essential that horses in training have free access to clean fresh water at all times.

KEY POINT

Feeding must be relative to daily exercise demand and feed intake in proportion to body weight. The ration must be provided in sufficient bulk to enable the horse to spend 8 to 10 hours eating, but in a volume that an individual horse can consume to meet energy and other needs.

High-energy feeds, such as grains, must be reduced by about one-third on light work days or to one-third on rest days. It is preferable to start the night before a rest day with the total feed intake maintained by offering good-quality grass or mixed legume-grass hay, or 50% cubes and long-stemmed hay. Reduction of grain in this way avoids overactive behavior and the risk of metabolic problems such as myopathies on resumption of regular exercise. The full grain intake should be reintroduced in a stepwise manner over the 2 days after the rest days. The feeding routine must be designed to limit boredom in horses on concentrate rations confined to stables, particularly overnight, to avoid such vices as wood chewing, stall walking, and windsucking. Provision of good-quality alfalfa or grass hay with the evening feed is recommended.

KEY POINT

A horse's appetite reflects its well-being and acceptance of the ration.

The most common causes of reduced appetite are discomfort from gastroduodenal ulceration in horses on high-grain diets and overwork or excessively fast work for the stage of training. Up to 50% of horses in advanced training that lose their appetite or start to leave an increasing amount of feed have gastric ulceration (see Chapter 7). The use of sweeteners, such as molasses, and B-group vitamin supplements in sweet feeds is popular to help maintain the appetite and acceptance of the ration in hard-working horses.

Energy

KEY POINT

The energy content of the ration must be in proportion to the exercise intensity and duration to maintain a desired body weight, condition score, a quiet tractable temperament, and optimum level of performance.

A horse less than 4 years old will require a diet that contains adequate energy for growth, musculoskeletal development, and performance. A horse housed under cold conditions or a nervous horse will require higher energy intake to maintain its body condition and performance.

Cereal grains such as oats, corn, barley, milo (grain sorghum), and to a lesser extent wheat and rice can be provided as the carbohydrate energy base relative to availability, cost, and individual palatability. Smaller-framed horses, such as Standardbreds and Arabians, or horses in hard work that have consistently poor appetites are best fed rations containing higher energy-density feeds of up to 30% corn or barley and 6 to 8% fat as a substitute for oats. This will help reduce the overall volume of the ration to a bulk that can be comfortably consumed.

In hotter climates or in horses exercising over longer distances, corn, barley or rice, and small volumes of polyunsaturated fats, such as corn oil or other vegetable oils, can be substituted for more fibrous grains, such as oats, to increase the energy density of the ration and to reduce heat waste accumulated from hindgut fermentation of fiber and excess protein. When fat is used as an energy-dense feed source to reduce ration volume, at least 1 cup or 250 mL (½ pint) is the minimum amount that is useful as a substitute for cereal grains in the feed. There is no advantage gained by adding more than 10% of fat relative to the grain intake,

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or 500 mL (1 pint) or 460 g (1 lb) per 5 kg (11 lb) of grain.

KEY POINT

Fats added to the diet may increase the overall utilization of carbohydrate energy and may have a glycogen-sparing effect in horses working for extended duration. Fat added as oil should be fresh because rancid oil will not be readily accepted or utilized efficiently.

SUBSTITUTION GUIDELINES

On a volume basis, 250-mL (½ pint) or 1 cup (8 oz) of polyunsaturated oil will replace 6 cups (1.5 L [3 pints] or 750 g) of whole oats, 4 cups (1.2 L [2.45 pints] or 700 g) of rolled barley, 3½ cups (850 mL [1% pints] or 700 g) of milo, or 3 cups (750 mL [1½ pints] or 640 g) of cracked corn in the ration with an equivalent amount of energy.

Fat as oil must be introduced in a stepwise manner over 10 to 14 days, starting at a volume of 40 mL (1.5 oz) twice daily in each feed, reducing the grain in proportion as the oil is increased. This will help ensure efficient digestion, utilization, and acceptance. It may take 2 to 3 months for a horse to adapt to fully use fat as an energy source for aerobic exercise.

Protein

KEY POINT

Adult racing and equestrian performance horses require a 10 to 12% crude protein diet, containing at least 0.36% lysine (about 36 g lysine/10 kg dry feed). In working horses, protein intake is related to energy need. Generally, as the energy content of the ration is increased by the addition of grain, the corresponding increase in protein supplied by the added grain will meet normal daily needs of protein and lysine in racing horses, particularly where alfalfa hay or cubes contribute at least 50% of the roughage base to the concentrate ration.

However, when grass hay is the predominant roughage, additional crude protein, such as that provided by 120 g (4 oz) of extracted soybean meal (45% crude protein) per 1 kg (2 lb) of grass hay (6-8% crude protein), is recommended.

Excess protein not digested in the small intestine may overflow into the hindgut and increase heat waste during fermentation (six times higher than for carbohydrates) and requires additional energy and higher water requirement for excretion. Bacterial protein and excess feed protein are not absorbed from the hindgut. During early training, or in 2- to 3-year-old horses that are still growing, an extra 2% protein can be added by providing 2 cups (400 g, or about 1 lb) of soybean meal (45% crude protein) or 3 cups of canola meal (36% crude protein) to provide additional lysine and other amino acids for muscle, blood, and skeletal development.

KEY POINT

When fats are used as an energy source and the protein contribution from grains is reduced accordingly, V2 cup (100 g, or 3½ oz) soybean meal (45% crude protein) or 1 cup (165 g, or 5½ oz) canola meal (36% crude protein) for each cupful of fat added will maintain adequate protein intake. Provision of an additional good-quality protein meal, such as 2 cups of soybean meal or 3 cups of canola meal in each of the two feeds following hard exercise, competition, or racing may aid musculoskeletal repair processes in the post-race period.

Fiber

KEY POINT

All rations should be based on long-stemmed hay, chopped hay, or roughage cubes at the minimum intake outlined in Table 18—3. Adequate good-quality fiber to a minimum of 1.0% of body weight, or no less than 35 to 40% by weight of the total ration, must be provided.

Provision of hay overnight and, when possible, field exercise and grazing on a daily basis are helpful to reduce boredom and maintain the appetite. Excessively dusty hay, or hay containing airborne molds, will increase the risk and severity of lower respiratory tract allergies such as chronic obstructive pulmonary disease (COPD) and reactive airway disease (RAD) in confined horses. Dry, brittle, dusty hay should be dampened by soaking in clean water for 10 to 15 minutes and allowing it to drain to a moist soft consistency before feeding. Moldy hay must be avoided because it may cause respiratory allergy and toxin-induced colic or digestive upset. In stabled or confined horses and after long-distance travel, supplementary hay should be provided in a trough located below chest height to avoid wastage and facilitate tracheal and lower airway drainage. Bedding should be as dust free as practically possible.

KEY POINT

In racing horses, a reduction of hindgut volume and weight by reducing fiber intake to 0.5% body weight may help increase race performance due to lower hindgut weight, provided that the animal maintains adequate hydration and hindgut function.

Minerals

KEY POINT

The minerals of major concern in horses on high-energy, grain-based diets are the macrominerals calcium, phosphorus, and magnesium; the electrolytes sodium, potassium, and chloride; and the microminerals iron, copper, zinc, manganese, iodine, and selenium. Heavily sweating horses require higher amounts of calcium, sodium, potassium, chloride, magnesium, iron, and selenium to replace sweat loss.

Supplementation with calcium to balance the higher intake of phosphorus on grain and grass hay-based rations is essential to maintain skeletal integrity and reduce bone and joint disease in young horses in training. About 60 to 90 g (2-3 oz) of calcium carbonate (ground limestone 39% elemental calcium) added to cereal grass-based rations, or 60 to 90 g (2-3 oz) of dicalcium phosphate (23% elemental calcium, 18% elemental phosphorus) added to alfalfa or legume-based feeds, will satisfy basic needs for a 450- to 500-kg (1000- to 1100-lb) working horse. Many commercial supplements containing a calcium-to-phosphorus ratio (usually 8 to 1 Ca:P for grain and grass hay rations and 2.5-3.0 to 1 Ca:P for legume-based diets) are available.

When alfalfa hay, chopped hay, or cubes are used as a primary roughage source, additional phosphorus may be required to meet metabolic needs and maintain the calcium-to-phosphorus ratio to within a range of 1:1 to 2:1. Addition of 60 to 90 g (2-3 oz), or 3 to 4.5 tablespoons, of dicalcium phosphate, or a similar amount of sodium triphosphate as a phosphorus source, to the concentrate feed will help provide additional phosphorus.

Trace minerals are best provided by a commercial supplement containing 50% NRC (1989) requirements of iron, copper, zinc, manganese, selenium, and iodine.

KEY POINT

The addition of 15 mL/100 kg (½ oz/220 lb) of polyunsaturated oil such as sunflower, safflower, or blended vegetable oil daily to the ration of an equestrian performance horse is considered to maintain hair-coat condition in horses on low-fat grain and hay-based diets.

Electrolytes

The addition of 60 g (2 oz) or 3 tablespoons of salt daily will assist in meeting sodium and chloride needs, maintain fluid intake, and increase the acceptance of concentrate rations in a horse in light training during cool weather with little or no regular access to pasture. Supplementation with additional potassium chloride contained in 90 g (3 oz), or 5 tablespoons, of lite salt combined with 40 g (1 oz), 2 tablespoons, Epsom salts (magnesium sulfate) to provide magnesium will assist in replacing electrolytes in sweat losses of 15 to 30 L daily in hot weather or when a horse is worked for a period of 90 to 120 minutes or longer.

KEY POINT

A horse must have access to water at all times when it is being supplemented with electrolyte mixtures.

Vitamins

KEY POINT

Rations based on cereal grains and grass hays with little access to pasture may be relatively deficient in vitamins A, D, and E and some B-group vitamins, particularly thiamine (vitamin B,) and folic acid.

An intake of at least 1½ kg (3 lbs) of sun-cured alfalfa hay will provide sufficient vitamin D to meet needs in racing horses. Hot air-dried alfalfa and other hays contain lower levels, and supplementation with 1500 to 2000 IU vitamin D is recommended, especially if either the calcium or phosphorus intake is marginal or imbalanced. Provision of up to 1000 IU vitamin E daily is considered beneficial in performance horses.

KEY POINT

When polyunsaturated oils are used to increase the energy density of the ration in small-framed horses or horses with a poor appetite, an additional 250 IU of vitamin E per cup (250 mL or 8 oz) of oil is recommended above the basal 1000 IU vitamin E daily. Because of the inherent poor stability of vitamin E in vitamin/mineral mixes

containing iron and copper, it is best to provide additional vitamin E as an individual supplement.

In horses in hard work with restricted access to pasture or green feed, a commercial supplement containing vitamins A, D, and a range of B-group vitamins, particularly vitamin B_1, B_2, B_{12}, and folic acid, to at least 50% NRC (1989) recommendations may help to maintain appetite and counteract reduced hindgut enterosynthesis on high grain, minimum fiber rations.

Water

Working and performance horses should have access to clean cool water at all times, particularly for heavily sweating horses, those exercising for extended periods, and when electrolytes are being added to the diet. Up to 50 L (13 gallons) daily will be required by working horses under these conditions.

RACING HORSES

A number of breeds of horses are used for competitive racing, including Arabians, Appaloosas, Quarter horses, Standardbreds, and Thoroughbreds. The ration must be matched to the animal's body weight, condition score, build, stage and intensity of training, temperament, and eating habits. Ration guidelines for a racing horse in early training are provided in Table 18-11.

Early Training Notes

1. If the horse is not eating all that it is fed, reduce the weight of the hay by 0.25% of body weight (250 g/100 kg, or 4 oz/100 kg) to ensure that the full concentrate intake is maintained. If the horse consumes all the ration and appears hungry, increase the overnight portion of long-stem hay to provide additional bulk and relieve boredom.

2. Concentrate feeds may be dampened with molasses and water if desired to reduce dust and increase acceptance. Where oil is added as an energy source, it too will help to reduce dust in the feed.

3. Wheat bran (240 g [8 oz]) can be added to the evening meal to provide a limited amount of additional phosphorus on high alfalfa diets. Alternatively, an additional 20 g (oz), or 1 tablespoon, of dicalcium phosphate (provides 3.6 g phosphorus) for each kg (2 lb) alfalfa will help to balance the calcium/phosphorus ratio and correct a relative deficiency of phosphorus. For young horses in early training, extra soybean meal (45% crude protein) may be mixed into the grain meal (60 g/kg (2 oz/2 lb) in each of the morning and evening feeds as an additional source of high-quality protein for muscle, blood, and bone development and modeling.

4. During hot weather, in apprehensive fillies or small-framed horses unable to consume the bulk of concentrate ration, polyunsaturated oil (e.g., corn, sunflower, or canola oil) can be substituted for some of the rolled barley or whole oats.

5. A commercial electrolyte replacer may replace the salt and lite salt in the ration.

6. On light work days, delete corn from the ration, and on rest days, reduce barley or oats to one-third (commencing the evening meal before the rest day) and increase the bulk of the ration with alfalfa or grass hay.

Advanced Training Guidelines

Ration guidelines for a racing horse in advanced training are provided in Table 18-12.

1. The part substitution of fat (oil) as an energy source for fast exercise and racing has been shown to reduce gut weight and may improve speed in horses racing over distances exceeding 1600 m.

2. Adjust grain intake to maintain body weight and condition score relative to the individual horse's exercise program.

3. Smaller-framed horses, such as Standardbreds and Arabians, and fillies may not be able to consume the bulk of a standard ration, especially when in hard or fast work stage of training. Ration volumes may need to be reduced by substituting energy-dense grains and oils for oats.

4. An additional protein meal is not required for the ration outlined in Table 18-12 when alfalfa is used as the roughage base. If alfalfa hay or cubes are replaced by grass hay, 1 cup (200 g, or 7 oz) soybean meal (45% crude protein) or 1½ cups (250 g, or *%Vi* oz) canola meal (36% crude protein), as well as 30 g (1 oz [½ tbsp]) calcium carbonate (39% elemental calcium) should be added to the morning and evening meals.

5. Supplementary B-group vitamins may be given to help maintain the appetite if access to green grass or grazing is not provided on a daily basis.

EVENTING/HORSE TRIALING/POLO PONIES

Competition in the cross-country or steeplechase phase of 3-day eventing or horse trialing is one of

TABLE 18-11. Dietary Guidelines: Racehorse, Early Training

Body weight: Racing horse, 450-500 kg (1000-1100 lb)
Condition score: Arabian, Standardbred 4-6 (USA), 1.5-2.5 (Aust)
Thoroughbred 4-6 (USA), 1.5-2.5 (Aust)
Quarter horse 5-7 (USA), 2-3 (Aust)
Stage of training: First 6 weeks, moderate work up to 30-40 min daily
Frequency of feeding: Three feeds daily, stabled; evening feed home-mixed sweet feed
Provision: 100% daily requirement (NRC 1989)

Ingredient	Weight	Approx. Volume
Morning feed		
Rolled barley or whole oats	1.5 kg (3 lb)	3 L (6 pints)
Cracked corn	450 g (1 lb)	600 mL (1.25 pints)
Alfalfa cubes*	1.0 kg (2 lb)	2 L (4 pints)
Vitamin E	1000 IU	—
Salt	30 g (1 oz)	1.5 tablespoons
Midday-early afternoon feed		
1-1.5 kg (2-3 lb) dampened, dust-free grass hay, adjust to appetite. If available, 1-2 hours of grazing late morning after concentrate feed has been consumed. Alternatively, graze on lead midafternoon or 2 kg (4.5 lb) green grass in stall and afternoon walking exercise.		
Evening feed		
Rolled barley or whole oats	1.5 kg (3 lb)	3 L (6 pints)
Cracked corn	450 g (1 lb)	600 mL (1.25 pints)
Alfalfa cubes*	1.5 kg (3 lb)	3 L (6 pints)
Molasses (optional)	1 cup in 1 cup warm water mixed into feed	
Dicalcium phosphate†	30 g (1 oz)	1.5 tablespoons
Lightly sweating horses—cool weather		
Salt	30 g (1 oz)	1.5 tablespoons
Heavily sweating horses—hot weather		
Salt	30 g (1 oz)	1.5 tablespoons
Lite salt	30 g (1 oz)	1.5 tablespoons
Magnesium sulfate	30 g (1 oz)	1.5 tablespoons
Dicalcium phosphate	30 g (1 oz)	1.5 tablespoons
Dextrose/glucose (optional)	60 g (2 oz)	3 tablespoons
Commercial vitamin/mineral supplement containing vitamins A, D, and B-group and trace minerals, including 5 mg chromium daily (optional) for lean muscle development		
Overnight	2 kg dampened grass and alfalfa (lucerne) hay mix	

*Chopped alfalfa hay (lucerne chaff) may be added by weight as an alternative.
†*Note:* Horses less than 4 years old, provide 30 g (1 oz). 1.5 tablespoons, of calcium carbonate as well as dicalcium phosphate in evening feed.

the most demanding types of equine athletic activity. The horse must be well conditioned and physically fit, yet calm and obedient to compete successfully and score well in all three phases.

Early Training

Initial training regimens include trotting and cantering exercise for 30 to 40 minutes daily, with dressage and jumping training for up to 60 minutes on alternate days. The dietary requirements for a 500-kg (1100-lb) eventing horse for these regimens are similar to those for a racing horse in early training, as outlined in Table 18-11.

If the horse has access to pasture during the day and is confined to a stall or dry lot overnight, then the midday feed of hay can be deleted. If the horse is confined for most of the time, then the three feeds as outlined in Table 18-11 should be offered. The dietary composition and intake

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TABLE 18-12. Dietary Guidelines: Racehorse, Advanced Training

Body weight: Racing horse, 450-500 kg (1000-1100 lb)
Condition score: As for early training, may be leaner
Stage of training: Advanced training, breezed 3 times weekly, intense work 10-15 min daily
Frequency of feeding: Three to four feeds daily, stabled; evening feed home-mixed sweet feed
Provision: 100% daily requirement (NRC 1989)

Ingredient	Weight	Approx. Volume
Morning feed		
Rolled barley or whole oats	1.5 kg (3 lb)	3 L (6 pints)
Cracked corn	1 kg (2 lb)	1.5 L (3 pints)
Alfalfa cubes*	1 kg (2 lb)	2 L (4 pints)
Vitamin E	1000 IU	—
Salt	30 g (1 oz)	1.5 tablespoons
Midday-early afternoon feed		
Rolled barley or whole oats	1 kg (2 lb)	2 L (4 pints)
Cracked corn	450 g (1 lb)	600 mL (1.25 pints)
This feed may be dampened with water and fed with 1 kg (2 lb) dampened, dust-free grass hay to appetite. If available, 1-2 hours of grazing after concentrate feed has been consumed. Alternatively, graze on lead midafternoon or 2 kg (4.5 lb) green grass in stall and afternoon walking exercise.		
Evening feed		
Rolled barley or whole oats	1.5 kg (3 lb)	3 L (6 pints)
Cracked corn	750 g (1.5 lb)	1 L (2 pints)
Alfalfa cubes*	1.5 kg (3 lb)	3 L (6 pints)
Wheat bran (optional)	240 g (8 oz)	1.25 L (2.5 pints)
Molasses (optional)	1 cup in 2 cups warm water mixed into feed	
Dicalcium phosphate†	90 g (3 oz)	5 tablespoons
Lightly sweating horses—cool weather		
Salt	30 g (1 oz)	1.5 tablespoons
Heavily sweating horses—hot weather		
Salt mix consisting of		
Salt	30 g (1 oz)	1.5 tablespoons
Lite salt	30 g (1 oz)	1.5 tablespoons
Magnesium sulfate	30 g (1 oz)	1.5 tablespoons
Calcium carbonate	30 g (1 oz)	1.5 tablespoons
Glucose/dextrose (optional)	60 g (2 oz)	3 tablespoons
Commercial vitamin/mineral supplement containing vitamins A, D, and B-group and trace minerals, including 5 mg chromium (optional) for lean muscle mass		
Vegetable oil may be substituted for part of the grain content as an energy-dense feed in hard-working horses, or to reduce bulk of feed in small-framed horses or horses with loss of appetite; refer to text.		
Overnight		2-3 kg dampened grass hay

*Chopped alfalfa hay (lucerne chaff) may be added by weight as an alternative.
†Horses less than 4 years old, 30 g (1 oz), 1.5 tablespoons, of calcium carbonate as well as dicalcium phosphate in evening feed.

should be adjusted to suit the individual training regimen and the horse's appetite limit.

If the animal is small-framed or has a nervous temperament, 1 cup (250 mL [8 oz]) of polyunsaturated oil can be substituted for cereal grains in each of the morning and evening feeds to increase energy density and provide an alternative noncarbohydrate form of energy. Refer to page 669 for substitution rates.

Advanced Training

Once a horse begins cantering and galloping exercise for 20 minutes daily in preparation for steeplechase and cross-country phases, the energy density of the ration must be increased accordingly. The ration guidelines for a racing horse outlined in Table 18-12 may be adopted, deleting the midday concentrate feed.

On showjumping, dressage practice, or competition days, the amount of barley or oats should be reduced to 1 kg (2 lb) morning and evening, with corn deleted from the morning feed on that day. Reduce grain on light work days and rest days as recommended in Table 18-12.

SHOWJUMPERS/HUNTERS/WESTERN PLEASURE HORSES

Showjumpers, hunters, and roping/cutting horses train and compete at medium intensities, with a ration requirement for a 500- to 550-kg (1100- to 1200-lb) horse similar to the diet outlined in Table 18-11 in energy and protein requirements. The part substitution of corn, barley, or vegetable oil for oats as an energy-dense source will help to reduce the bulk of the ration to meet appetite limits in a smaller-framed horse or when the appetite is reduced by hard work.

SHOW HORSES/DRESSAGE HORSES

Although performance demand in terms of speed is not required for these horses, adequate energy must be provided for 40 to 60 minutes of daily training while maintaining a controllable temperament and optimum coat and body condition (see Condition Score, Table 18-5). Larger-framed and heavy body weight dressage horses may require 10 to 15% more bulk of feed than an adult show horse.

Grain intake is normally limited to 2 to 2.5 kg (4.5-5.5 lb) daily for a 500- to 550-kg (1100- to 1200-lb) equestrian performance horse, with roughage intake increased by 50% of the amounts outlined in Table 18-11. The use of oil as an alternative energy source is recommended for horses with nervous temperaments, or those that become "hyperactive" on grain-based diets.

KEY POINT

Because good coat condition is essential, the addition of 15 mL/100 kg (½ oz/200 lb) of sunflower, safflower, or blended oil daily to the basic ration is often given to promote a glossy hair coat. Coat gloss can be enhanced by including a daily vitamin/mineral supplement containing vitamins A and D, iron, copper, and zinc. Vitamin E supplementation may be provided only on alternate days in lightly worked horses.

ENDURANCE/TRAIL-RIDING HORSES

Studies have indicated that endurance horses expend up to 10 times more energy in a 100-km (60-mile) ride than a racing horse in a distance race. Many successful Arabian-bred endurance horses receive only half the amounts of cereal grain normally provided for racing horses. Endurance horses appear to be more sensitive than other performance horses to excessive amounts of soluble carbohydrates. Fats, such as corn and vegetable oils, can boost the energy density of the ration, and metabolism of fats at medium-intensity exercise may have a glycogen-sparing effect in long-distance aerobically exercised horses performing at less than 450 m/min.

When horses are regularly trained over 10 to 15 km (6-8 miles) daily, substitution of fat as vegetable oil in the ration for oats will increase energy density and reduce bulk to enable the horse to consume adequate energy to its appetite limit (Table 18-13). Supplements of energy-dense vegetable oil, ranging in volumes from 500 mL (1 pint [2 cups]) to 1 L (2 pints [4 cups]), divided between morning and evening feeds, have been used with success in endurance horses on minimal grain diets. Rations containing amounts of oil (fat) above 16% of the concentrate portion are likely to reduce muscle glycogen stores. Oils and fats should be introduced in a stepwise manner until the desired amount is reached in 10 to 14 days. Additional vitamin E (250 IU vitamin E per cup of oil in addition to the standard daily amount of 1000 IU vitamin E) is recommended, as is extra protein meal, such as ½ cup soybean meal (45% crude protein) or 1 cup canola meal (36% crude protein) per cup of fat.

Adequate fiber from grazing, grass hay, or mixed hay is essential to maintain efficient digestive function on grain and fat-based diets. It will ensure adequate water storage in the hindgut as a reserve against sweat loss during extended exercise.

KEY POINT

The intake of alfalfa hay or cubes as the major roughage should be restricted during long-term training to a maximum of 45 to 50% by weight of the roughage intake, with the balance provided by lower-calcium grass hays or pasture. Excess calcium intake from an alfalfa hay-based diet will suppress parathyroid gland function. During competition in excess of 4 hours' duration, sweat loss lowers blood calcium concentration, which is not rapidly replenished because of

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TABLE 18-13. Dietary Guidelines: Endurance Horse

Body weight: Endurance horse, 400-450 kg (900-1000 lb)
Condition score: 3-4 (USA), 1.5-2.0 (Aust)
Stage of training: 15-20 km daily ride at walk, trot, and canter
Frequency of feeding: Daily grazing, stabled overnight, two concentrate feeds daily
Provision: 80% NRC (1989) requirements from concentrates, 20% pasture contribution

Ingredient	Weight	Approx. Volume
Morning feed		
Rolled barley or whole oats	1.5 kg (3 lb)	3 L (6 pints)
Cracked corn	450 g (1 lb)	600 mL (1.25 pints)
Alfalfa cubes*	1 kg (2 lb)	2 L (4 pints)
Polyunsaturated oil	250 mL (8 oz)	1 cup
Vitamin E	1250-1500 IU	—
Salt	30 g (1 oz)	1.5 tablespoons
Midday-early afternoon feed		
Depending on grazing intake, provide up to 1-1.5 kg (2 lb) dampened, dust-free grass hay to appetite, avoiding excess alfalfa hay (see text)		
Evening feed		
Rolled bailey or whole oats	1 kg (2 lb)	2 L (4 pints)
Cracked corn	450 g (1.5 lb)	600 mL(1.25 pints)
Alfalfa cubes*	1.5 kg (3 lb)	3 L (6 pints)
Soybean meal	200 g (7 oz)	1 cup
Polyunsaturated oil	250 mL (8 oz)	1 cup
Molasses	1 cup in 1 cup warm water mixed into feed	
Dicalcium phosphate	60 g (2 oz)	3 tablespoons
Medium sweating horses (45 min exercise)		
Salt	30 g (1 oz)	1.5 tablespoons
Lite salt	30 g (1 oz)	1.5 tablespoons
Heavily sweating horses (long rides/hot weather; 45-60 min exercise)		
Salt mix consisting of		
Salt	30 g (1 oz)	1.5 tablespoons
Lite salt	30 g (1 oz)	1.5 tablespoons
Magnesium sulfate	30 g (1 oz)	1.5 tablespoons
Calcium carbonate	30 g (1 oz)	1.5 tablespoons
Dextrose/glucose (optional)	60 g (2 oz)	3 tablespoons
Commercial vitamin/mineral supplement containing vitamins A, D, and B-group and trace minerals, including iodine and selenium. Chromium 5 mg daily may facilitate carbohydrate metabolism.		
Overnight	1.5-2 kg (3-4.5 lb) dampened grass hay	

Refer to text for dietary manipulation prior to competitive endurance rides.
*Chopped alfalfa hay (lucerne chaff) may be added by weight as an alternative.

suppressed parathyroid action. Low blood calcium concentration increases the risk of tying-up and synchronous diaphragmatic flutter (the "thumps") during competition.

Fiber traps water in the large bowel and provides a fluid reservoir to replace sweat loss and combat dehydration during a long-distance ride. However, excessive fiber intake increases digestive bulk and body weight and may reduce competitiveness.

Supplements of calcium, other than provided in the grain and grass hay ration, should be limited to 0.5% of total feed (5 g/kg elemental calcium) or approximately 15 g (½ oz) of dicalcium phosphate, or 10 g (oz) of calcium carbonate per kg of concentrate or grain feed. This amount is adequate to maintain skeletal integrity and replace sweat loss during prolonged training periods without depressing parathyroid function. Daily provision of trace minerals such as copper, iron, zinc, manganese, iodine, and selenium to 50% NRC

(1989) levels in a commercial supplement is also recommended.

Supplements of 25,000 IU vitamin A, 1000 IU vitamin E, and a range of B-group vitamins are considered beneficial to maintain appetite, general vitality, and stamina in endurance horses. Doses of 3000 to 4000 IU vitamin E daily for 7 to 10 days before competition are often provided. Electrolyte depletion from sweat losses (amounting to 10 to 20 L daily) during training in hot weather must be replaced by daily supplementation of salt mixes containing sodium, potassium, chloride, magnesium, and calcium. This will help prevent dehydration, exercise myopathies, and synchronous diaphragmatic flutter (the "thumps") in the 1 to 2 days before and during a competitive ride.

The first requirement after exercise is water, followed by electrolytes and feed.

Endurance Training Guidelines

Refer to general feeding guidelines for performance horses (p. 667) and early training race horses (p. 671). For competition, feeding amounts and management presented in Table 18-13 may be adapted as follows:

1. **Preride feeding:** Many trainers reduce roughage levels to limit digestive mass and body weight during the 4 to 5 days before a competitive ride. This can be achieved by increasing corn by 25% and replacing overnight grass hay with 1 kg (2 lb) alfalfa hay to maintain calcium and protein intake without compromising parathyroid activity.

2. **Ride day:** At about 5 to 6 hours (no closer) before a competitive long ride, offer a mixture of 2 kg (4.5 lb) crushed corn and 2 kg (4.5 lb) broken-up alfalfa cubes or chopped hay (chaff), and one cupful (250 mL [8 oz]) oil (if the horse is routinely fed extra fat), dampened with 1 cup (250 mL [8 oz]) of a 50:50 molasses-water mix. Mix in half the evening allowance of salt mix for a horse that sweats heavily. Allow free access to water until the start of the ride and limited amounts of 1 to 2 kg of dampened or soaked alfalfa hay. Feeding 500 g (1 lb) of sweetened grain mix about 10 minutes before the start of the ride and before leaving each vet check station may slow the onset of fatigue without affecting utilization of added fats.

3. **During the ride:** If available, allow the horse to drink small quantities of fresh water from streams, etc. Intake of cold water will not cause colic if a horse immediately continues exercise. This is particularly important during a ride under hot humid conditions. A half dose of an electrolyte paste mix or liquid rehydration fluid may be given by syringe over the tongue before allowing the horse to drink. At least 2 L (4 pints) of water should be taken in after electrolytes are given in this way. Often horses will not drink sufficiently at checkpoints because of fatigue or excitement.

4. **At the vet check stations:** Place a bucket of cool (not cold) water in reach of the horse to allow it to drink as it requires. During humid weather, where a horse has sweated heavily during a ride, it may be useful to give 40 g (l oz) or 2 tablespoons of heavy sweat salt mix (see Table 18-13), made into a paste with 2 tablespoons of dextrose (glucose), deposited over the tongue by syringe, with access to water at all times. About 4 to 6 L (1-1.5 gallons) of 50:50 corn-alfalfa cube dampened sweet feed as recommended before the start or a commercial sweet feed may be offered at checkpoints. However, many horses prefer to consume dampened alfalfa or sweet grass hay provided in a hay net. A similar feed with electrolytes and water can be given at the completion of a ride.

5. **After the ride:** After cooling off, offer electrolytes, followed by a small amount of water to drink initially. Feeding up to 1 1½ kg (3 lb) of the standard grain and fat diet, with added electrolytes, used during training at 3- to 4-hour intervals for the first 18 to 24 hours will help restore muscle glycogen stores.

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Breeding Horses

MARES

Optimum fertility and properly balanced nutrition are interrelated. An adequate energy intake is paramount to meet condition standards and ensure minimal nutritional limitations to breeding success. Brood mares may be classified into three groups: nonpregnant (open or dry mares), pregnant, and lactating (wet mares). Nonpregnant

mares can be divided into maiden mares (fillies if less than 4 years old) and older nonpregnant mares that have bred previously.

NONPREGNANT MARES

Mares in Poor Condition

In practical terms, a mare to be bred should have an optimum condition score of 5 to 6 (USA; see Table 18-5). Thin or poorly conditioned mares should be supplemented with a grain mix or hay to increase energy intake in a stepwise rising plane of nutrition during the 4 to 6 weeks until mating.

KEY POINT

More heavily conditioned mares should be maintained at a constant body condition without obesity. Overly fat mares should be fed to maintain their condition because a loss of body weight will reduce subsequent fertility.

There is no advantage in a "flushing" feeding regimen, but poorly conditioned mares should be fed to increase body weight to a moderate condition score of 5 to 6 (USA) for breeding (see Table 18-5).

Maiden Mares

A maiden mare, especially one retired from a training stable to be bred or an overly fat well-cared-for mare, requires individual attention to ensure she does not lose weight before breeding. Maiden mares newly introduced to an established mare group are often chased away from feed tubs by the older mares. A number of additional feed tubs positioned away from the main feeding area should be provided to ensure that maiden or lower-social-order mares can feed without anxiety and disturbance. It is best to segregate maiden mares into a separate group away from older or socialized mares on larger breeding farms.

Mares in Training

Mares sent direct from training barns to be bred will often lose condition once the high-energy ration is discontinued. They may fail to cycle and conceive early in the season. It is best to maintain such a mare on the race training ration and exercise regimen and breed the mare while in work. Once pregnancy is confirmed, the ration can be slowly reduced over a 4- to 6-week period to the level recommended for pregnancy.

KEY POINT

Do not reduce the energy intake of an overly conditioned mare or a mare in early pregnancy during the first 90 days after conception because it will result in a higher risk of embryonic abortion.

Older Mares

Many older Thoroughbred mares may lose condition during the winter months as a result of teeth problems, advanced age, or heavy parasite burdens. Loss of body weight can cause inward sloping of the perineal area and increase fecal contamination of the vulva and the risk of pneumovagina and breeding-tract infection. It is best to allocate underweight older mares to a separate group of mares. Check their teeth, treat for parasites, and feed a higher-energy ration to build up body condition, which will help achieve a more upright perineal conformation.

PREGNANT MARES

First 8 Months After Breeding

Many owners tend to overfeed pregnant mares and underfeed lactating mares. Dietary guidelines for a mare during the first 8 months of pregnancy are outlined in Table 18-14. Regular fortnightly monitoring and adjustment of the ration should be carried out to ensure the mare maintains body weight and condition. In many cases, spring pastures will maintain pregnant mares in good condition, with supplementary hay provided overnight, if necessary. Under drought, winter, flood, and snowbound conditions, pasture availability should be monitored daily and supplementary concentrates or hay provided, particularly to pony breed mares. Even short-term starvation over a 12- to 24-hour period if grazing is restricted because of adverse weather may predispose a pregnant pony mare to acute hyperlipemia syndrome (refer to Chapter 7).

Last 90 Days Before Foaling

The energy, protein, and calcium intake should be increased during the last 90 days of gestation, as the unborn foal makes half its growth during this period. The increase in energy, protein, and calcium can be achieved by providing 3 kg (6.6 lb) alfalfa hay to replace grass hay as the supplementary feed. Alternatively, each month until foaling, add an extra 500 g (1 lb) barley or oats, 100 g (3 oz) soybean meal or 125 g (4 oz) canola meal,

TABLE 18-14. Dietary Guidelines: Mare Prior to Breeding

Condition score: 4–5 increasing (USA), 1.5-2.0 increasing (Aust)
Body weight: 450-500 kg (1000-1100 lb) mare
Nonpregnant, 4 weeks before breeding, poor general condition
Frequency of feeding: Grazing, one concentrate feed daily in evening, hay overnight in sheltered area
Provision: Maintenance plus 10%

Ingredient	Weight	Approx. Volume
Rolled barley or whole oats	2.5 kg (5.5 lb)	5 L (10 pints)
Alfalfa cubes*	2.5 kg (5.5 lb)	5 L (10 pints)
Vitamin E	500-1000 IU	—
Salt	30 g (1 oz)	1.5 tablespoons
Molasses	1 cup in 1 cup warm water	mixed into feed
Dicalcium phosphate	60 g (2 oz)	3 tablespoons
Commercial vitamin/mineral supplement containing vitamin A		
Overnight	2-3 kg (4.5-6.5 lb) dampened grass or alfalfa hay or ad libitum in hay rack; adjust the amount of feed to achieve and maintain a fleshy condition score at the time of breeding.	

*Chopped alfalfa hay (lucerne chaff) may be added by weight as an alternative.

and 20 g (oz) or 1 tablespoon dicalcium phosphate to the ration outlined in Table 18-14.

Pastured mares will usually exercise sufficiently to maintain good body condition without excessive weight gain. Exercise can be encouraged by locating feeding and watering facilities well apart. Where a heavily pregnant mare is confined to a small yard, walking on the lead for 15 to 20 minutes at least every second day will provide beneficial exercise. Mares can be lightly exercised under saddle up to 1 month before their due foaling date, with care to avoid physically stressful exercise.

Lactating Mares: First 3 Months

KEY POINT

It is essential to avoid weight loss during the first 2 to 3 months of lactation, particularly in overly fat mares; otherwise, milk production and subsequent fertility will be reduced.

If a nursing (wet) mare is bred and conceives within the first 2 to 3 months of lactation, any weight loss may result in early embryonic abortion and a return to service. The peak of lactation occurs 4 to 10 weeks after foaling. A lactating mare can consume up to 3% of her body weight of a dry feed diet each day. The failure of many lactating mares to breed and maintain conception within the first 3 months after foaling may be related to deficiencies of energy and of protein and phosphorus content of the diet. In cold weather, the concentrate ration of a lactating mare should be increased by 10 to 15% to meet elevated demands to maintain body warmth.

The dietary intake of a lactating mare must be evaluated regularly to ensure that the mare is maintaining body condition. This will help maintain adequate milk production and optimum fertility to breed in the current season.

Dietary guidelines for a mare during the first 3 months of lactation are outlined in Table 18-15.

KEY POINT

A good-quality 14 to 16% crude protein ration will also serve as a cofeed for the mare's nursing foal to encourage it to mimic and develop eating habits and reduce nutritional stress at weaning (see Table 18-15).

Lactation: 3 Months to Weaning

Once a mare has passed her lactation peak, then the demand for energy, protein, calcium, and phosphorus is reduced. Using the ration outlined in Table 18-15 for a 500-kg (1100-lb) mare, the amount of grain can be reduced by 500 g (1 lb)

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TABLE 18-15. Dietary Guidelines: Mare—Lactation

Condition score: 6-7 (USA), 2.5-3.0 (Aust)
Body weight: 500 kg (1100 lb) mare
First 3 months of lactation
Frequency of feeding: Grazing, one concentrate feed daily in evening, hay overnight in sheltered area. Divide into two feeds during cold weather.
Provision: 80% NRC (1989) supplemented by grazing on spring pastures; the processed grain in the ration enables it to be used as a cofeed for a foal.

Ingredient	Weight	Approx. Volume
Rolled barley or whole oats	3 kg (6.6 lb)	6 L (12 pints)
Cracked, or preferably extruded corn if ration used as a foal cofeed	1.5 kg (3 lb)	2.5 L (5 pints)
Alfalfa cubes*	4 kg (9 lb)	8 L (16 pints)
Soybean meal	300 g (10 oz)	2 cups
Wheat bran (optional)	450 g (1 lb)	2.5 L (5 pints)
Salt	60 g (2 oz)	3 tablespoons
Molasses (optional)	1 cup in 2 cups warm water mixed into feed	
Vitamin E	500-1000 IU	—
Dicalcium phosphate	120 g (4 oz)	6 tablespoons
Commercial vitamin/mineral supplement containing vitamins A and D and zinc, copper, and other trace minerals.		
1-2 kg (2-4.5 lb) supplementary mixed alfalfa and grass hay can be provided to appetite, depending on pasture availability. Evaluate condition regularly and adjust ration to maintain optimum condition score.		

*Chopped alfalfa hay (lucerne chaff) may be added by weight as an alternative.

per month, the soybean and bran omitted, and the dicalcium phosphate reduced to 60 g (2 oz) daily, with hay fed to appetite to maintain the mare in a moderate to good condition score. If the mare failed to conceive during the first 3 months of lactation, then the energy level of the ration should be maintained to avoid body weight loss until the mare is bred successfully, confirmed in foal, and is at least 3 months pregnant.

STALLIONS

A resting stallion should be fed to maintain a trim to moderate body condition (condition score 6 to 7 [USA] or 2.5 to 3.0 [Aust]) and provided with an opportunity for paddock exercise. Alternatively, the stallion can be lunged for 10 to 15 minutes two to three times per week, or if tractable, ridden under saddle to ensure physical fitness.

Breeding Season

In practice, the nutritional need of a working stallion during the breeding season is related to the age of the stallion, his appetite and condition, and the number of times he is bred each week. Some breeding farm managers prefer stallions in "show condition" with a condition score above 7 (USA) or 3.0 (Aust) to exhibit to visiting owners of mares, but a breeding stallion should not be allowed to become excessively fat. A diet containing 12 to 14% crude protein is adequate for breeding purposes, because higher protein diets are unlikely to increase fertility and libido. Only good-quality feeds and hays should be used. Stallions should be kept fit by regular exercise by lungeing at a slow to medium trot for 10 to 15 minutes twice weekly if the opportunity for paddock exercise is not available.

KEY POINT

It is important to avoid excessive or hard regular exercise because it may lead to fatigue and reduce the stallion's interest in mares and his breeding libido.

A diet as outlined in Table 18-11 for a horse in training, omitting the corn, would be adequate to maintain a 500-kg (1100-lb) working stallion during the breeding season. Supplementation with 15 mL of polyunsaturated oil per 100 kg (½ oz/220 lb) body weight to promote coat condition and a broad-spectrum vitamin and mineral supplement,

providing 10,000 IU vitamin A, 500 IU vitamin D, and 200 IU vitamin E per 100 kg (220 lb) body weight, is recommended on a daily basis for regularly used stallions. During hot weather, 15 g (½ oz) salt per 100 kg (220 lb) body weight, or a commercial electrolyte replacer, will help to maintain hydration and physical well-being. When a stallion is not provided with green pasture grazing or green feed on a daily basis, an oral supplement of B-group vitamins and perhaps vitamin C in stallions over 15 years of age may help maintain appetite and vitality.

FURTHER READING

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Clinical Nutrition of Sick Horses

The recovery, appetite, and health of horses suffering from minor disease, trauma, sepsis, or specific organ disease can be enhanced by specifically formulated diets. Horses often become inappetent after minor injury, infectious disease, or surgery or as a result of excessively hard work.

KEY POINT

The diet formulated for sick horses must be palatable, meet the specific needs of the animal, and, in the case of rehabilitation from organ disease, avoid nutrient overload that could suppress metabolic activity and delay recovery.

It is also important to ensure that horses are properly nourished before surgery and are provided with an adequate diet during recovery. Diets can be formulated for either complete nutritional support or partial supplementation, depending on the horse's appetite and ability to eat and digest feeds. Many commercial feed companies provide dietary advice on suitable feeds for sick or injured horses. The amount of feed and water consumed must be recorded along with routine clinical assessment of a sick horse. During recuperation, serial monitoring of blood variables, including hematology, plasma total protein, albumin, electrolyte and acid-base analyses, and muscle and hepatic enzymes is recommended.

KEY POINT

Sick, injured, or hospitalized horses are often confined and are less active and have up to 25% lower energy needs than those of equivalent healthy horses on a maintenance diet.

When extensive tissue healing is in progress, a diet containing adequate energy and good-quality protein should be provided. There must be no delay in evaluating the animal's condition and providing appropriate nutritional support to assist and enhance recovery.

The dietary guidelines in Tables 18-16 to 18-22 are based on a 450-kg (1000-lb) adult horse and can be apportioned to individual patient body weight. As a guide, most sick or injured horses can be maintained on an intake of 1.5 to 2.0% body weight, or 1.5 to 2.0 kg/100 kg (1½-2 lb/100 lb) body weight. Because these specific diets are meant only for short-term nutrition of sick horses, no special recommendations for young horses, working horses, or lactating mares are given. Adjustments to meet their specific needs, as outlined in Tables 18-6 to 18-15, may be necessary if the diets are fed for extended periods.

Infection and Sepsis

Septic conditions commonly encountered in horses include peritonitis, pleural effusion, and severe cellulitis after trauma. Meeting specific nutrient needs may improve a horse's clinical state and retard the rate of catabolism and amino acid drain that can occur in severely traumatized horses and those with septic conditions.

KEY POINT

Horses recovering from sepsis require a palatable diet containing highly digestible energy, good-quality protein, and other nutrients to maintain metabolic function.

Because the infection itself may partition nutrients from general metabolism, an energy-dense diet, containing essential amino acids and supplements of B-group vitamins and vitamins E and C, is required to ensure metabolic function and maintain the immune response to infection during the recovery phase.

TABLE 18-16. Dietary Guidelines: Severe Trauma or Infection

Body weight: Adult, 450 kg (1000 lb)
Frequency of feeding: Divide into two or three feeds daily, hay ad libitum
Provision: Maintenance plus 20%

Ingredient	Weight	Approx. Volume
Cracked, or preferably extruded corn	2 kg (4.5 lb)	3 L (6 pints)
Boiled barley (wet weight)	2 kg (4.5 lb)	3 L (6 pints)
Soybean meal	350 g (12 oz)	2 cups
Alfalfa cubes*	2 kg (4.5 lb)	4 L (8 pints)
Polyunsaturated oil	250 mL (8 oz)	1 cup
Brewer's yeast	60 g (2 oz)	3 tablespoons
Vitamin E	1000 IU	—
Zinc supplement	200-400 mg	Commercial supplement or 1.0 g (Vi teaspoon) zinc oxide or alternatively 2.5 g (½ teaspoon) zinc sulfate daily
Dicalcium phosphate	60 g (2 oz)	3 tablespoons
Salt	45 g (1.6 oz)	~2 tablespoons
Molasses	1 cup with 1 cup hot water mixed into feed	
Alfalfa or alfalfa-grass hay mix ad libitum to appetite		

*Chopped alfalfa hay (lucerne chaff) may be added by weight as an alternative.

Dietary guidelines for a severely traumatized or septic horse are given in Table 18-16.

Many septic horses will have reduced appetites, and every attempt should be made to improve their desire to eat and increase their nutrient intake. Dampened feeds, sweetened with *2%* molasses or 0.5% yucca solution, and chopped apples and carrots may be useful to rekindle interest in eating. Good-quality alfalfa hay (dampened), oaten, or Timothy hay can be provided to satisfy appetite. In stabled horses, fresh green grass (1.5 kg [3 lb]) provided daily may stimulate interest in feed. For growing horses, an extra 250 mL (8 oz) or 1 cup of soybean meal and 60 g (2 oz, or 3 tbsp) dicalcium phosphate should be added to the total ration. The ration should be fed on an ad libitum basis, with food available at all times but limited to the maximum daily intake recommended.

Wheat bran (225 g [8 oz]) (250 mL or ½ pint) mixed with an equal amount of crushed oats and 1% molasses may be soaked with boiling water and mixed into the meal when warm to provide a useful appetite stimulant. (Add boiling water and soak for 10-15 minutes until cool.)

Canola meal or linseed meal, at 1½ times the weight of soybean meal to give equivalent protein, is often more palatable for sick horses requiring a good-quality protein source than soybean meal initially. Once the appetite is regained, soybean meal can be reintroduced because it has a better balance of essential amino acids.

In horses recovering from peritonitis, it is best to offer small amounts of feed up to 2 kg (4.5 lb) every 4 to 6 hours to avoid digestive overload and discomfort caused by large infrequent feeds. It will also help to regulate insulin responses to carbohydrate intake and improve the overall metabolic function.

KEY POINT

Overloading with energy and amino acids must be avoided, because excessive nutrient intake can lead to other complications such as impactions, colic, laminitis, and diarrhea during the recovery phase.

Severe Trauma and Burns

In contrast to healthy horses, sick or injured horses may not always be hungry, and the diet must be processed to improve digestion and increase their appetite and feed intake.

Well-ground, pelleted, and particularly extruded feeds are often more palatable and better digested in the small intestine in a horse with a limited appetite. If the chopped or processed feed is dry or the alfalfa cubes are hard to chew, they should be soaked in warm water until swollen,

TABLE 18-17. Dietary Guidelines: Severe Burns, Trauma, and Inappetence

Body weight: Adult, 450 kg (1000 lb)
Frequency of feeding: Every 8 hours (3 times daily)
Provision: Maintenance plus 25%
Slurry composition feed every 8 hours

Ingredient (Each 8 Hourly Mix Contains)	Each Feed Weight	Each Feed Approx. Volume
Dehydrated cottage cheese	225 g (8 oz)	300 mL (0.75 pint)
Dextrose/glucose powder	120 g (4 oz)	8 tablespoons
Alfalfa cubes*	1 kg (2 lb)	1.5 L (3 pints)
Polyunsaturated oil	125 mL (4 oz)	0.5 cup
Salt	15 g (0.5 oz)	1 tablespoon
Dicalcium phosphate	20 g (0.6 oz)	1 tablespoon
Brewer's yeast	20 g (0.6 oz)	1 tablespoon
Vitamin E	500 IU	—
Vitamin C	2.5 g	teaspoon
Water		Add water to volume approximately 4 L (8 pints) to prepare a liquid slurry suitable to flow through a large-bore nasogastric tube.

*Chopped alfalfa hay (lucerne chaff) may be added by weight as an alternative.

then broken up and mixed into a slurry. The amount of feed can be adjusted, increasing as the animal's clinical condition improves over 7 to 10 days. An ad libitum supply of good-quality dampened grass hay or fresh green feed, if available, should be provided to help minimize boredom between meals and encourage interest in feeding.

Good-quality commercial sweet feeds containing 12 to 14% crude protein, fortified with 1 cup of soybean meal per 2.5 kg (5.5 lb) grain-based sweet feed and with added molasses may improve the appetite and provide additional available amino acids and other nutrients. Brewer's yeast (60 g [2 oz]) added to this mix or, alternatively, an oral vitamin B-group vitamin supplement may also help maintain the appetite.

A parenteral dose of B-group vitamins, given each day or on alternate days, may help to improve general well-being and appetite. Theoretically, for a 450- to 500-kg (1000- to 1100-lb) horse, a compounded commercial or individual supplement containing up to 10 g vitamin C, 1000 IU vitamin E, as well as 25,000 IU vitamin A and 200 to 400 mg zinc may be beneficial to aid epithelial regrowth and healing in the case of burns and large areas of traumatized skin and tissue.

Often sick or inappetent horses can be tempted with chopped apples or carrots, and many prefer to nibble fresh grass or dampened hay from the floor of a stall. In severe cases, when the horse is totally inappetent, force feeding of a slurry by nasogastric tube may be required (Table 18-17).

Esophageal Disease

In horses with esophageal complications, semiliquid or slurry diets may be passed through an indwelling nasogastric tube or delivered by an esophagostomy placed in the middle to lower third of the neck. Dietary guidelines for a slurry diet are suggested under trauma and infection (see Table 18-17). Volumes of 1½ to 2 L (3-4 pints) per 100 kg (220 lb) body weight can be given every 6 to 8 hours relative to the animal's condition and clinical improvement. The animal should be observed regularly for signs of metabolic complications such as laminitis (stance, weightbearing discomfort, and a digital pulse in the hooves) and medical problems such as low-grade colic, diarrhea, and dehydration. Adequate volumes of intravenous fluids may need to be given to maintain hydration and acid-base balance (see Therapy, Chapter 19).

Enterolithiasis

Feeding a diet with 8 to 10% crude protein based on controlled amounts of grains and grass hay

with minimal magnesium and phosphorus and maintaining an acidic cecal pH between 6.5 and 6.7 has been reported to be effective in decreasing the recurrence of enteroliths in susceptible horses. Diets should contain a minimal amount of alfalfa to reduce protein, calcium, and magnesium intake. Wheat bran should not be included because it may increase the incidence of enterolithiasis in susceptible horses. Provision of a diet as suggested for a 450-kg (1000-lb) geriatric horse is suitable (as outlined in Table 18-23), omitting the soybean meal and replacing the alfalfa cubes with chopped grass hay to lower the protein intake. Adding 120 mL (4 oz) or half a cup of apple cider vinegar to each of two feeds daily, or alternatively 250 mL (8 oz) or 1 cup to a single feed, may also help to decrease the risk of enterolith formation.

KEY POINT

Although high-grain diets will reduce cecal pH by encouraging fermentation of excessive soluble carbohydrates in the hindgut to D-lactic acid, the risk of laminitis and low-grade diarrhea and hyperactivity due to high energy intake are increased.

When these diets are found to encourage wood chewing, as triggered by reduced cecal pH, 2 kg (4.5 lb) grass hay, dampened with 50:50 molasses-water solution in a trough and offered overnight to stabled horses, may reduce the risk of this chewing habit.

Chronic Diarrhea

In most cases, diarrhea in adult horses results from large bowel dysfunction. Common primary causes include an abnormal or disrupted fermentation process, low cecal or hindgut pH due to high carbohydrate intake in grain or lush pasture, chemical- or plant-induced hypermotility of the bowel, altered fluid absorption, and physical irritation of the bowel wall in heavily parasitized horses. Release of large numbers of hypobiotic cyathostomes from gut wall 4 to 7 days after anthelmintic treatment or seasonal pasture flushes also should be considered.

KEY POINT

Underlying infection with cyathostome parasites cannot be confirmed by fecal egg counts. Release of hypobiotic larvae and development of heavy burdens of immature, small strongyles may occur when sexual maturity is reached at 4 weeks after hypobiotic release.

RESTING THE LARGE BOWEL

It is unwise to fast horses for more than 18 to 24 hours to reduce large bowel activity, even in horses that have undergone large colon resection or mares recovering from rectovaginal reconstructive surgery. This is because hypoproteinemia may exacerbate the diarrhea. Pony breeds may risk developing hyperlipemia if starved for more than 12 hours after surgery or bowel resection. Frequent, small, low-residue meals should be provided, containing 12 to 14% good-quality crude protein, additional fat as an energy source, and adequate phosphorus. Intake of fiber as hay should be restricted to 0.75 to 1.0% body weight daily divided into three and four meals for the first 25 to 30 days after surgery.

KEY POINT

Frequent small feeds are recommended during the first 7 to 10 days after surgery to reduce gut distention and overload. There is also an increased risk of anaerobic bacterial colonization of the devitalized starved bowel and bloodborne septicemia.

REDUCED-RESIDUE DIETS

A maintenance ration with a low fiber residue, containing 12 to 14% crude protein with available amino acids, will maintain normal small intestinal function, protein, and energy uptake while helping to reduce large bowel activity. Commercial extruded feeds and pelleted diets will meet nutrient requirements. The energy content of the ration can be boosted by adding 5 to 8% fresh vegetable oil as fat. (Usually, up to 1 cup [250 mL or 8 oz] provided in each morning and evening meal.) Fat must be introduced in a stepwise manner with 45 to 60 mL (1½-2 oz) added at 3-day intervals over 10 to 14 days to ensure acceptance and optimum fatty acid utilization. When available, specialized high-protein commercial liquid diets administered by nasogastric tube will minimize residues and rest the large bowel. Guidelines for a low-residue diet are given in Table 18-18.

REESTABLISHING NORMAL FERMENTATION

If abnormal fermentation is suspected after a history of chronic diarrhea or high-dose antibiotic therapy, as evident by passage of poorly fermented food or sour, pasty-type feces after grain overload, large bowel flora can be reestablished in otherwise clinically normal horses.

TABLE 18-18. Dietary Guidelines: Persistent Diarrhea

Body weight: Adult, 450 kg (1000 lb)
Frequency of feeding: Divide into four feeds daily (~2 L |4 pintsl per feed)
Provision: Maintenance. Grass hay or cubes ad libitum

Ingredient	Weight	Approx. Volume
Cracked or preferably extruded corn*	2 kg (4.4 lb)	3.5 L (7 pints)
Soybean meal*	450 g (1 lb)	3 cups (1.5 pints)
Boiled linseed meal	300 g (10 oz)	2 cups (1 pint)
Polyunsaturated oil	375 mL (13 oz)	1.5 cups (1.5 pints)
Alfalfa cubes†	1 kg (2 lb)	2 L (4 pints)
Brewer's yeast	60 g (2 oz)	3 tablespoons
Salt	60 g (2 oz)	3 tablespoons
Vitamin E	1000 IU	—
Molasses	Half cup in half cup hot water mixed into feed	
If acidotic, add 60 g (3 tablespoons) sodium bicarbonate to morning and evening feeds.		
Ad libitum good quality grass hay overnight to appetite.		

*Preferably extruded to improve small intestine digestion.
†Chopped alfalfa hay (lucerne chaff) may be added by weight as an alternative.

KEY POINT

A diet containing 20% of the roughage of soluble fibers from beet pulp, soybean hulls, or chopped grass hay is useful to encourage fermentation in the large intestine.

Guidelines for a diet to reestablish large bowel flora are given in Table 18-19.

Diets with a high alfalfa content may prolong recovery, and in this case a fiber base of chopped grass hay dampened to reduce dust is preferred. Specialized commercial *Lactobacillus acidophilus* and mixed cultures, as provided by Protextin (Probiotics International, Somerset, UK) and other similar products, are available for horses. Human preparations may be used at five to seven times

TABLE 18-19. Dietary Guidelines: Reestablishment of Large Bowel Fermentation

Body weight: Adult, 450 kg (1000 lb)
Frequency of feeding: Divide into three feeds daily
Provision: Maintenance

Ingredient	Weight	Approx. Volume
Steam rolled barley/oats	2 kg (4.5 lb)	3.5 L (7 pints)
Alfalfa cubes*	3 kg (6.5 lb)	6 L (12 pints)
Wheat bran	400 g (1 lb)	2 L (4 pints)
Brewer's yeast	60 g (2 oz)	3 tablespoons
Dicalcium phosphate	30 g (1 oz)	1.5 tablespoons
Salt	30 g (1 oz)	1.5 tablespoons
Molasses	1 cup in 1 cup hot water mixed into feed	
Mixed into each before feeding		
Yogurt (natural)	100 mL (3 oz)	5 tablespoons
Alternatively, yogurt or a slurry of probiotic or *Lactobacillus acidophilus* mixture can be given over the tongue before feeding.		
Good-quality grass hay, or soluble fibers in beet pulp and soybean hulls, can be offered ad libitum to increase roughage mass for fermentation (see text).		

*Chopped alfalfa hay (lucerne chaff) may be added as an alternative.

TABLE 18-20. Dietary Guidelines: Low-Grade Diarrhea in Working Horses

Body weight: 450 kg (1000 lb)
Frequency of feeding: Divide into three equal feeds. Grass hay ad libitum to appetite
Provision: 30 min medium-intensity exercise daily

Ingredient	Weight	Approx. Volume
Rolled barley	3.5 kg (7.5 lb)	6.5 L (13 pints)
Cracked, or preferably extruded corn	2 kg (4.5 lb)	3 L (6 pints)
Soybean meal	450 g (1 lb)	3 cups (1½ pints)
Wheat—fine middlings (pollard) or flour	225 g (8 oz)	2 cups (1 pint)
Grated apples, red	6 apples	(Let stand for 1-3 hours after grating before mixing into feed)
Grass hay, chopped	900 g (2 lb)	6 L (12 pints)
Glucose powder (sweetener)	100 g (3.3 oz)	6 tablespoons
Salt	60 g (2 oz)	3 tablespoons
Lite salt	30 g (1 oz)	1.5 tablespoons
Calcium carbonate	60 g (2 oz)	3 tablespoons
Dicalcium phosphate	60 g (2 oz)	3 tablespoons
Vitamin E	1000 IU	
Commercial vitamin/mineral supplement		

The ration may be slightly dampened with warm water. Do not sweeten with molasses. If diarrhea persists, check fecal pH for hindgut acidosis, feces for parasite eggs at least 6 weeks after worming, and other bacteriologic tests.
Good quality grass hay ad libitum to appetite.

the human dose rate for a 450-kg (1000-lb) horse. These cultures may be given in each meal for 2 to 3 days initially, divided between two feeds, and continued for 4 to 5 days if response is slow.

Diarrhea in Working Horses

Occasionally, horses in training will develop a low-grade, chronic form of diarrhea on high-grain (starch) rations due to cecal and hindgut acidosis, resulting in a fecal pH of less than 6.4. Reducing the grain intake and providing more roughage may enable the horse to stay in training. Once the consistency of the feces has returned to normal, gradually change back to the normal racing diet. Substitution of up to 30% of the grain with fat as an energy source will help reduce the risk of starch overload into the large intestine. (See page 669 for substitution rates.) The product *Founderguard* (Vetsearch, Sydney, Australia), containing virginiamycin (1%) in a special release pelleted form, is available in Australia and New Zealand to prevent hindgut D-lactic acidosis in horses on high-grain diets.

Provide good-quality grass hay ad libitum overnight. Avoid feeding alfalfa hay or cubes initially, but these may be gradually reintroduced over 4 to 5 days replacing half the grass hay overnight as a choice. Calcium carbonate also should be reduced to 30 g (1 oz). Natural-flavored yogurt (90 ml [3 oz]) or a commercial *Lactobacillus acidophilus* mixture may be given by syringe over the tongue before the morning and evening feeds for 2 to 3 days if reduced hindgut fermentation is suspected by passage of undigested feed.

Guidelines for a diet to aid in the control of low-grade, persistent diarrhea are given in Table 18-20.

KEY POINT

The primary cause of low-grade diarrhea ("cow pat droppings") in racing and performance horses on high-grain diets is due to hindgut overload of soluble, highly fermentable carbohydrates from the small intestine into the cecum and large colon.

Production of excess D-lactic acid during fermentation increases motility of the large bowel and lowers fecal pH. In chronic cases, measurement of fecal pH relating to D-lactic acid and volatile fatty acid content can be carried out by a standardized method.

Using a 50-mL measure, add 50 mL of distilled

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or deionized water to a 50-g sample taken from the *inner central area of freshly passed feces.* Mix to dilute the feces in the water, then *immediately* immerse a pH probe into the suspension. Wait until the pH reading stabilizes to determine the pH. A pH reading below 6.4 indicates an acid hindgut and below 6.2, risk of hyperacidity leading to suppression and death of bacterial flora and onset of low-grade laminitis and foot soreness. Fecal pH can be increased by modifying the grain-to-roughage ratio, and in severe cases of chronic diarrhea, measures to recolonize the hindgut flora may be necessary.

Renal Disease

A short-term maintenance diet to restrict protein, calcium, and phosphorus intake should be considered for horses with renal disease, complemented by appropriate medication and acid-base maintenance.

KEY POINT

Limitation of calcium intake is necessary in renal patients to prevent hypercalcemia.

The diet should be based on good-quality cereal forage, such as grass hay or chopped cereal hay with a low protein content. The energy should be provided by a low-protein, low-calcium, energy-dense carbohydrate source such as corn (preferably extruded), cracked barley, or brown rice, or in part by fat. Extruded grain feeds are well digested in the small intestine and reduce nitrogen overload into the hindgut. Legume meals and hays, chopped alfalfa hay, or pellets should be avoided to limit protein and calcium intake. Wheat and rice bran and fine middlings (pollard) should be eliminated from renal diets because of their high phosphorus content.

In inappetent horses, divide the ration into four equal parts, add sufficient water to make a slurry, and administer by nasogastric tube every 6 hours until the horse regains appetite. A parenteral daily dose of B-group vitamins also may help to stimulate appetite and maintain vitality.

Guidelines for a diet for horses recovering from renal disease are given in Table 18-21.

TABLE 18-21. Dietary Guidelines: Renal Disease

Body weight: Adult, 450 kg (1000 lb)
Frequency of feeding: Divide into three equal feeds daily
Provision: Maintenance

Ingredient	Weight	Approx. Volume
Extruded corn	1.4 kg (3 lb)	3 L (6 pints)
Extruded or boiled barley (wet weight)	1.4 kg (3 lb)	2.5 L (5 pints)
Polyunsaturated oil	230 g (8 oz)	1 cup
Cereal chopped hay, pellets, or cubes	2 kg (4.5 lb)	11 L (22 pints)
Brewer's yeast	30 g (1 oz)	1.5 tablespoons
Molasses	1 cup in 1 cup water, mixed into feed	
Salt	20 g (0.6 oz)	1 tablespoon

Good-quality grass hay should be provided, approximately 2 kg, to appetite, avoiding high-protein alfalfa or legume hay mix, and fresh water available at all times. Do not add protein meals; maintain at 8-10% crude protein.

Hepatic Disease

The liver is the first organ to receive nutrients after absorption from the bowel lumen. Where reduced hepatic function is present, diets must provide energy and protein in addition to maintenance requirements to allow normal regenerative processes. There is no need to restrict protein below maintenance, but high-quality protein sources must be provided, based mainly on cereal grains and hays, to reduce hepatic overload with aromatic amino acids. Limited amounts of soybean meal, up to 50 g/100 kg (1 oz/100 lb) body weight, may be provided to ensure adequate protein intake.

KEY POINT

Although polyunsaturated oils are useful to boost energy intake, they should be avoided because fatty acid accumulation in damaged hepatocytes may occur as a result of reduced metabolic function.

A range of supplementary B-group vitamins, given either orally or parenterally, 5 g (1.5 teaspoons) of oral vitamin C, and 1000 IU of oral vitamin E are recommended. Small frequent feeds, preferably six times daily, are useful to avoid loading of the liver.

Because many horses with liver disease have reduced appetite, provision of 1 kg (2 lb) fresh grass, morning and evening, may improve interest in food and provide additional roughage without risking excessive energy intake. Avoid legume greenfeed or more than 30% of the roughage intake as alfalfa hay.

Dietary guidelines for horses with hepatic disease are given in Table 18-22.

Other Specialized Diets

LAMINITIS

The severity of laminitis (founder) varies from subclinical to low-grade clinical forms (with signs of reduced stride length and sore feet) to acute laminitis with collapse of the internal support structure of the hoof and pedal bone rotation. Studies indicate that 80% of laminitis in otherwise healthy horses and ponies is due to overload of soluble, rapidly fermentable carbohydrates from excess grain or lush pasture intake (see Chapter 4).

TABLE 18-22. Dietary Guidelines: Hepatic Disease

Body weight: Adult, 450 kg (1000 lb)
Frequency of feeding: Divided into six equal feeds daily. Good quality grass hay ad libitum to appetite
Provision: Maintenance

Ingredient	Weight	Approx. Volume
Extruded corn	1.4 g (3 lb)	1 L (2 pints)
Steam rolled or boiled barley	2 kg (4.5 lb)	3.5 L (7 pints)
Cereal chopped hay, pellets or cubes	2 kg (4.5 lb)	11 L (22 pints)
Soybean meal	250 g (8 oz)	1 cup
Molasses	1 cup in 1 cup warm water mixed into feed	
Dicalcium phosphate	30 g (1 oz)	1.5 tablespoons
Calcium carbonate	60 g (2 oz)	3 tablespoons
Salt	30 g (1 oz)	1.5 tablespoon
Vitamin C	10 g	2½ teaspoons
Vitamin E	1000 IU	—

Commercial B-group supplement (use in preference to brewer's yeast). Do not add fat as an energy source (see text). Access to good quality grass hay after each meal has been eaten and overnight.

KEY POINT

Horses and ponies susceptible or suffering from laminitis should not be allowed to graze lush pasture overnight because high levels of soluble sugars are contained in wilted plants after sundown.

Although there may be a clinical need to restrict the diet in cresty or overweight ponies and other acutely foundered horses, good-quality protein intake must be maintained to promote laminar repair, even if the energy intake is restricted. An additional good-quality protein source containing lysine, methionine, and a range of other essential amino acids, such as ½ cup (100 g or 3 oz) soybean meal, or ¾ cup (135 g or 4.5 oz) canola meal per 100 kg (220 lb) body weight must be provided on a grass hay diet. This extra protein must be provided for at least 3 months to assist laminar regrowth. Supplements of 100 mg zinc, 10,000 IU vitamin A, and 500 IU vitamin D per 100 kg (220 lb) body weight in a commercial vitamin and mineral mix are recommended during the 2- to 3-month recovery period after a laminitic crisis.

Because laminar bonding is strengthened by calcium and hoof regrowth and hardness is promoted by biotin, additional calcium as provided by 20 g (oz [1 tbsp]) dicalcium phosphate and 3 mg biotin per 100 kg (220 lb) of body weight are recommended.

In a diet containing more than 4 kg (9 lb) of alfalfa hay or cubes daily, additional calcium is not required, but benefit may be obtained from biotin supplementation.

GERIATRIC HORSES

Aged horses in retirement at pasture often lose condition during winter or summer when the pasture is sparse or dry, despite adequate supplementary hay. Health problems related to poor dentition, thyroid or pituitary adenomas, and reduced ability to digest fiber and protein and absorb calcium and phosphorus, as compared with young horses, also may result in weight loss and reduced appetite.

KEY POINT

Weight loss due to sickness or an inadequate diet is hard to regain in aged horses.

Old mares have been shown to have lower plasma vitamin C levels compared with younger horses.

In old horses unable to chew grains and long-stem hay effectively because of poor dentition, dampened commercial pelleted or extruded feeds with added vitamins and chopped hay (chaff) are convenient and will avoid problems such as "choke." A suggested diet for a geriatric horse is given in Table 18-23.

Extruded corn and other grains, or barley cooked by boiling for 10 minutes, will improve small intestinal digestion and utilization in old horses compared with crushed or rolled raw grains. Boiled barley (3.0 kg [6.5 lb] wet weight) can be added during cold weather or as a twice-weekly treat for a 450- to 500-kg (1000- to 1100-lb) aged horse. Additional calcium should be added to the ration of horses over 16 years of age because of less efficient uptake, by increasing dicalcium phosphate to 90 g (3 oz), or 5 tablespoons, daily.

TABLE 18-23. Dietary Guidelines: Geriatric Horse

Body weight: Adult, 450 kg (1000 lb)
Condition score: 5-6 (USA), 2-2.5 (Aust)
Frequency of feeding: Divide into two feeds daily, or one when good pasture is available
Provision: Maintenance plus 10%

Ingredient	Weight	Approx. Volume
Steam rolled barley or crushed oats	2 kg (4.5 lb)	4 L (8 pints)
Crushed or preferably extruded corn	2 kg (4.5 lb)	3 L (6 pints)
Soybean meal	250 g (8 oz)	1 cup
Alfalfa cubes*	1 kg (2 lb)	2 L (4 pints)
Dicalcium phosphate	30 g (1 oz)	1.5 tablespoons
Calcium carbonate	30 g (1 oz)	1.5 tablespoons
Salt	20 g (0.6 oz)	1 tablespoon
Molasses	1 cup in 2 cups warm water to soften feed; soak for 30-60 min before feeding; add only half volume of water if boiled barley is provided	

A commercial mineral/vitamin supplement may be added for horses in poor condition. Aged horses with poor teeth may not be able to chew long-stem hay. Provide dampened hay cubes or chopped hay (chaff) to appetite and access to green grazing if available.

*Chopped alfalfa hay (lucerne chaff) may be added by weight as an alternative.

KEY POINT

In old horses with concurrent renal disease, the calcium supplement, soybean, and alfalfa should be restricted to half the recommended amount. An equivalent amount of chopped grass hay should be added to replace the roughage contributed by the alfalfa cubes in this case.

In old mares, 5 g (1.5 teaspoons) daily of vitamin C (ascorbic acid) may be included in the ration. If extra roughage is provided, dampened chopped alfalfa or grass hays and alfalfa cubes are preferable in old horses with poor dentition, in which long-stem hay cannot be fully utilized. The ration can be divided into two feeds where grazing is restricted or alternatively given as a night feed where pasture is palatable and the horse is able to graze during the day. Alternatively, for a 450- kg (1000-lb) aged horse, a pelleted ration (3 Mcal [13 MJ]/kg of 10-12% crude protein) can be offered at the rate of 4 kg (9 lb) mixed into 2 kg (4.5 lb) alfalfa cubes dampened with 1 cup molasses in 2 cups warm water to soften the meal as an evening feed or divided into two feeds.

During cold weather or when the aged horse needs to gain body weight, the ration volume can be increased by 15 to 20% and adjusted to appetite and condition, and adequate shelter and a blanket covering provided.

KEY POINT

Where appetite is reduced or other internal disease limits the bulk or volume that can be consumed, the grain content can be decreased and vegetable oils added to increase energy density.

The general substitution rate is 1 cup (250 ml [8 oz]) vegetable oil as a replacement for 4 cups (1 L) pellets or 6 cups (1.5 L) crushed oats or 3 cups (750 mL) crushed corn.

When the grain content is reduced by the inclusion of oil, introduce oil in a step-wise increment over 10 to 14 days and add ½ cup (120 g or 4 oz) soybean meal (45% crude protein), or 1 cup (240 g or 8 oz) canola meal (36% crude protein) for each cup of oil added to replace protein removed as the grain content is reduced. Up to 1 cup of vegetable oil in each of two meals can be

mixed into the ration, with a corresponding stepwise reduction in grain content. Always ensure adequate roughage by providing additional alfalfa cubes to make up the volume of the ration or to appetite, particularly under cold conditions.

Note: In aged horses with hepatic disease, extruded grains to supply well-digested carbohydrates should be provided in preference to fat as an energy source.

FATTENING AN UNDERWEIGHT HORSE

The three most common causes of poor condition in horses are heavy parasite burdens, sharp-edged molar teeth and other dental problems, and inadequate quantity or poor quality of feeds. However, the single most common cause is undernutrition. Feeding times interrupted by vices such as aerophagia or "windsucking" can result in ill-thrift despite an adequate ration. An individual horse's likes and dislikes may affect appetite and feed acceptance. Position in the group pecking order in a herd of grazing horses can affect feed intake in those intimidated by aggression. Sudden withdrawal of high-energy rations when a horse is turned out from training can lead to significant weight loss within 7 to 10 days.

Once the underlying cause of weight loss, such as sharp-edged molar teeth or heavy internal parasite burden, is recognized and remedied, a stepwise increase in the quality and quantity of the ration, complemented by appropriate exercise, will assist recovery. A suggested diet to improve the condition of an emaciated horse is outlined in Table 18-24.

TABLE 18-24. Dietary Guidelines: Increasing Body Weight

Body weight: Adult, 400 kg (900 lb)
Condition score: Less than 2 (USA), less than 0.5 (Aust)
Underweight: Thin, 50-100 kg (100-220 lb) underweight
Frequency of feeding: Divide into three feeds for stabled horses, with supplementary hay; one feed daily in pastured horses, with supplementary hay if required
Provision: Maintenance plus 30% with daily light exercise

Ingredient	Weight	Approx. Volume
Rolled barley or whole oats	1.5 kg (3 lb)	3 L (6 pints)
Cracked, or preferably extruded corn	1 kg (2 lb)	1.5 L (3 pints)
Alfalfa cubes*	2 kg (4.5 lb)	6 L (12 pints)
Molasses	1 cup in 1 cup warm water mixed into feed if appetite poor	
Brewer's yeast or equivalent commercial vitamin/mineral supplement	60 g (2 oz)	3 tablespoons
Grass hay or alfalfa-grass hay mix	3 kg (6.5 lb) daily or 1 kg (2 lb) at each feed, ad libitum overnight to appetite	
Salt	40 g (1.3 oz)	2 tablespoons
Ad libitum alfalfa or grass hay to appetite		

*Chopped alfalfa hay (lucerne chaff) may be added by weight as an alternative.

KEY POINT

The increase in feed intake and condition should be incremental over a 6- to 8-week period to avoid metabolic and digestive problems and should be complemented by regular light exercise.

Where a horse has poor dentition, crushed or extruded grains or pellets and chopped hay should be provided as the base for the ration.

A repeat worming 3 weeks after the initial treatment will remove developing cyathostomes released from hypobiotic gut reservoirs. However, repeat worming within 4 to 7 days of the initial drench may be necessary in horses in which high *Strongylus* spp. fecal egg counts have contributed to the ill-thrift condition.

KEY POINT

Most poorly conditioned horses will be hungry, so bulky rations with adequate hay will help to keep them satisfied.

Pastured horses will exercise themselves during warmer weather. Provision of hay should be assessed in relation to available pasture. Light exercise for 10 minutes daily should be given to stabled or confined horses to improve overall health and fitness.

REDUCING THE WEIGHT OF A FAT HORSE

Dietary restriction and controlled exercise can be used to reduce body weight in an obese horse.

TABLE 18-25. Dietary Guidelines: Weight Reduction

Body weight: Adult, 600 kg (1300 lb)
Condition score: Above 8 (USA), above 4 (Aust)
Overweight: 50-150 kg (110-330 lb) too heavy
Frequency of feeding: Divide into three feeds daily in stabled horses to prevent boredom
Provision: Low energy, encourage fat catabolism

Ingredient	Weight	Approx. Volume
Alfalfa cubes, crushed*	3 kg (6.5 lb)	6 L (12 pints)
Oat hulls, or clean chopped straw	2 kg (4.5 lb)	11 L (22 pints)
Soybean meal	450 g (1 lb)	2.25 cups
Wheat bran	180 g (6 oz)	1 L (2 pints)
Brewer's yeast (or commercial B-group vitamin supplement)	60 g (2 oz)	3 tablespoons
Salt	30 g (1 oz)	1.5 tablespoons
Dicalcium phosphate	75 g (2.5 oz)	3.5 tablespoons
Molasses	1 cup in 1 cup warm water mixed into feed to reduce dust	
Overnight for stabled horses	500 g (1 lb) grass hay or 2 kg (4.5 lb) green feed	

*Chopped alfalfa hay (lucerne chaff) may be added by weight as an alternative.

Obesity is a common problem in horses on rations that exceed their energy and exercise needs. It is best to provide low-energy feeds and water with adequate protein, fat-soluble and B-group vitamins, minerals, and electrolytes combined with controlled exercise. A suggested diet to safely reduce excess weight in a horse is outlined in Table 18-25.

KEY POINT

Horses should not be denied feed, because ponies and some Thoroughbreds are particularly susceptible to hyperlipemia when starved or fasted for more than 8 to 12 hours. Overweight cresty ponies or any horse with laminitis must not be starved to reduce body weight.

Overweight mares should not be feed-restricted to reduce body weight before breeding; otherwise, their fertility and chances of conception may be reduced. Pregnant mares should not be placed on a diet to lose weight until they are at least 90 days pregnant to avoid increased risk of embryonic loss and abortion in early pregnancy. After this time, a planned stepwise reduction of feed and increase in light daily exercise will improve fitness and aid in reducing body condition.

In horses with access to good-quality pasture, restricting grazing to 30 minutes each morning and evening will help control energy intake, combined with a bulk roughage "filler" such as 250 g (8 oz) low-energy grass or cereal hay per 100 kg (220 lb) of body weight overnight to prevent boredom. Any weight-reduction program should include regular exercise, such as 10 to 20 minutes of daily aerobic work at the trot and loping canter under saddle or on the lunge. Horses should be confined to a bare yard (not a sand yard) or stabled in cold weather with unpalatable bedding of rice hulls or clean wood shavings.

A ration to provide half the maintenance energy, plus adequate protein, minerals, and vitamins, will assist in weight loss. Because horses are always hungry, especially those on a restricted diet, allowing access to even a grazed-out short pasture will encourage them to further overgraze the palatable plant species and increase risk of sand colic in sandy soil areas.

HORSES AT REST

Mature horses that have retired or are being rested at grass should be able to maintain body condition on good-quality pasture. A supplementary feed of good-quality alfalfa or cereal-mixed hay may be offered when pasture is sparse, short, or of poor feed value. A commercial ration or pelleted concentrate feed may be necessary when a horse

TABLE 18-26. Dietary Guidelines: Resting Horse

Body weight: Adult, 450 kg (1000 lb)
Condition score: 5-7 (USA), 2-3 (Aust)
Horse at rest on little pasture or confined
Frequency of feeding: One feed, hay other feed
Provision: Maintenance

Ingredient	Weight	Approx. Volume
Steam rolled barley or whole oats	2 kg (4.5 lb)	4 L (8 pints)
Alfalfa cubes*	2 kg (4.5 lb)	4 L (8 pints)
Dicalcium phosphate	30 g (1 oz)	1.5 tablespoons
Molasses	1 cup in 1 cup warm water mixed into feed	
Salt	30 g (1 oz)	1.5 tablespoons
Confined horses		
Grass hay (usually overnight)	3 kg (6.5 lb)	

*Chopped alfalfa hay (lucerne chaff) may be added by weight as an alternative.

is kept indoors for the winter period. Feeding guidelines for maintaining a horse at rest are given in Table 18-26.

Ad libitum good-quality grass hay should be provided to satisfy appetite rather than offering specific portions, particularly overnight to confined horses. Body weight and condition should be monitored and the ration adjusted accordingly. Confined horses should be given some light exercise by lungeing for 10 to 15 minutes or 30 minutes of walking on the lead daily to maintain vitality and appetite and reduce boredom.

BEHAVIORAL DISORDERS ASSOCIATED WITH FEEDING

Horses can develop a variety of behavioral disorders such as wood chewing, eating dirt, and aggressive behavior related to feeding. A selection of the common behavioral disorders, their predisposing causes, and measures for prevention are outlined in Table 18-27.

PLANTS POISONOUS TO HORSES

Many common weeds and native plants and certain pasture and some garden plants, plant byproducts, and bedding materials can be poisonous to horses. Clinical signs and severity range from acute poisoning within 6 to 12 hours to long-term cumulative toxicity that can result in debilitation and death. Most poisonous plants are normally not palatable to horses grazing on good pasture. Horses are careful and selective when grazing, and they normally will only sample and eat toxic plants when there is little else to graze or when pasture species are eaten out, dried off, or contaminated with debris or droppings. Plant poisoning is more common in newly introduced horses or young horses eager for new tastes, during drought, or when plants or trees overhang a bare lot or yard.

KEY POINT

Generally, the more succulent and green a plant or brightly colored its flower, the more poisonous it is to horses and livestock.

Plant poisoning should be suspected when a number of horses in a group of grazing horses exhibit similar clinical signs such as nervousness, incoordination, symptoms of photosensitivity, colic, or diarrhea, or are found dead.

KEY POINT

The toxic substances are in low concentrations in most plants. A horse normally has to consume 1 to 3% of its body weight of a dried plant or up to 5 to 10% of its body weight of a green plant before it will exhibit signs of toxicity and poisoning. However, as little as 100 g (3 oz), or less in a pony, of the dried leaves, flowers, or stems of Water Hemlock, Oleander, Yew, or Poinsettia can be acutely fatal to a horse. Once plant poisoning is suspected, horses should be removed from the pasture or measures taken to prevent further access to the plant or overhanging tree.

In hand-fed horses, allowing a horse to feed on trimmings and prunings from garden plants or providing lawn clippings as a green feed treat may lead to colic and plant poisoning. Stall bedding such as wood shavings can contain plant materials than can cause contact allergies on the skin or toxicity if eaten. The plants included in Table 18-28 are common to pastures, gardens, and graz-

Text continued on page 708

TABLE 18-27. Behavioral Disorders Associated with Feeding

Problem	Clinical Signs	Predisposing Causes	Dietary/Management Prevention
Behavioral eating habits	Scattering feed	Greediness	Change feed over 7-10 days
		Sudden feed changes	Provide deep feed tubs
		Boredom	Place welded reinforcing mesh 4 inch (100 mm) squares to fit snugly into feeder and laid over feed to prevent scattering
			Relieve boredom in stalls: hay overnight, exercise during day
	Aggressive feeding	Aggressive nature	Feed away from other horses
		Food withdrawal	Firm discipline
		Confinement and set feeding times	
	Impatience when feeding: kicking walls and tubs, pawing floor	Unknown	Feed horse first
		Impatient nature	Ensure regular feed times at set intervals in stabled horses
		Irregular feed times	
	Dropping of feed: quidding	Sharp-edged cheek teeth	Check molar teeth for sharp edges
		Dark feed area: feeder located in dark, solid-walled corner	Relocate feed tub to open area near window, aisle, doorway. Provide low-wattage night light in stall
			Ensure visual contact with other horses
	Rapid feed intake: gorging	Irregular feed times	Check molar teeth for sharp edges
		Aggressive nature	Place smooth stone or salt block in feeder to slow consumption or, alternatively, place welded reinforcing mesh 4 inch (100 mm) squares cut to fit snugly into feeder and laid over feed to slow intake
		Sharp-edged teeth	Feed chopped hay 30 min before concentrates
	Slow feed intake: selective eater, poor appetite	Teeth problems	Check molar teeth for sharp edges
		Distraction at feed times	
		Bulky feeds	Provide palatable feeds, reduce bulk of feeds to minimum safe levels of 0.5% roughage by body weight
		Unpalatable feeds	
		Nervous, pain	Treat underlying medical conditions
		Respiratory conditions	
		Early fast work	Avoid fast exercise too early in training: introduce fast work in increments over 2-3 weeks
			After 1 min rest to regain breathing and oxygenation, cool down exercise at steady trot to reduce muscle lactic acid accumulation

TABLE 18-27. Behavioral Disorders Associated with Feeding *Continued*

Problem	Clinical Signs	Predisposing Causes	Dietary/Management Prevention
Pica/abnormal eating habits	**Coprophagy: eating manure**	Boredom Confinement Low-fiber diets **Imbalanced diets**	Provide chopped hay overnight **Vitamin B_{12} therapy: 5000 µg IV once weekly for 3 weeks, 1000 µg fed daily for 10-14 days** **Vitamin/mineral supplement** **Drench with 500 mL cooking oil as a single dose** **Hang large plastic bottle as play toy in stall**
	Pica, crib biting, wood chewing	Boredom Confinement Lack of phosphorus Pelleted rations Low-fiber rations **Hindgut acidosis from high-grain diets**	Ensure correct Ca:P ratio **Cap rails, gates, etc. with metal sheeting** **Apply hot-tasting or bitter deterrent preparations to cribbed surfaces** Dilute pellets with chopped hay or moistened cubes
	Acrophagia windsucking, poor condition	Boredom Confinement Pleasurable sensation	**Windsucking neck straps: 3-inch-wide leather strap** Apply hot- or bitter-tasting deterrent preparations on target surfaces
	Tail and mane chewing	Young horses in cold weather	Apply hot-tasting or bitter deterrent preparations smeared onto outside tail hairs only
		Boredom in confined young horses	Hang large plastic bottle as play toy in stall Provide adequate roughage overnight, feed on time, three feeds daily
		Mineral deficiencies	Provide trace mineral supplement or lick block
	Dirt or sand eating	Boredom	Shift to new well-grassed pastures bimonthly
		Mineral deficiencies	Provide calcium and trace mineral supplement or lick block
		Confinement	Provide strong play toy when confined to yard Drench with mucilloids (400 g [about 1 lb]) or paraffin oil to remove sand Mix 60 g Montmorillonite clay (bentonite) into feed daily for 10-14 days
	Obesity/overeating	**Too much feed in relation to exercise**	Reduce ration Increase exercise Do not starve ponies (see text)

TABLE 18-28. Plants Poisonous to Horses

Garden Plants/Ornamental Shrubs and Trees Poisonous to Horses

Common Name	*Botanical Name*	*Locality*	*Toxic Compound*	*Poisonous Part*	*Amount*	*Onset of Poisoning*	*Symptoms*	*Table 18-29 Quick Reference Key for Clinical Signs*
Azalea or laurels	*Rhododendron* spp. *Kalmia* spp.	Common house plants Ornamental plant in Southern USA	Cardiac glycosides Gryandoxins and arbutin	Leaves	Plant trimmings	Excessive salivation Sudden death in 12-24 hours	Horse found dead after cardiac failure, nervous signs, arrhythmias	B, D, G
Bitterwood	*Quassia simarouba*	Central and South America	Unknown Possible allergic or	Irritant sap causes mouth blisters	Unknown	1-2 days	Salivation, vesicular dermatitis-like blister lesions on face	G
Buckeye or horse chestnuts	*Aesculus* spp.	Eastern and Southern States of USA	Aesculin glycoside	New growth leaves and nuts	Unknown	6-12 hours	Colic, muscle tremors, paralysis	C, B
Castor oil plant	*Ricinus communis*	Gardens, waste areas, river banks Tropical areas, South and Southeast USA	Phytotoxin Pyridine alkaloid (ricinine)	Seeds Byproducts in contaminated feeds	25 g (1 o/) Beans arc fatal 1-2% in feed	6-48 hours	Nervous signs, trembling, sweating, depression, diarrhea, tachycardia, colic, liver damage, kidney damage	B, A, C, D
Cestrum Daphne	*Cestrum* spp. *Daphne odora* *Daphne mezereum* (Mezereon)	Gardens Gardens, Woodlands in Europe	Alkaloids (Solanine) Coumarin	All parts, clippings Leaves, berries, bark	Unknown Plant trimmings	6-24 hours Fatal 24-36 hours	Diarrhea, nervous signs Prevents blood clotting, internal bleeding, death	A, B D
Delphiniums, larkspurs	*Delphinium* spp. (80 species)	Gardens and forests Western states	Ditterpenoid Alkaloids	All parts	1-2% body weight *D. barbeyi:* 0.5% body weight	Depends on amount eaten	Salivation, convulsions, death	B, G, D
Foxglove	*Digitalis purpurea*	Gardens, ornamental	Cardiac glycosides	All parts, especially seeds	Plant trimmings	Sudden death	Nervous signs, cardiac damage, found dead	B, D
Hemlock, spotted European hemlock	*Conium maculatum*	Gardens—general weed, early spring in waste areas	Pyridine alkaloid (Conine)	Green parts, hay is safe	2-2.5 kg (4-5 lb) fresh leaves	2 hours-2 days Rarely eaten by horses	Nervous signs, paralysis, convulsions, colic, death	B, C, D
Ivy or snake vine	*Tinospora smilacina*	Gardens, creeper on walls	Unknown	All parts, wilted clippings	Clippings in refuse	Depends on amount eaten	Photosensitivity-sunburn-type lesions, sudden death	F, D
Lantana	*Lantana camara*	Ornamental, Southern states of USA	Rehmannicacid (Lanladene)	Plants, especially fleshy parts and dark (lowers	Variable	Depends on amount eaten Animals stand in shade	Photosensitivity, sunburn, diarrhea, liver damage	F, A

Mistletoe	*Viscum album*	Trees in gardens	Possible atropine-likc compound	Leaves and stems	Unknown, possibly 1-2 kg *(2-45* lb)	Depends on amount eaten	Dilated pupils, salivation, incoordination, hypersensitivity	B, G
Monk's hood or aconite	*Aconitum* spp.	Gardens, forests Western states of USA	Diterpenoid alkaloid	All parts	Clippings in refuse	Sudden death	Salivation, convulsions, death	B, G, D
Oleander	*Nerium oleander*	Ornamental around stables Across Southern states of USA	Potent Cardiac glycoside (Oleandrin)	All parts, flowers	Wilted leaves less bitter, 15-30 g (0.5-1 oz) toxic, 30-40 leaves	12 hours fatal	Digestive upsets, cardiac damage, sudden death	C, D
Oaks—Gambels oak, shinnery oak	*Querus* spp.	Shrubs and trees, gardens, paddock trees	Tannins	Leaves and flower buds in spring, bark, acorns (green)	Unknown	Death in 24 hours, may live for 5-7 days	Depression, inappetance, colic, death	B, C, D
Parsley	*Petroselinum sativum*	Gardens	Furocoumarins	Leaves	Horses like taste	12-24 hours	Photosensitivity-sunburn-type lesions	F
Pigface (portulaca)	*Portulaca* spp.	Gardens and pasture as weed Australasia	Oxalate compound	Leaves and stems	Large amounts	2-4 days	Nervous signs, tetanic spasms, incoordination, death	B, D
Poinsettia	*Euphorbia pulcherrima*	Garden ornamental Australia	Irritant latex sap	Leaves and sap	Plant trimmings	Unknown	Diarrhea, convulsions, death. Irritant sap, blisters on nose	A. B, D
Poinsianna (coral tree)	*Erythrina* spp.	Gardens Native to Australasia	Unknown	Bark	Unknown (chewing bark)	12-24 hours	Nervous signs, death	B, D
Poppy	*Papaver* sp.	Gardens	Isoquinolinc alkaloids	Roots and leaves	Plant in refuse	12 hours	Dermatitis, blurred vision, nervous signs	B, F
Potato	*Solatium tuberosum*	Gardens and cultivated vegetable	Soianinc	Green tubers, whole potatoes	Unknown	Unknown	Nervous signs, colic. choke on whole potato	B, C
Privet hedge plant	*Ligustrum vulgare*	Temperate regions, abandoned home sites, gardens, hedges and shrubs	Ligusirin and other gut irritants	Leaves and fruits	Unknown	1-2 hours after ingestion	Gut irritants—diarrhea, colic and incoordination, weakness, convulsions, death	A, B, C, D
Prunus shrubs	*Prunus* spp.	Plant often in garden rubbish		Wilted leaves	Unknown	12 hours	Convulsions, diarrhea	A. B, D

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TABLE 18-28. Plants Poisonous to Horses *Continued*

Garden Plants/Ornamental Shrubs and Trees Poisonous to Horses

Common Name	*Botanical Name*	*Locality*	*Toxic Compound*	*Poisonous Part*	*Amount*	*Onset of Poisoning*	*Symptoms*	*Table 18-29 Quick Reference Key for Clinical Signs*
Rhubarb	*Rheum rhaponticum*	Garden vegetable	Chrysophanic acid, oxalates	Leaves	0.5-1 kg (1-2 lbs) leaves Do not feed leaves as treats	Few hours	Red colored urine, salivation, colic, diarrhea, staggers	C, A, B, G
Soursob, sorrel, shamrock	*Oxaiis* spp.	Gardens, wasteland, grazing land in fall	Oxalate	All parts wilted after spraying	Unknown	2-4 weeks	Calcium imbalance, kidney damage, gastroenteritis, diarrhea	A, B
Sugar gum	*Eucalyptus cladoxalyx*	In gardens and paddocks East coast of Australia	HCN glycoside (Prunasin)	Wilted leaves, clippings	Unknown	12-24 hours	Convulsions and sudden death	B, D
White cedar	*Melia azedarach*	Gardens east coast of Australia	Resinoids in wilted leaves and berries	Berries	Unknown	12-24 hours	Collapse and sudden death	B, D
Wisteria	*Wisteria sinense*	Gardens, wall creeper	Irritant juice	Seeds, pods, clippings	Garden refuse	6-24 hours	Colic, diarrhea	A, C
Yellow oleander (Be Still or Lucky Nut tree)	*Thevetia peruviana*	Garden ornamental Southern states	Cardiac glycosides (fhevetin)	Leaves, clippings	Small amount 25 g (1 oz) fatal	Sudden death	Digestive upsets, cardiac damage, sudden death	C, D
Yew tree, Japanese yew	*Taxus* spp. *Taxus cuspidata*	Widespread garden, ornamental, natural forest tree	Alkaloid—taxine cardiotoxic effects	Eating shade tree, trimmings in refuse, green or dry (not seed coat)	0.1-0.5% body weight, readily consumed	Sudden death 5 minutes	Usually found dead next to source. Trembling, incoordination, cardiac damage, diarrhea	B, A, D

Fodder Crops Potentially Poisonous

Alfalfa Blister beetle intoxication	*Medicago sativa*	Great Plains States and North of USA Blister beetles baled into hay mid to late summer	Cantharidin	Beetles crushed into hay	2-5 beetles produce colic	Stored hay can remain toxic for years. Onset time depends on dose.	Depression, loss of appetite, colic, shock, death	B, C, D
Alfalfa legumes and clovers	*Medicago sativa* *Trifolium* spp.	Wet, humid conditions	Mycotoxin Slaframine Alkaloid	Black or brown mold spots on legumes	Unknown	Several days	Profuse salivation, diarrhea, abortion in	G, A, abortion
Clovers medics (alsikc clover)	*Trifolium* spp. *Medicap* spp. *(Trifolium hybridum)*	Pasture legumes Widespread	UV sensitive pigments, estrogens	Plant flowers	Grazing spring flush	1-3 days	Photosensitivity-sunburn-type lesions, partial blindness, runny eyes, diarrhea, infertility in mares	F, A, infertility
Corn	*Zea mays*	Widespread Stored damp, sweating in bulk silo	Mold, *Fusarium moniliforme*	Moldy grain, wet harvest	10 ppm 80-100% corn contaminated	1-6 weeks	Incoordination, wandering, brain damage, liver failure,	B, liver failure, D
Ergot of Paspalum	*Paspalum* spp.	Grain	Ergot fungus *(Claviceps paspali)*	Seed heads, sticky black seeds	Mixed into feed	Variable	Hypersensitivity, muscle tremors, staggers, drooling, abortion in	B, G, abortion
Ergot of rye	*Secale cereale*	Grain	Fungal endophyte, liver-damaging saponins	Grain	Mixed into feed	Variable	Colics, abortion in mares	C, abortion
Kleingrass	*Panicum coloratum*	Pasture grass, Southwestern states of USA		Rapid growth pasture, hay	1-3% body weight	Relative to amount ingested	Depression, poor performance liver failure, jaundice with Dhotosensitivitv	B, F, liver failure
Kikuyu	*Pennisetum clandestinum*	Coastal, tropical and subtropical areas	Nitrates or oxalates	Highly fertilized, rapidly growing, pastures	Unknown Grazing 1.5% body weight Runners cause acute blockage	2-8 months in young horses and lactating mares	Calcium binding—lameness, epiphysitis, hopping gait, paralysis, fibrous runners may cause intestinal	B, H, C
Linseed (flax)	*Linum usitatissimum*	All areas when fed in horse's feed	Cyanogenic glycosides or nitrates	Seeds and wilted plants	Approx. 0.5-1 kg of unboiled seeds (seeds must be added to boiling water to destroy toxic compounds)	12-24 hours coma/death	Labored breathing, drooling, muscle tremors, staggers, coma, death	B, G, D
Lupins	*Lupinum* spp.	All areas when fed as grain	Alkaloid (rhomopsin)	Seed or mold toxin on stubble *(Note:* white seed not toxic)	Lupin stubble	Death in 2 days	Colic, jaundice, liver damage, animal lifts front legs high off the ground	C, E, H, D

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TABLE 18-28. Plants Poisonous to Horses *Continued*

Fodder Crops Potentially Poisonous								
Common Name	*Botanical Name*	*Locality*	*Toxic Compound*	*Poisonous Part*	*Amount*	*Onset of Poisoning*	*Symptoms*	*Table 18-29 Quick Reference Key for Clinical Signs*
Panic (green panic) Setaria Buffel grass Argentine grass Dallas grass	*Panicum* spp. *Setaria sphacelata* *Cenchrus ciliaris* *Paspalum* spp.	Subtropical areas pasture	Oxalates	Rapidly growing, fertilized pasture	Continuous ingestion	Relative to grazing mix and season	Oxalates bind up calcium, bone demineralization, nutritional secondary hyperparathyroidism, malabsorption causes skeletal deformities, big head	E, H, lameness
Perennial ryegrass	*Lolium perenne*	Widespread especially in temperate areas	Lolitrem released by alkaloid fungus in rapidly growing young shoots	Young shoots	1-3% body weight	1-2 weeks after exposure to toxic pasture	Staggers, incoordination (caused by endophyte fungus)	B
Phalaris	*Phalaris* sp.	Pasture grazing	Alkaloid	Rapidly growing young shoots after fire or drought	1-3% body weight	12-24 hours	Staggers, death	B, D
Singletary pea Wild winter pea Caly pea	*Lathyrus hirsutus*	Widespread forage and natural pasture	Unknown	Seeds in hay (harvest pea before seed heads appear)	Seed pods in hay	Few weeks to months	Stand with hind and front feet forward Hind paralysis—stringhalt movement	B, lameness stringhalt
Sorghum Sudan grass Johnson grass	*Sorghum* sp.	Southwest and eastern states of USA; high rainfall areas	Cyanogenic glycoside (low glycoside varieties now prevalent)	Hay/green feed	1-3% body weight	Depends on intake	Hind limb staggers, bladder inflammation and dribbling urine, scalding of skin, diarrhea, labored breathing, abortion	B, H, A, abortion
Tall fescue Wild tall fescue	*Festuca arundinacea*	Southwest states of USA; Australia	Claviceps mold toxin	Pasture and hay	15% of pregnant mares affected	Toxic during last 60 days of pregnancy	Extended pregnancy, lack of milk, still births, acute lameness, skin rot at fetlock	H, pregnancy and lactation problems

Algae: blue-green algae	*Anacystis cyanea* *Anabaena circinalis*, *Microcystis aeruginosa*	Widespread on dams and rivers	Polypeptide toxin, cyanobacteria produce hydrogen cyanide	Wilting and dead algae blooms, toxin in water	Variable degree of toxicity dependent on weather conditions	Usually rapid	Jaundice, photosensitization, tremors, weakness, colic, diarrhea, death	B, C, A, F, D
Avocado (Guatemalan variety only)	*Persea americana*	South America or where cultivated	Unknown	Green leaves and green fruit	Unknown (ripe fruit is safe)	Up to 48 hours relative to amount	Colic, diarrhea, edema ventral areas, edema of head, muscle damage	C, A, loss of milk in mares
Balsam Desert spurge	*Euphorbia eremophilia*		Cyanide compound or chemical irritant	Leaves and stem milk may be irritant	Unknown	Soon after ingestion, 2-6 hours	Colic, sudden death Photosensitivity reactions	C, F, I, blisters on mouth
Billy buttons	*Craspedia chrysantha*	East coast of Australia	Compound unknown (cardiac glycoside)	Dry plants	Unknown	12-24 hours	Temporary blindness, diarrhea, nervous signs	A, B
Birdsville indigo (Nine-leafed indigo)	*Indigofera linnaei*, *Indigofera dominii*	Sandy soils of intermittent water courses in central Australia, not present in North America	Antagonists (possibly nitrocompounds) to amino acid arginine, indospicine	Dry leaves most toxic	4-5 kg of fresh plant daily for at least 2 weeks Horses can be protected from toxin by feeding alfalfa (lucerne) chaff, peanut or cottonseed meal and gelatin	After 3 weeks continuous grazing of green plant, often when there is nothing else to eat	"Birdsville horse disease"—only affects horses (Australia), depression, paralysis of hind quarters, toe dragging, collapse when stressed, loss of condition, liver damage, convulsions and staggers	B, H
Black locust (false acacia)	*Robina pseudoacacia*	Eastern states of USA	Phytotoxin in bark is very toxic if ingested	Chewing bark	Very toxic	Soon after ingestion	Weakness and depression, hind paralysis, loss of appetite, colic and diarrhea	B. H, C, A
Black-berried nightshade	*Solarium nigrum*	East of the Rockies of USA	Alkaloid Solanine	All parts, especially green berries, sparse pastures	0.5-43 kg (1—10 lb)	Soon after ingestion	Depression and weakness, diarrhea and colic	B, C, A
Black walnut (tree)	*Juglans nigra*	Widely distributed in USA	Juglone	Fresh furniture shavings as bedding, eating bedding	Black shavings 25% of bedding	12-24 hours	Increased heart rate, swelling in legs (edema), lethargy and laminitis (founder)	B, laminitis
Bracken fern	*Pteridium aquilinum*, *P. esculentum*	Northwest to upper Midwest USA; many other countries	Thiaminase destroys vitamin B_1 (responsive to vitamin B_1 therapy)	Entire plant, green parts, especially new fronds in late summer. Leaves in sun-dried hay	5-7 kg (12-15 lb) daily; 30-50% of green pasture diet	Cumulative 30-60 days	Initial weight loss incoordination, front legs crossed, hindlimbs wide apart, tachycardia, staggering, trembling, head jerking, arching of back, collapse	A, B
Buckwheat	*Fagopyrum esculentum*	Widespread in agricultural areas	Fagopyrin	Photosensitive pigment	Unknown	Unknown	Photosensitivity-sunburn-like lesions	F
Buttercups	*Ranunculus* spp.	Widespread wet areas	Ranunculin glycoside	Green plants	Unknown	12-24 hours	Blistering of mouth, salivation, colic, diarrhea	G, C, A, blisters on mouth

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TABLE 18-28. Plants Poisonous to Horses *Continued*

Weeds and Other Plants

Common Name	*Botanical Name*	*Locality*	*Toxic Compound*	*Poisonous Part*	*Amount*	*Onset of Poisoning*	*Symptoms*	*Table 18-29 Quick Reference Key for Clinical Signs*
Cape tulip (one or two leafed Cape tulip)	*Homeria* spp.	Southern Australia temperate climates	Cardiac glycosides in the leaves and bulbs	Leaves and bulbs, also contaminant in meadow/grass hay	Unknown	Sudden death 12-24 hours	Nervous signs and diarrhea Sudden death	B, A, D
Capcwecd dandelion	*A rctotheca calendula*	Widespread, especially in coastal areas	Nitrates	Leaves are toxic	Unknown, takes up to *2-4* weeks	Sudden death possible, chronic poisoning Causes nerve damage	Nervous signs, muscle tremors, staggering, collapse May contribute to "Australian stringhalt" If horse moved to clean paddock, clinical signs decrease	B, H, Australian stringhalt
Choke cherry Wild black cherry	*Prunus virginiana*	Fairly widespread	Cyanogenic glycosides	Leaves and fruit, drought, frost, wilting or rapidly growing plants	Very toxic	Death: few minutes to 60 minutes	Clinical signs due to cyanide poisoning Trembling, collapse, respiratory arrest	B, D
Coffeeweed Coffee senna Wild coffee	*Cassia occidentalis*	Roadsides, waste areas Eastern states of USA	Alkaloids	Seeds most toxic	Unknown	Hours to several days	Gastric upset and muscle damage, diarrhea, and staggers Death due to cardiac failure	C, B, D
Cotton fireweed	*Senecio quadridentatus*	Widespread in coastal areas of east Australia	Pyrrolidine alkaloids in all parts	Contaminates hay, grazing green plants	Unknown	Cumulative buildup over 1-2 years	Liver damage, chronic scouring, loss of condition-sudden death	C, A, D
Crab's eyes Rosary pea Jequirity	*Abrus precatorius*	Dry grazing areas north Australia	Toxalbumin (Abrin)	Seeds	Unknown	Unknown	Colic, staggers, death	C, B, D

Cress (camel poison, buckbush)	*Gyrostemon australasicus*	Dry grazing areas north Australia	Compound unknown	Leaves and small pale flowers	Unknown	Unknown	Causes fluid buildup in the lungs, coughing, nasal discharge, foaming from nostrils, labored breathing, jaundice, then death	D, coughing, jaundice
Crofton weed	*Eupatorium adenophorum*	Temperate areas, especially coastal	Tremetol	Flowering plant in spring	Large amount of plant must be ingested	Gradual, usually over 2 months	Causes fluid buildup in lungs, horse coughs within a few weeks, has reduced exercise tolerance, labored breathing, depression, and then death Horses find plant palatable	D, labored breathing, coughing
Death camas	*Zigadenus nutallii*	West of Mississippi USA on sandy plains, foothills of Rockies	Steroidal glycosidal alkaloid	Grazing plant in sparse pasture in Spring	4.5 kg (10 lb) fatal	Hours to several days	Signs within several hours—depression, staggering, salivation, lower heart and respiratory rates Death in several days	B, G, D
Elderberry	*Sambucus* spp.	Ditches, streams, underground runners	Cardiac glycosides	All parts, especially succulent leaves	Unknown, rapid death	Several hours	Labored breathing, staggers, coma, death	B, D
False acacia (black locust)	*Robinia pseudoacacia*	Eastern and central Australia	Phytotoxin in bark is very toxic if ingested	Chewing bark	Very toxic	Soon after ingestion	Weakness and depression, hindlimb paralysis, loss of appetite, colic and diarrhea	B, H, C, A, hindlimb paralysis
Fat hen	*Chenopodium album*	Temperate areas Irri sated areas	Cyanogenic glycoside or nitrates	Leaves	Unknown, mixed in green feed	12-24 hours	Diarrhea when mixed into green chopped forage	A
Fiddleneck Yellow bun-weed Tar weed	*Amsinckia* spp.	Pacific coast USA	Pyrrolizidine alkaloids	Grazing or in hay, especially seeds	25-75 kg (50-150 lbs)	Cumulative over several weeks	Weight loss, colic, loss of appetite, "poor doers," drowsiness, staggers, circling, aimless walking, liver damage, jaundice, sweating, death	C, B, D, weight loss, jaundice

Table continued on following page

TABLE 18-28. Plants Poisonous to Horses *Continued*

Weeds and Other Plants								
Common Name	*Botanical Name*	*Locality*	*Toxic Compound*	*Poisonous Part*	*Amount*	*Onset of Poisoning*	*Symptoms*	*Table 18-29 Quick Reference Key for Clinical Signs*
Field bindwood Convolvulus Morning glory	*Convolvulus arvensis*	Widespread in USA	Tropane alkaloids; atropine-like action	All parts of the plant	Unknown	Unknown	Colic, flatulence, dilated pupils, tachycardia	C, dilated pupils, tachycardia
Fircweed	*Senecio madagasca riensis*	Australasia	Pyrrolizidine alkaloids	Leaves, stems, and flowers	Unknown	Cumulative, delayed death in 6-48 hours	Excessive salivation, dehydration, muscle weakness, collapse Liver and kidney damage—brown urine	G, B, E, D, brown urine, jaundice
Flatweed catsear	*Hypochaeris radicata*	Temperate, especially coastal areas	Toxic compound unknown	Whole plant, especially leaves	Unknown, ingestion of succulent plant after rains	Gradual 2-3 weeks after rains	May contribute to "Australian stringhalt" condition—exaggc rated hindleg "goose-stepping action"	B, H, Australian stringhalt
Green cestrum	*Cestrum parqui*	Coastal areas of Southern Australia	Solanine-type alkaloid which is toxic all year round	All of plant	Unknown	2–6 months or sudden death, calcinosis—hypercalcemia	Weight loss, lameness increases humped up in back and short steps Lies down frequently Excitement, diarrhea due to irritation of the bowel	A, H, D, lameness, weight loss
Hoary alyssum	*Berteroa incana*	Northern states of USA	Unknown	Green forage in dry pasture, baled in hay	Unknown	2-3 days after eating contaminated hay	Soft swellings in limb (edema), clinical signs subside in 2-4 days after withdrawal of hay	B, A, weight loss
Horse tail Mares tail Scouring rush	*Equisetum arvense*	Widely over northwest, northern, midwest states of USA, wet and cold areas	Thiaminase enzyme similar to bracken fern	Plant in dried hay	Unknown	Cumulative	Initial weight loss, incoordination, front legs crossed, hindlegs wide apart, tachycardia, staggering, trembling, head jerking, arching of back, collapse	B, A, weight loss, tachycardia

Hounds tongue	*Cynoglossum officinale*	Scattered	Pyrrolizidine alkaloids	Leaves	Unknown	Cumulative: several weeks to months	Weight loss, drowsiness, staggers, aimless walking, jaundice, liver damage	A, B, E, weight loss, jaundice
Inkwced Poke weed	*Phytolacca* spp. *Phytolacca americana*	Coastal and northern Queensland, Australia, Eastern and Southern USA	Oxalic acid, saponins, alkaloids in all parts, the roots are the most toxic	All pans, especially roots	Unknown	2-3 hours	Gastric irritation, colic. bloody diarrhea, labored breathing, convulsions, death	A, B, C, D, hemorrhage, diarrhea, labored breathing
Iron wood Cooktown ironwood	*Erythrophleum chlorostachys*	Northern Australia	Cardiotoxic alkaloids	Leaves, twigs and pods of tree	28-60 g of dried leaves causes death	Sudden death	Diarrhea, depression, blindness, death One of the most dangerous toxic plants in northern Australia	A, B, D
Locoweed Milk vetch	*Astragalus* spp. *Oxytropis* spp.	*Astragalus* spp.— western states of USA *Oxytropis* spp.— central states of USA, east coast of Australia	Alkaloid locoine {*Astragalus* can accumulate selenium)	Whole plant throughout year	30% body weight	6 weeks	Early stages— incoordination and visual disturbances; dangerous to ride, rearing and failing back on haunches, circling, wild behavior if excited, convulsions and death	B, D, abnormal gait, convulsions
Marshmallow Small-flowered marshmallow	*Malva parviflora*	Australia	Malvalic acid or nitrates	Seeds	Unknown	Several days to weeks; death in hours-5 days	Staggers, shoulder twitching, sweating, blood-tinged froth at nostrils, convulsions, cvanosis, death	B, H, D
Mexican poppy	*Argemone* spp.	East coast of Australia	Isoquinoline alkaloids	Stems, leaves, fruits and seeds	Seeds in grain may swab for opioids in racehorses	6-24 hours	Colic—often contaminates hay fed to horses, is also a common weed in grain crops	C
Milkweed	*Asclepias* spp.	Widespread in pastures in USA	Cardiac glycosides	Plants only grazed in drought or overstocking	0.5-2.0% body weight	Rapid death	Horses found dead due to cardiac block	D

Table continued on following page

TABLE 18-28. Plants Poisonous to Horses *Continued*

Weeds and Other Plants								
Common Name	*Botanical Name*	*Locality*	*Toxic Compound*	*Poisonous Part*	*Amount*	*Onset of Poisoning*	*Symptoms*	*Table 18-29 Quick Reference Key for Clinical Signs*
Mimosa West Indian lead tree, virvi, cowbush	*Leucaena leucocephala*	Tropical states, West Indies, South Africa, North Australia	Minosine arnino-like compound	Whole plant, selenium accumulator	Unknown	Unknown, selenium toxicosis	Hair loss, abortion, hoof shedding, lameness, teratogenic	H, hair loss, abortion
Mountain laurel	*Kalmia latifolia*	Eastern and southern states of USA	Grayanotoxins and glycosides	All parts of plant	Plant trimmings	12-24 hours	Colic, salivation, frequent defecation, staggers	C, B, G, salivation, defecation
Nardoo	*Marsilea drummondi*	Northwestern New South Wales, Australia	Unknown, possibly a thiaminase; causes vitamin B_1 deficiency	Green plant especially toxic during wet season	Unknown	Very high content of tlhiminase in lush fronds; toxic if eaten in large amounts	Nervous signs—incoordination, stumbling, falling, excitement, whinnying, yawning, pawing, partial blindless, recovery or death	B, D, yawning, blindness
Onion grass	*Romulea longifolia*	Widespread weed in Australia	Unknown physical obstruction	All parts of fibrous plant	Unknown	Death within 24 hours	Fibrous balls obstruct horse's large intestine	I, D
Pituri	*Duboisia hopwoodi*	Arid central and western Australia	Nicotine alkaloids	Ixaves, contaminates chopped hay (chaff)	Unknown	Unknown	Colic, diarrhea, staggers, tremors, convulsions	A, B, C
Podgrass Arrowgrass Goosegrass	*Triglochin* spp.	Elevated wet areas of USA	Cyanide compounds	Leaves	Contaminates hay	Rapid death in 2-6 hours	Sudden death, initial labored breathing, muscle tremors, ataxia	B,D
Poison hemlock (also see Hemlock)	*Conium maculatum*	Widespread	Piperidine, alkaloids	Green leaves or roots Safe when dried in hay	2-2 1/2 kg (4-5 lb) fresh leaves fatal	2 hours-2 days Fatal in 5-10 hours	Excitement then depression, lack of awareness, trembling, collapse, death	B, D
Pokeweed Poke	*Phytolacca americana*	Rich soils, new pastures Eastern states of USA	Oxalic acid, alkaloids, saponins, glycoproteins	All parts, roots most toxic	Unknown	2-3 hours	Gastric irritation, colic, bloody diarrhea, difficult breathing, convulsions, and death	A, B, C, D, hemorrhage, diarrhea, labored breathing

Ragwort Stinking Willie Common groundsel Bitterweed	*Senecio* spp. *Senecio jacobaea* *Senecio vulgaris* *Senecio* spp.	South Pacific Northwest USA	Pyrrolizidine alkaloids	All parts (can contaminate hay)	40-70 kg (50-150 lb)	Cumulative several weeks	Weight loss, colic, loss of appetite, drowsiness Staggers, circling, aimless walking Liver damage, jaundice, sweating, death	B, C. D, weight loss, jaundice
Rattleweed Rattle box Rattlepods	*Crotalaria* spp.	South and west to Texas USA; northern Australia	Pyrrolizidine alkaloids	Leaves (can contaminate hay), fibrous stems	Unknown	Cumulative several weeks	Weight loss, colic, loss of appetite, fibrous stems can obstruct cecum, compulsive wandering, staggering, yawning Lung and liver damage, jaundice, death	B, C, D, weight loss, jaundice, labored breathing
Rayless goldenrod (Jimmy or burrow weed)	*Haplopappus* sp.	Southwest USA in open grazing	Tremetol toxin	Whole fresh plant or baled in hay	0.5-2.0% body weight lethal, cumulative toxin	Depends on dose, sudden death	Stiffness in gait, incoordination, sluggishness, sweating, throat paralysis or sudden death	B, H, D, patchy sweating
Red maple	*Acer rubrum*	Eastern States but also Texas and Rockies USA	Unknown, possible oxidant, summer and fall	Wilted red maple leaves in fall, or trimmed branches	1.5 g/kg body weight	Few hours to 5 days	Loss of appetite, depression, weakness, anemia, jaundice and respiratory distress, coma prior to death	B, D. jaundice, loss of appetite, abortion
Russian knapweed	*Centaurea repens* *Acroptilon repens*	Pacific coast to Colorado, Rocky Mountain states USA	Alkaloid	Green or dried plant	59-71% body weight, more toxic than yellow star thistle	Cumulative 30 days, causes nigropallidal encephalomalacia	Brain damage—inability to hold or chew food or drink, wandering, swelling in face, death by starvation or inhaling food or water	B, D, wandering, face swelling
Salvation Jane Paterson's curse	*Echium lycopsis*	California localized areas USA, Australia	Pyrrolizidine alkaloids	All parts (purple flower—rosette young plant)	Unknown	Cumulative, several weeks to 2-3 years exposure	Weight loss, colic, loss of appetite, poor doers, compulsive wandering, staggering, yawning, lung and liver damage, jaundice, death	B, C, E, D
Serviceberry Saskatoon berry	*Amelanchier alnifolia*	High-altitude slopes, Rocky Mountains	Cardiac glycosides	Leaves	Unknown	Few hours, rapid death	Labored breathing, staggers, coma, death	B, D
Shepherd's purse	*Capsella bursapastoris*	Grazing areas	Unknown	Fibrous and mature plant	Gut obstruction	Unknown	Mechanical obstruction of bowel by fibrous balls of material	Bowel obstruction, C
Sleepy grass	*Stipa* spp. *Stipa viridula*	New Mexico	Oral abrasions, hypnotic drug	Rough plant		Lacerates mouth Hypnosis	Mouth lesions—loss of appetite, drowsiness	I, G, B (hypnosis)

Table continued on following page

TABLE 18-28. Plants Poisonous to Horses *Continued*

Weeds and Other Plants

Common Name	*Botanical Name*	*Locality*	*Toxic Compound*	*Poisonous Part*	*Amount*	*Onset of Poisoning*	*Symptoms*	*Table 18-29 Quick Reference Key for Clinical Signs*
St. John's wort Klamath weed	*Hypericum perforatum*	Pacific and Atlantic coast southeast of USA; Australia	Photosensitizing agent Hypericin	Leaves and flowers, lush green plants	Large amounts	24 hours to 5 days	Sunburn-type lesions. Horses with white areas of skin most affected—skin peels or rots off	F
Swainsonia Smooth darling pea	*Swainsona galegifolia*	Arid areas of Australia	Alkaloid: swainsonine	All parts of plant	Possible addictive grazing	Up to 5 weeks to develop after daily feeding	Characteristic "star gazing," staggering, loss of condition. Horse recovers after 6 weeks if removed from source	B, star gazing
Thorn apple (jimson weed)	*Datura stramonium*	Temperate areas of Australia, USA, New Zealand	Alkaloid: solanine	All parts of plant	Seeds in grain	7-17 days	Dilation of pupils, "blindness," dry nose, constipation, tachycardia, thirst, paralysis, coma, death	B, D, dilated pupils, blindness
Variegated thistle	*Silybum marianum*	Coastal and inland areas of Australia	Nitrate	All parts of plant, especially when young, wilted, or in wet hay	Nitrate increases after herbicide spray	12-24 hours	Colic, diarrhea, especially when as a contaminant in hay	A, C
Vetch (locoweed) Milk vetch Woody aster Golden weeds Prince's plume	*Astragalus* spp. *Xylorrhiza* spp. *Haplopappus* spp. *Stanleya* spp.	Mississippi to western slope of Rockies	Accumulate selenium, teratogenic	Fresh plants Dry plants in hay	Depends on selenium level	Addictive: acute, 6-24 hr; subacute, days to several weeks; chronic, weeks to months; teratogenic	Acute: fatal, depression, diarrhea, respiratory failure. Subacute: blind staggers, blindness, weakness, paralysis. Chronic: alkali disease, weight loss, poor appetite, loss of hair from mane and tail. Coronary separation, cracked and broken away hooves—lameness	B, A, H, D, weight loss, hoof damage

Water hemlock Cowbane	*Cicuta virosa*	Wet areas, ditches—upper midwest to Pacific	Cicutoxin resin	Leaves in spring Dried leaves in hay less toxic Roots extremely toxic	250 g (8 oz) of roots are fatal	10-60 minutes, rapid death	Nervous stimulation, muscle tremors in neck, fear Incoordination, labored breathing, collapse, convulsions, death	B, D, labored breathing
White snakeroot (snakeroot or richweed)	*Eupatorium rugosum*	Midwest south eastern states in wooded pastures—late summer	Tremetol toxin	Grazing fresh stalks above snow, or baled in hay	1-4.5 kg (2-10 lb) 0.5-2.0% body weight lethal	Depends on dose—sudden death	Trembling, sluggishness, stiffness in gait, incoordination, sweating, throat paralysis or death	B, H, D, sweating
Whitewood (west whitewood)	*Atalaya hemiglauca*	Eastern Australia	Toxic compound unknown	Leaves and fruit	150-240 g	12-24 hours, rapid death	Colic, muscle weakness, collapse Death due to cardiac damage	C, B, D
Wild blue flax	*Linum* spp.	Widely distributed USA	Cyanogenic glycosides	Young succulent plants	Unknown	6-24 hours, rapid death	Labored breathing, drooling, staggers, coma, death	B, D
Wild jasmine (day-blooming jessamine)	*Cestrum diumuin*	Wasteland, Texas and Florida of USA	Steroidal, vitamin D-like compound	Toxic all year round	Unknown	2-6 months, calcinosis—hypercalcemia	Weight loss, lameness increases humped up in back and short steps Lies down frequently	H, weight loss, humped back
Wild tobacco	*Nicotiana* spp.	Widespread	Alkaloids, teratogenic	Leaves and seed heads	Unknown	Unknown	Staggering, coma, death; teratogenic	B, D
Yellow burr weed Yellow forget-me-not	*Amsinckia* spp.	Coastal areas of Australia	Pyrrolizidine alkaloids	All parts, ingested during grazing or in hay	25-65 kg (60-140 lb)	Cumulative over several weeks	Weight loss, colic, loss of appetite, drowsiness, staggers, circling aimlessly when walking Liver damage, jaundice, sweating, death	C, E, D, weight loss, jaundice
Yellow star thistle St. Bamaby's thistle Star thistle	*Centaurea solstitialis*	California USA and east and southern Australia	Alkaloid	Green or dried plant, particularly if on green forage	86-200% body weight	Cumulative over 3-11 weeks, causes nigropallidal encephalomalacia	Brain damage—inability to hold or chew food or drink Swelling in face Death by starvation or inhaling food or water	B, D, face swelling
Zamia palm	*Cycas* spp.	Northern Australia	Glycosides	Seeds	Unknown	Unknown	Paralysis, staggers, liver damage	B, E, jaundice

TABLE 18-29. Major Clinical Signs of Plant Poisoning in Horses

Category	Major Clinical Signs
A	Diarrhea, digestive upset, physical irritant effect in horses
B	Brain damage, nervous signs, staggers
C	Colic and gut pain
D	Death
E	Cumulative poisoning
F	Photosensitivity and sunburn-like lesions (photodermatitis)
G	Salivation and slobbering
H	Lameness
I	Mechanical injury to mouth

The categorized clinical signs can be used to identify plants listed in Table 18-28 as possible causes of poisoning in horses. Some plants cause a number of clinical signs, with the major sign listed first in Table 18-28.

ing areas of North America, Europe, and the Southern Hemisphere. It is not an exhaustive list, and certain weeds, trees, and native plants not included may be known locally as causing poisoning in horses. A detailed description of plants in the Northern Hemisphere reported to be poisonous to horses is provided by Lewis (1995), and an overview of plants associated with poisoning in Europe is provided by Frape (1997) and in Australasia by Kohnke (1998).

The major clinical signs associated with each poisoning or toxic reaction are listed in Table 18-29. Table 18-28 lists the common names of plants that have been reported to be toxic to horses in alphabetical order in each group. This handy cross-referencing system helps to reduce the repetition in listing poisonous plants that induce a number of clinical signs, and the plants can be conveniently identified using the categorized codes in Table 18-29. Plants found in North America that may cause selenium toxicity are listed in Table 18-30. A search of the pasture or horse holding areas can be carried out to locate, identify, and, if necessary, prohibit further access to the plant(s) or control them by using selective herbicides, trimming, or removal.

FURTHER READING

Clarke, L. L., Roberts, M. C, and Argenzio, R. A.: Feeding and digestive problems in horses: physiologic responses to a concentrated meal. *Vet. Clin. North Am. Equine Pract. Clin. Nutr.* 6:433, 1990.

Frape, D. L.: *Equine Nutrition and Feeding,* 2nd ed. Oxford, UK: Blackwell Science, 1997, pp. 347-352.

Kohnke, J. R.: *Feeding and Nutrition of Horses: The Making of a Champion.* Sydney: Vetsearch International, 1998, pp. 45-65, 171-201.

TABLE 18-30. Plants Causing Selenium Toxicity (Primarily Rocky Mountain and Great Plains Regions of USA, alkaline soil areas)

	Common Name (Botanical Name)	Clinical Signs
Plants that actively accumulate high concentrations of selenium on selenium soils. Obligate plants require selenium for growth—indicator plants of high selenium soils. Most not palatable to horses.	Golden weeds *(Haplopappus* spp.) Milk vetches *(Astragalus* spp.) Prince's plume *(Stanleya pinnata)* Woody aster *(Xylorrhiza glabriuscula)*	Selenium toxicity is manifested by defects of hooves, brittle hair, and fetal abnormalities.
Plants that will accumulate selenium on selenium soils. Secondary selenium accumulator plants do not require selenium but will accumulate it.	Asters *(Aster* spp.) Bastard toadflax *(Comandra pallida)* Beard tongue *(Penstemon* spp.) Broom weed Turpentine *Gutierrezia* spp. r Snake or match weed Gumweed *(Grindelia squarrosa)* Indian paintbrush *(Castilleja* spp.) Ironweed *(Sideranthus grindelioides)* Saltbush *(Atriplex* spp.)	

Data from Lewis, L.D.: *Equine Clinical Nutrition: Feeding and Care.* Philadelphia: Williams & Wilkins, 1995.

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CHAPTER 19

Therapy

Reuben J. Rose and David R. Hodgson

Sedation and Anesthesia

Christina Dart, David R. Hodgson, and Reuben J. Rose

Sedation and general anesthesia are part of the daily routine of equine practice. A wide variety of tranquilizers, sedatives, and anesthetics are available and may be administered alone or in combination. In this section we will not attempt to cover in detail all the drugs available for sedation and/or anesthesia or discuss complex aspects related to anesthesia. Rather, we provide an overview of the drugs commonly available and suitable to use in general practice to produce chemical restraint, analgesia, and anesthesia. Some pathophysiologic and technical aspects of equine anesthesia are also addressed.

MAKING THE DECISION FOR STANDING CHEMICAL RESTRAINT OR GENERAL ANESTHESIA

The decision whether to use standing chemical restraint or general anesthesia depends on

- Horse temperament
- Age and condition
- Type of procedure to be performed
- Facilities available

In most cases this decision is clear and often dictated by the procedure to be performed. Chemical restraint is suitable if recumbency is not necessary and is almost always sufficient for noninvasive, nonpainful diagnostic procedures (e.g., radiography) and for very short interventions associated with little pain.

Standing chemical restraint in conjunction with local anesthesia can allow selective surgical procedures such as standing castration and the suturing of lacerations. General anesthesia is used for procedures requiring recumbency and/or more complete immobilization (e.g., cast application) and for more extensive surgery.

KEY POINT

Whether standing chemical restraint or general anesthesia is chosen, always make sure facilities and surroundings are safe for both patient and personnel. Safety is particularly important during induction of and recovery from general anesthesia. If safety cannot be ensured in the field, transport to an appropriate facility may be a better choice.

SEDATIVES/TRANQUILIZERS

Sedatives and tranquilizers can be used alone or in combination to produce varying degrees of sedation (Table 19-1). Some of these drugs also have analgesic properties. Besides these desirable effects, all sedatives and tranquilizers have undesirable side effects (e.g., hypotension, ataxia).

KEY POINT

Most commonly used sedatives and tranquilizers have undesirable side effects, usually affecting cardiopulmonary function. Therefore, sedatives and tranquilizers should be used with caution (low dose) in old, debilitated, or sick horses.

TABLE 19-1. Intravenous Doses of Commonly Used Drugs for Sedation in Horses*

Drug	Dose Range (mg/kg)	Preanesthetic Dose (mg/kg)
Acepromazine	0.01-0.06	0.03-0.05
Xylazine	0.2-1.1	0.3-0.6
Detomidine	0.0025-0.02	0.0025-0.005
Romifidine	0.04-0.12	0.04
Morphine†	0.05-0.3	0.08
Butorphanol†	0.02-0.03	0.02
Meperidine†	1.0-4.0	IM

*Intramuscular doses are two to three times intravenous dose

†May not reliably produce sedation, may cause restlessness and excitation instead. Best administered with other sedatives/tranquilizers in the table.

Increasing the dose increases both the desirable and adverse effects. Excessive doses therefore should be avoided. Instead, combining different sedative/tranquilizers is recommended if desired effects cannot be obtained with a single drug. The effects obtained may vary between individual horses, and in excited horses, generally most sedatives are less effective.

KEY POINT

Horses that have received sedatives for standing chemical restraint and appear adequately or even deeply sedated may still strike and kick upon painful stimulation.

KEY POINT

The same drugs and drug combinations are used for both standing chemical restraint and preanesthetic sedation. Low doses have to be used for preanesthetic sedation to keep the undesirable effects to a minimum.

KEY POINT

Horses placed in stocks after being sedated for standing chemical restraint may support themselves by leaning with their neck against the front bar. This can lead to airway obstruction and/or decreased blood flow to the brain and cause the horse to collapse.

Acepromazine

EFFECTS AND CLINICAL USE

- Acepromazine is a phenothiazine derivative tranquilizer. When used at recommended doses, acepromazine produces tranquilization or mild sedation. Acepromazine has no analgesic properties. Because of the moderate sedative effects and the lack of analgesic effects, horses can be aroused relatively easily and react adversely to painful stimuli. Acepromazine often is used for preanesthetic medication and for situations where only mild sedation is necessary. It will calm nervous and "pushy" horses.
- Acepromazine produces peripheral vasodilation and hence hypotension. Therefore, it is contraindicated or should be used with caution in horses that are old, dehydrated, or have a history of recent hemorrhage (e.g., deep cuts involving great vessels).
- Respiratory function is minimally affected by acepromazine.
- Occasionally, acepromazine will have little sedative effect in a horse. Increasing the dose will not improve sedation but will worsen hypotension. Excessive doses of acepromazine may cause restlessness and induce hallucinatory effects.
- In stallions, acepromazine has been reported in a small number of cases to induce permanent prolapse of the penis. In geldings, penile prolapse has not been reported to be permanent and hence acepromazine can be useful when undertaking examination of the prepuce and penis.
- Acepromazine most commonly is given intravenously (IV) but can be given intramuscularly (IM), or subcutaneously (SC). Administration in a paste form also is possible. Onset of action is slow, taking 20 to 30 minutes for maximum effect, even after intravenous administration. The duration of action is 4 to 6 hours.
- Acepromazine can be combined with (α_2-adrenergic agonists (xylazine, detomidine) or opioids (butorphanol, morphine, meperidine) to achieve more pronounced sedation and/or analgesia. These combinations, especially acepromazine and α_2-adrenergic agonists, have to be given with caution (low doses) when used for preanesthetic sedation.

KEY POINT

- Indications: *for mild sedation without analgesia, preanesthetic sedation*
- Side effects: *hypotension, hypothermia, may*

cause permanent penile prolapse in stallions
Contraindications: *shock, dehydration, hemorrhage, use in stallions*
Other: *slow onset of action, long duration of action*

Adrenergic Agonists: Xylazine, Detomidine, and Romifidine

EFFECTS AND CLINICAL USE

- Xylazine, detomidine, and romifidine are α_2-adrenergic agonists that produce dose-dependent sedation, analgesia, and muscle relaxation. They can be used for standing chemical restraint or for preanesthetic sedation.
- Xylazine was the first of the α_2-adrenergic agonists produced to be used as a sedative for veterinary patients and since its availability to the equine practitioner in the early 1970s has gained great popularity. Detomidine and romifidine are two of the more recently developed α_2-adrenergic agonists and specifically marketed for the use in large animals, especially equine patients. To date, xylazine and detomidine have been used more commonly than romifidine.
- Compared with xylazine, detomidine is more potent and has a longer duration of action. Romifidine, in comparison with xylazine and detomidine, produces less sedation and ataxia but has a longer duration of action. These three drugs produce a number of changes in cardiopulmonary function, including
  - o Decrease in the heart rate and may produce bradyarrhythmias (i.e., second-degree atrioventricular block)
  - o Adverse effects on blood pressure; initially there is an increase followed by a marked decrease
  - o A decrease in cardiac contractility and hence cardiac output
  - o A decrease in respiratory rate
- The clinical indicators of the cardiopulmonary effects of these α_2-adrenergic agonists are for a horse to have slow sometimes irregular pulse, low respiratory rate, and grayish mucous membrane color.
- Xylazine, detomidine, and romifidine therefore should be used with caution in horses with bradyarrhythmias, in old horses, and in horses suffering from dehydration or shock.
- Xylazine and detomidine will increase urine production. This may make their use contraindicated in dehydrated horses and horses with urinary tract obstruction.

In cattle, when given during the last trimester of pregnancy, xylazine may induce premature labor. Although this side effect has not been reported in mares, caution should be used when administering the α_2-adrenergic agonists to mares in advanced stages of pregnancy.

KEY POINT
Xylazine, romifidine, and detomidine cause laryngeal relaxation, and therefore during endoscopic examination of the larynx, assessment of laryngeal asymmetry and dysfunction will be difficult.

Despite the undesirable cardiopulmonary effects of xylazine and detomidine, they remain one of the most frequently used analgesic drugs for pain associated with colic. They provide good visceral analgesia without which more detailed assessment and examination, including rectal examination, may not be possible. Compared with detomidine, xylazine has the advantage that the analgesia is short lived and therefore will not mask clinical signs, which are used to assess progress.

Xylazine often is used as a preanesthetic before surgery for colic. There is a possibility that xylazine may worsen hypotension caused by the release of intestinal endotoxins into the systemic circulation, such as occurs during surgical correction of twisted or strangulated intestine.

In some horses, profuse sweating may occur after administration either of xylazine or detomidine.

Xylazine, detomidine, and romifidine may be administered IV, IM, or SC. Onset of peak effect is within 3 to 5 minutes following IV administration and within 5 to 15 minutes following IM or SC administration. Duration of sedation and analgesia is around 30 to 40 minutes for xylazine and 90 to 120 minutes for detomidine.

Both xylazine and detomidine can be administered epidurally to provide excellent, even surgical analgesia with no (xylazine) or reduced (detomidine) sedative and systemic side effects compared with IM or IV administration.

Xylazine and detomidine have been combined with acepromazine or opioids (morphine, butorphanol, meperidine) to enhance sedative and/or analgesic effects.

The effects of xylazine, detomidine, and romifidine can be reversed with α_2-antagonists such as yohimbine (0.05-0.125 mg/kg IV) or tolazoline (1.0-4.0 mg/kg IV).

KEY POINT

- Indications: *Mild to deep sedation and/or analgesia of relatively rapid onset, colic pain, preanesthetic sedation*
- Side effects: *hypotension, ataxia*
- Contraindications: *Bradycardia, dehydration, shock*
- Other: *Effects can be reversed*

Opioids: Morphine, Butorphanoi, and Meperidine

EFFECTS AND CLINICAL USE

- Morphine, butorphanoi, and meperidine are the principally used opioids in equine practice. Opioids consist of a large variety of naturally occurring, semisynthetic, and synthetic compounds. Included are morphine, butorphanoi, and meperidine. Butorphanoi most commonly is used in equine practice. Opioids bind with specific receptors in the brain and spinal cord. Morphine and meperidine exert agonistic effects and butorphanoi agonistic and antagonistic effects on these receptors.
- Opioids are effective analgesics, and depending on the species, they also have either sedative or excitatory effects. Unfortunately, horses are susceptible to the excitatory effects of opioids, which include restlessness and pacing, with horses generally showing increased activity of varying degrees. In the horse, therefore, opioids are almost always used in combination with a tranquilizer (acepromazine) or sedative (xylazine, detomidine) to minimize these excitatory effects. Often these combinations produce good sedation and analgesia (neuroleptanalgesia).
- The analgesic potency of butorphanoi is two to five times that of morphine, and the analgesic potency of meperidine is about one tenth that of morphine. The duration of action and the degree of sedation produced depend on the dose given, on whether or not pain is present, and the intensity of pain.
- Combinations of opioids and tranquilizers or sedatives can be used for standing chemical restraint and for preanesthetic sedation (Table 19-2).
- Apart from sedation and analgesia, opioids may produce bradycardia, hypotension, depressed respiration, decreased gastrointestinal motility, and sweating.
- Morphine and butorphanoi may be administered IV, IM, or SC. Meperidine should not be given TV because this may induce massive release of histamine, which can cause cardiovascular collapse.

TABLE 19-2. Intravenous* Doses of Common Drug Combinations for Sedation in Horses

Drugs Used	Standing Chemical Restraint (mg/kg)	Preanesthetic Sedation (mg/kg)
Acepromazine Butorphanol	0.04 0.08	0.02-0.04 0.02-0.03
Acepromazine Meperidine	0.04 0.6	—
Acepromazine Morphine	0.03 0.2-0.6	—
Acepromazine Xylazine	0.03 0.2-0.6	—
Xylazine Butorphanol	0.7 0.03	0.4-0.6 0.02-0.08
Xylazine Morphine	0.6 0.3-0.6	0.4-0.6 0.05-0.1
Detomidine Morphine	0.01 0.16-0.6	0.0025 0.05-0.1
Detomidine Morphine	0.01 0.03	0.0025 0.02-0.03

*Intramuscular doses are two to three times intravenous doses.

- Morphine and meperidine are controlled drugs, whereas butorphanol, depending on the country, may or may not be controlled. For controlled substances, purchase and use need to be accurately recorded and they must be stored in a secure area at all times.
- In case of complications or excessive side effects, morphine, meperidine, and butorphanol can be reversed with naloxone (5.0-20 μg/kg IV). This will, however, also reverse the analgesic effects of these drugs.

KEY POINT

- Indications: *Mild to deep sedation with analgesia, preanesthetic sedation, to enhance analgesia and sedation of other sedatives or tranquilizers*
- Side effects: *Excitement*
- Contraindication: *Should not be used alone to produce sedation, may instead cause excitation*
- Other: *Effects can be reversed*

GENERAL ANESTHESIA

In equine practice, general anesthesia is used for both noninvasive diagnostic procedures and for surgical procedures. General anesthesia ideally should produce a state of unconsciousness (hypnosis), immobilization, muscle relaxation (hyporeflexia), and analgesia. The drug(s) used for general anesthesia should permit rapid and smooth induction and recovery, should be easily controllable, and should have few adverse effects. Of the anesthetic drugs commonly used in horses, few have analgesic effects. Although unconsciousness will avoid conscious perception of pain and create suitable surgical conditions, biochemical mechanisms responsible for the generation of pain will still run their course. Once anesthesia is discontinued, the horse will be able to perceive pain and respond accordingly. For painful procedures, analgesia can be improved by the concomitant use of local anesthetics and/or administration of analgesics pre- and postoperatively.

Due to size and temperament, induction of general anesthesia in adult horses most commonly is done using intravenous anesthetics, whereas maintenance of general anesthesia is accomplished using either IV or inhalation anesthetics. For short-term anesthesia (less than 1 hour), IV anesthesia is suitable for both induction and maintenance (total IV anesthesia), whereas for anesthesia of longer duration (more than 1 hour), IV anesthetics are used for induction and inhalation anesthetics for maintenance.

KEY POINT

Preanesthetic medication (using tranquilizers, sedatives, analgesics) is recommended (see Tables 19-1 and 19-2). Sedation reduces stress in the patient and, therefore, makes for a smoother induction and anesthetic period in general. Premedications reduce the dose requirement for both induction and maintenance agents and hence lessen their adverse effects. Preanesthetic sedation can improve analgesia for surgical anesthesia and it will allow a smoother recovery.

INDUCTION OF ANESTHESIA

KEY POINT

Induction agents should be administered to effect and according to patient need. Aspects such as familiarity with a particular induction regimen, suitability of that induction regimen for a given induction facility (e.g., field, theater room floor, against induction table), adequate patient premedication, and availability of assisting personnel need to be considered to ensure a successful and safe induction.

Barbiturates: Thiopentone and Thiamylal

EFFECTS AND CLINICAL USE

- The thiobarbiturates, thiopentone and thiamylal, are ultra-short-acting barbiturates. They are very similar, but thiamylal is slightly more potent (1.5 times) than thiopentone and hence more toxic. Onset of action is rapid. Following the rapid administration of a recommended induction dose, anesthesia occurs in 20 to 30 seconds and recovery occurs in 10 to 20 minutes. Emergence from anesthesia depends on shift of the drugs from the brain to lean body tissues. Thiopentone and thiamylal, and barbiturates in general, produce little analgesia. Thiobarbiturates produce significant cardiovascular depression when administered as a bolus or in large doses. The cardiovascular effects include tachycardia, tachyarrhythmias and bradyarrhythmias (ventricular and atrial origin), and initial hypertension, followed by hypotension if top up doses are given. Thiobarbiturates depress respiration, which is more severe following very rapid administration and when large doses are given.
- The potency of thiobarbiturates is increased in acidotic patients.
- The thiobarbitutates readily cross the placental barrier and into the fetal circulation. Doses of barbiturates that do not produce anesthesia in the mother are sufficient to severely depress fetal respiration.
- Thiobarbiturates are metabolized in the liver. They should not be used in animals suffering from liver disease.
- Occasionally, the thiobarbiturates when used as the sole induction agent may cause some excitation during induction. This usually can be avoided by adequate preanesthetic sedation.

KEY POINT

Thiopentone and thiamylal should be administered with caution and at reduced doses in sick, debilitated, and depressed horses to reduce the incidence and severity of cardiac arrhythmias and respiratory depression and to avoid overdosage. Care needs to be taken in administration of the thiobarbiturates to fit racehorses, and thiopentone and thiamylal should not be used for anesthesia for cesarian section.

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DOSE AND ADMINISTRATION

- Thiopentone and thiamylal can be prepared in 2.5% to 10% solutions:
  - o 2.5%: add 200 mL of 0.9% sterile NaCl or sterile water for injection to 5 g of powder
  - o 5%: add 100 mL of 0.9% sterile NaCl or sterile water for injection to 5 g powder
  - o 10%: add 50 mL of 0.9% sterile NaCl or sterile water for injection to 5 g powder
  - o Solutions should be discarded after 3 days of nonrefrigerated storage. Solutions with precipitations present should not be used. The 10% solution is most practical for induction of anesthesia in adult horses weighing from 400 to 600 kg.
- **Dose: Thiopentone and thiamylal: 5-10 mg/kg IV** (following premedication)

KEY POINT

Thiopentone and thiamylal need to be given strictly IV Extravascular administration will cause tissue necrosis. In case of accidental extravascular injection, infiltration of the area with 200 to 400 mL of 0.9% NaCl and 2% lidocaine will minimize necrosis of tissue and associated pain. Thiobarbiturates should only be given through a properly placed indwelling IV catheter.

Nonbarbiturates: Ketamine, Guaifenesin, and Propofol

KETAMINE

Effects and Clinical Use

- Ketamine is a dissociative anesthetic. It causes disruption of central nervous system (CNS) function characterized by central nervous stimulation and muscle rigidity (catalepsy) and produces superficial analgesia. Ocular and swallowing reflexes usually are maintained. At higher doses, ketamine can cause convulsions and upon recovery may produce a state of fear and restlessness. Ketamine increases intraocular and intracranial pressures.
- Ketamine depresses respiration by causing an apneustic (breathholding) respiratory pattern. After ketamine administration, heart rate usually is increased and blood pressure is maintained.
- Onset of action is 60 to 90 seconds after intravenous administration, and the effects last 10 to 30 minutes, depending on the dose administered and the degree of surgical stimulation.

Dose and Administration

- Ketamine alone is unsuitable for anesthetic induction in the horse. When using ketamine, good premedication is imperative to minimize its excitatory effects and to provide muscle relaxation. Acepromazine or α_2-agonists, such as xylazine and detomidine, have proven to be suitable for premedication. In particular, the combinations of the α_2-agonists with ketamine usually cause a smooth induction. Ocular and swallowing reflexes are still maintained and analgesia is good.
- Ketamine also may be used in combination with guaifenesin for induction of anesthesia.
- **Dose: 1.0-2.2 mg/kg, depending on premedication and drug combination**

KEY POINT

Ketamine only should be used as an induction agent following heavy sedation or standard premedication in combination with guaifenesin. It is contraindicated in horses with a history of seizures, those suffering from recent head trauma, and horses presented with near perforating or perforating lesions of the cornea.

GUAIFENESIN, GUAFEN (GLYCERYL GUAIACOLATE)

Effects and Clinical Use

- Guaifenesin is used in anesthesia for its muscle-relaxing and sedative properties. It is a centrally acting muscle relaxant and produces relaxation of the skeletal muscles but does not affect function of the diaphragm and hence respiration. It relaxes laryngeal and pharyngeal muscles and, therefore, eases intubation of the trachea. Excessive doses may produce paradoxical muscle rigidity and an apneustic breathing pattern.
- Guaifenesin has few adverse effects on cardiopulmonary and other organ function. Although it crosses the placental barrier, it appears to have minimal effects on the fetus. Guaifenesin is not used alone, but it is a useful adjunct to other anesthetic drugs. It potentiates the CNS depressant and muscle-relaxing effects of the premedicants and induction agents.

Dose and Administration

- Guaifenesin has to be given strictly IV because of its tissue irritating effects. Guaifenesin is commercially available as a 10% solution in 500-mL vials. It also can be prepared by adding 50 g of guaifenesin and 50 g of dextrose to 0.5 or 1 L of warm sterile water, or 50 g of guaifenesin to 0.5 or 1 L of warm 5% dextrose. Homemade solutions have a limited shelf life and may precipitate if not kept warm. At the

10% concentration, guaifenesin may occasionally cause transient hemolysis and hemoglobinuria in horses. Should this be a concern, the 10% solution can be diluted to 5% with an equal volume of 5% dextrose or 0.9% NaCl.

- Depending on the premedication and induction regimen chosen, it may be sufficient to give guaifenesin at gravity flow. More commonly, however, it is administered at an increased speed by pressurizing the airphase in the container. The latter permits the beneficial effects of guaifenesin to be achieved within a reasonable time. Guaifenesin is administered until sedation and muscle relaxation (knuckling) are evident, which after standard premedication takes approximately half of the total calculated dose. The primary induction agent is then injected into the line to effect. Guaifenesin is continued until the horse is recumbent, which may or may not require the total dose of guaifenesin.
- **Dose: 50-100 mg/kg (0.5-1 mL/kg of 10% solution), given to effect**

KEY POINT

Thiopentone, thiamylal, and ketamine can be added to the guaifenesin solution and be given to effect for induction: 2 g thiopentone or thiamylal to 500 mL of 10% guaifenesin for a 450-kg premedicated horse; 1 g ketamine to 500 mL 10% guaifenesin for a 450-kg premedicated horse.

PROPOFOL

Clinical Effects and Use

- Propofol produces clinical effects similar to the thiobarbiturates. After intravenous injection, the onset of anesthesia is within 10 to 15 seconds and recovery is within minutes. Propofol has no analgesic effects. Propofol causes a dose-dependent decrease in arterial blood pressure but little change in heart rate. Propofol depresses respiration and causes initial periods of apnea similar to the thiobarbiturates. Other organ system effects are minimal. Emergence from anesthesia is due to both redistribution and rapid biotransformation in the liver. In contrast to the thiobarbiturates, propofol is noncumulative, and even after continuous administration, propofol allows a rapid almost complete recovery.

Dose and Administration

- Except for foals, propofol is not commonly used in the horse. This is due to the high cost of propofol, which prohibits its use, and the large volume necessary for induction of an adult horse.
- Premedication is recommended because propofol may cause some excitement during induction in the nonpremedicated patient.

"Triple-Drip" Anesthesia (Combination of Guaifenesin-Ketamine-Sedative)

BACKGROUND

KEY POINT

Where anesthesia is likely to be necessary for longer than 15 minutes but less than 1 hour, the combination of guaifenesin with ketamine and some form of sedative (usually xylazine or detomidine) is a reliable and effective form of anesthesia.

The advantages of the technique are that no equipment is necessary for administering gaseous anesthesia and recovery from anesthesia is excellent.

CLINICAL USE

- Ketamine (1 g) and xylazine (500 mg) is added to either a 5% solution (1 L) or 10% solution (500 mL) of guaifenesin. After induction of anesthesia with xylazine and ketamine, an infusion rate of approximately 2 mL/kg/h is used for the 5% solution and 1 mL/kg/h for the 10% solution.
- It is possible to replace the xylazine with detomidine at 5 mg/L of the 5% solution and 5 mg/500 mL if a 10% solution is used.
- Blood pressure is well maintained with less hypotension than with halothane anesthesia, and the recovery from anesthesia is generally smooth.

ADVANTAGES

- Prolonged anesthesia is possible with a minimum of anesthetic equipment.
- Smooth recovery from general anesthesia is usual.

DISADVANTAGES

- It is more difficult to control anesthetic depth than with gaseous anesthesia.
- Recovery from anesthesia can sometimes be prolonged.

MAINTENANCE OF ANESTHESIA

Short-Term Anesthesia (less than 1 hour)

For very short procedures, the duration of action of a standard induction may provide enough time

without the need for continuing the anesthesia. To extend the duration of action of the drug(s) used for induction:

1. Give small incremental top up doses of the anesthetic induction drug.
2. Give a continuous infusion of a combination of guaifenesin and the anesthetic induction drug (ketamine, thiopentone, thiamylal).

KEY POINT

Most IV anesthetics commonly used for equine anesthesia have cumulative effects. Therefore, the larger the total dose given, the more likely recovery will be delayed and of poor quality.

Total IV anesthesia, although usually used for procedures of short duration, is not necessarily safer than inhalation anesthesia of equal duration. Therefore, monitoring of the horse and any supportive care should be similar to that during general anesthesia. In addition, an endotracheal tube of appropriate size and a source of oxygen always should be available.

Long-Term Anesthesia (greater than 1 hour)

GENERAL CONSIDERATIONS

- Inhalation anesthetics should be the maintenance agents of choice if the duration of anesthesia is longer than 1 hour. In equine anesthesia, halothane and isoflurane are the commonly used inhalation anesthetics.
- Inhalation anesthetics are used in many species. They have the advantage of being relatively easily controlled and hence can be titrated to patient need. They produce unconsciousness and muscle relaxation but are poor analgesics. Both halothane and isoflurane depress respiratory and cardiovascular function in a dose-dependent manner, and they also affect other organ system function. Hypotension and respiratory depression are common problems during inhalation anesthesia in the horse. Often, the tensions of $C0_2$ in the blood are abnormally high, whereas oxygen tensions are abnormally low, despite the horse breathing 100% oxygen.
- Halothane but not isoflurane sensitizes the myocardium to catecholamines, increasing the probability of cardiac arrhythmias.
- Inhalation anesthetics have the advantage of being eliminated largely unchanged by the lungs; therefore, recovery from anesthesia does not rely on other organ function such as the liver. Only a minor portion (20% for halothane, 0.25% for isoflurane) of the inspired inhalant is eliminated by biotransformation in the liver.

Halothane is more potent (minimum alveolar concentration of anesthetic [MAC] for the horse: 0.88%) than isoflurane (MAC for the horse: 1.31%) and hence vaporizer settings for isoflurane generally need to be higher compared with halothane. Isoflurane is less soluble than halothane; and therefore, induction, recovery, and response to change in vaporizer setting are somewhat faster with isoflurane than halothane.

The administration of inhalation anesthetics requires special equipment, such as vaporizers, oxygen source, and flow meter, and an anesthetic breathing circuit suitable for the large size of equine patients. One cheap alternative is the use of a to-and-fro anesthetic machine, which can be constructed from a plastic bucket (Fig. 19-1). The necessity for endotracheal intubation and the method of administration of the inhalant in 100% oxygen has the great advantage of providing a protected and patent airway and of allowing the patient to breathe an oxygen-enriched gas mixture.

Some form of waste gas scavenging system should be installed to protect personnel from the adverse effects caused by exposure to low concentrations of anesthetic agents.

Knowledge of the effects of the inhalant agents and familiarity with and proper functioning of the anesthetic equipment are important to ensure safety of inhalation anesthesia.

KEY POINT

Before induction of general anesthesia

- *Make sure the vaporizer is full.*
- *Check the breathing circuit for leaks by occluding the y-piece (which connects to the endotracheal tube), closing the relief (pop off) valve, and filling the circuit with oxygen until the rebreathing bag is well expanded.*

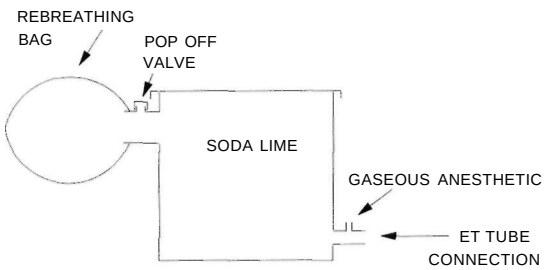


Figure 19-1. Cross-sectional drawing showing a "to-and-fro" anesthetic machine constructed using a plastic bucket with a top that is capable of sealing.

Discontinue oxygen flow. If the size of the bag and hence pressure in the circuit remains unchanged, the system is not leaking.
- *Check gas pressure in the oxygen tank to ensure enough oxygen is available for the planned duration of anesthesia.*
- *Make sure that the carbon dioxide-absorbing capacity of the absorber (soda lime) is not exhausted. Fresh soda lime granules can be easily crumbled between fingers and feel moist, whereas exhausted absorber is hard to crumble and feels dry. Color change of the absorber is reversible, and therefore, absence of color change does not mean good absorbing capacity.*
- *Leave the rebreathing bag filled with oxygen.*

Endotracheal Intubation

Endotracheal intubation in the horse is relatively easily achieved, provided the patient is adequately anesthetized. Horses, compared with other species, appear to have a less-sensitive larynx and hence tolerate an endotracheal tube even at light planes of anesthesia. Intubation is performed blindly. Endotracheal tubes should have an inflatable cuff, which should be checked for leaks before intubation.

KEY POINT

For endotracheal intubation
- *Place mouthgag (a piece of large-bore pipe may suffice)*
- *Extend the head well*
- *Advance the endotracheal tube (for the average adult horse, the tube size varies from 26-30 mm internal diameter) along the hard palate into the pharyngeal area*
- *Rotate the tube while advancing into the trachea*
- *Confirm proper placement by checking for air movement*
- *Once the breathing circuit is connected, inflate the endotracheal tube cuff while compressing the rebreathing bag and only until no gas can be heard to leak around the tube*

VAPORIZER SETTINGS AND OXYGEN FLOWS

- At the beginning of inhalation anesthesia and for approximately 10 minutes, both oxygen flow and vaporizer settings have to be high to effectively prime the circuit with oxygen and inhalation agent. Both can be decreased subsequently. As a general rule, vaporizer settings should be titrated to the horse's requirement and the following settings are only approximate guidelines. They are based on the assumption that the induction was provided for an adequately anesthetized patient.

KEY POINT

The initial O_2 flow should be 10 L/min and the initial vaporizer setting should be as follows: halothane 3 to 4% and isoflurane 3 to 4%. These initial flow rates and vaporizer settings are maintained for 5 to 10 minutes, depending on the depth of anesthesia. The maintenance O_2 flow should be 4 to 5 L/min and the maintenance vaporizer settings are as follows: halothane 1.5 to 2.5% and isoflurane 2 to 3%.

SUPPORTIVE CARE DURING ANESTHESIA

- *Patient positioning and padding:* When in lateral recumbency, the upper legs should be supported in a horizontal position. If the duration of anesthesia is to be greater than 30 minutes, some padding should be provided for the shoulder. Waterbeds or airbeds placed under the patient are most effective in preventing pressure over the radial nerve in horses in lateral recumbency and when in dorsal recumbency they assist in positioning the horse and protect the gluteal area. Correct patient positioning and padding will greatly reduce the incidence of postoperative myopathy and neuropathy.
- *Fluid therapy:* For any elective surgical procedure, if the duration of anesthesia is more than 30 minutes, IV fluids should be given at a rate of 5 to 10 mL/kg/h (3-5 L/450 kg/h). A balanced electrolyte solution should be used.
- *Body temperature:* With the exception of ill patients, horses maintain normal body temperature well during anesthesia. Depending on the environmental temperature and the extent of patient draping, body temperature in horses may increase during anesthesia.
- *Eye lubricant* should be placed in both eyes to avoid damage to the cornea.

Monitoring of the Anesthetized Patient

- Monitoring serves to ensure both optimal depth of anesthesia and vital organ system homeostasis. Homeostasis of tissue and organ function depends on delivery of oxygen to tissue and therefore depends on normal cardiopulmonary

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TABLE 19-3. Monitoring the Depth of Anesthesia

	Depth of Anesthesia		
Eye Sign	**Adequate**	**Too Deep**	**Too Light**
Eye position	Rotated anteromedially	Central	Central
Palpebral reflex	Present but slow	Absent	Present
Spontaneous blinking	Absent	Absent	Present
Tear production	Absent but cornea moist	Absent; cornea dry	Present

function. Monitoring of cardiovascular and pulmonary function is an integral part of patient monitoring during anesthesia.

- Anesthetic depth and cardiopulmonary function are to a large extent interrelated, and assessment of the one may reveal information about the other. For example, heart rate changes with changes in anesthetic depth.
- Often it is believed that the use of high-tech instrumentation is essential for successful patient monitoring. One has to realize, however, that even the most sophisticated equipment is useless if the values are not read and properly interpreted.

KEY POINT

The most important part of successful patient monitoring is having a knowledgeable person assessing the patient continually and entering the important objective variables on an anesthetic monitoring form, together with relevant events and observations.

- Much information can be collected simply by observation, palpation, and auscultation and will suffice for conclusive interpretation. Technical monitoring equipment aids in more objective monitoring. The most useful equipment is that to record the electrocardiogram (ECG), blood pressure, arterial blood gases, and oxygen saturation of hemoglobin.

Monitoring Depth of Anesthesia

- In the horse, position of the eye and degree of depression of protective eye reflexes are a useful indicator of anesthetic depth (Table 19-3).

KEY POINT

During ketamine anesthesia, even at adequate anesthetic depth, horses may still blink spontaneously and nystagmus may be present.

Monitoring of Cardiovascular Function

- The most important variables include heart rate, pulse strength, capillary refill time, and mucous membrane color (Table 19-4). Special equipment is required to assess arterial blood pressure, which is one of the most sensitive indicators of anesthetic depth. The indirect method (cuff and Doppler flow or oscillometric method) or direct method (arterial catheter connected to anaeroid manometer or pressure transducer and electronic measuring device), ECG, arterial blood gases, and oxygen saturation of hemoglobin in peripheral blood (pulse oximeter) are all valuable techniques to assess anesthetic depth.

Monitoring of Respiratory Function

- Assessment of respiratory function is undertaken by examination of respiratory rate, tidal

TABLE 19-4. Typical Values for Cardiovascular and Respiratory Variables for Anesthetized and Awake Horses

	Anesthetized	**Awake**
Heart rate (bpm)	30-35	30-45
Respiratory rate (bpm)	5-20	8-20
Tidal volume (L)	4-5	8-12
Capillary refill time (s)	<2.5	<2.0
Systolic blood pressure (mm Hg)	100-120	120-140
Diastolic blood pressure (mm Hg)	50-80	60-100
Mean blood pressure (mm Hg)	70-100	80-120

volume (by observing the frequency and extent of movements of the rebreathing bag), and mucous membrane color (see Table 19-4).
- Special equipment is required to assess arterial blood gases, tensions of CO_2 in exhaled air (capnograph), and oxygen saturation of hemoglobin in peripheral blood (pulse oximeter).

Troubleshooting

HYPOTENSION

- Most drugs used for premedication and anesthesia cause hypotension. Hypotension is particularly common during inhalation anesthesia.

KEY POINT

During anesthesia, mean arterial blood pressure should be at least 80 mm Hg.

- Adequate blood pressure is particularly important during longer anesthetics. If blood pressure can only be physically assessed, the pulse in a peripheral artery (facial, metatarsal) should feel strong and the artery should be palpable.

KEY POINT

For the treatment of hypotension

- *Make sure that the anesthetic depth is not too great. The vaporizer should be turned down and the patient kept anesthetized as lightly as possible.*
- *Increase rate of IV fluid.*
- *Catecholamines (inotropes), e.g., dobutamine 5µg/kg/min IV may be given.*

- Dobutamine is a commonly used catecholamine to improve blood pressure during anesthesia in horses. It has a rapid onset of action and in most horses increases blood pressure effectively. Dobutamine can be titrated to effect and has few side effects. It is administered by adding one vial of dobutamine (250 mg) to 1 L of 5% glucose or 0.9% NaCl (or 1/2 vial of dobutamine [125 mg] to 500 mL of 5% glucose or 0.9% NaCl) and piggy backing the solution into the IV fluid line. The dobutamine infusion should be commenced at a slow drip rate and increased gradually until the hypotension is corrected. Heart rate may decrease as blood pressure increases. Occasionally, dobutamine will increase heart rate more than pressure. If this is the case, dobutamine infusion should be reduced until there is a pressure response without an increase in heart rate, and if this is not possible the dobutamine infusion should be discontinued.

HYPOVENTILATION, HYPOXEMIA

- Hypoventilation and/or hypoxemia are common problems in anesthetized horses and especially when they are in dorsal recumbency.
- Hypoventilation is a consequence of insufficient tidal volume, insufficient respiratory rate, or a combination of both. Hypoventilation leads to an accumulation of $C0_2$ and a decrease in O_2 tension (hypoxemia) in the arterial blood.
- Hypoxemia may occur in anesthetized recumbent and especially dorsally recumbent horses, even if ventilation is adequate. Oxygenation of arterial blood depends on proper matching of ventilation and perfusion. Commonly in recumbent horses, the small airways in the dependent parts of the lung close but remain perfused. Blood circulating through these parts of the lungs will not be oxygenated and when admixed to the arterial circulation will decrease the oxygen tension in the arterial blood. The ventilation/perfusion mismatch affects CO_2 elimination much less than oxygenation and thus normal $C0_2$ tension does not automatically mean normal arterial oxygenation.
- Even when connected to an anesthetic machine and breathing approximately 100% oxygen, horses can become hypoxemic.
- A blood gas machine is necessary to determine arterial partial pressure of CO_2 ($Paco_2$) and O_2 (Pao_2). The Pao_2 tensions in normal awake horses is 80 to 100 mm Hg. The Pao_2 in anesthetized horses breathing room air should be at least 60 mm Hg. The Pao_2 in anesthetized horses breathing an oxygen-enriched gas mixture (approximately 100%) should be at least 200 mm Hg.
- For adequate pulmonary ventilation, a respiratory rate of 6 to 10 breaths per minute and a tidal volume of 4 to 5 L are necessary and can be assessed by the number and extent of rebreathing bag movements.

KEY POINT

Anesthetized horses especially when in dorsal recumbency are prone to hypoxemia. Hypoventilation is one of the factors contributing to hypoxemia. Early support of inadequate ventilation will help to prevent or minimize the degree of hypoxemia.

- To treat hypoventilation:
  - o Intubate trachea if not on inhalation anesthesia
  - o Connect to anesthetic machine or to a demand valve if total IV anesthesia is used and no machine available

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- o Close "pop off" (relief) valve on anesthetic machine
- o Make sure bag is full enough to deliver a breath
- o Squeeze rebreathing bag for about 1 to 2 seconds, using both arms, to inflate the lungs
- o Repeat 6 to 10 times/min
- o Ensure that the patient is not getting too deep (anesthetic uptake will be increased with the increased ventilation) by turning the vaporizer down
- o When using demand valve, activate the valve for 1 to 2 seconds and try to mimic a spontaneous breath

KEY POINT

Treating hypoxemia

- *Increase oxygen concentration in inspired air. For nonintubated horses on total IV anesthesia, place small-bore plastic tubing into nasal passage and deliver 100% oxygen at a flow of 10 to 15 L/min. For horses on inhalation anesthesia, oxygen concentration in inspired air is already maximal.*
- *Make sure ventilation is adequate and, if not, assist ventilation (see treatment of hypoventilation).*
- *Improve perfusion (see treatment of hypotension).*
- *Make sure that the horse is not too deep.*
- *Change horse's position if possible (i.e., from dorsal to lateral recumbency).*

SEDATION AND ANESTHESIA IN FOALS

KEY POINT

Foals are not simply a miniature edition of an adult horse. Their responses to anesthesia and sedation may be different from adult horses because of physiologic differences, particularly in the first month of life.

Physiologic Aspects Relevant for Sedation and Anesthesia in Foals

- Foals have a high metabolic rate and have high heart and respiratory rates. Because they have an immature cardiovascular system, hypovolemia is poorly tolerated. At the same time, they are very susceptible to fluid overload.
- In foals less than 1 week old, characteristics of the fetal circulation such as patent ductus arteriosus or incomplete closure of the foramen ovale still may be present.
- Foals also have an immature respiratory system, which makes them more susceptible to hypoxemia. The chest wall is compliant, which predisposes to a flail chest.
- Hypoglycemia may be present in foals held off food or in sick foals.
- In 1- to 2-week-old foals, the development of hepatic and renal function is incomplete, and therefore drug metabolism and excretion may be prolonged. However, by 1 month of age, hepatic and renal function are developed, and metabolism and excretion of drugs should be similar to adult horses.
- Foals are susceptible to hypothermia. Hypothermia will adversely affect cardiovascular function (bradycardia, vasoconstriction) and result in slow drug elimination, causing prolonged recovery from anesthesia.

Anesthetic Equipment

- Foals up to approximately 150 kg can be anesthetized using a standard small animal (human) anesthetic machine fitted with a circle-type breathing system and an out-of-circuit precision vaporizer. The rebreathing bag should be 3 to 5 L and the soda lime container I to 2 L in size.
- Cuffed endotracheal tubes of 10 to 16 mm internal diameter are suitable. For bigger foals that need adult horse size endotracheal tubes, the same anesthetic machine can be used as for adult horses but with a smaller rebreathing bag.

Preanesthetic Preparation

- *Clinical examination,* including heart rate and rhythm, respiratory rate, rhythm and respiratory effort, and body temperature should be performed.
- In sick foals, *packed-cell volume* (PCV), *plasma total protein* (TP), and *blood glucose* should be measured. *Serum/plasma electrolytes* and *venous acid base* status also should be measured in cases where electrolyte abnormalities are suspected, such as with ruptured bladder and gastrointestinal disorders. *Arterial blood gases* should be measured in cases showing signs of respiratory dysfunction.
- *Body weight* should be measured.
- An *ECG* should be performed in cases with electrolyte abnormalities (especially hyperkalemia).
- In sick foals, an *intravenous catheter,* 16 gauge 30 mm (1.25 inch), should be placed into the jugular vein to facilitate correction of hydration status, serum electrolyte, and blood glucose abnormalities before induction of anesthesia.

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- In foals without systemic illness, to minimize preanesthetic stress, a venous catheter can be inserted into the jugular vein following (mask) induction of anesthesia.
- Feed should not be withheld in sucking foals, because of the risk of hypoglycemia. Solid feed intake is limited in this age group, and any adverse effects resulting from solids are less significant than in the adult.
- The mare should be sedated before handling the foal and/or before induction of anesthesia. Acepromazine IV 0.03 to 0.05 mg/kg (15-20 mg for the average size horse) or acepromazine 0.02 mg/kg (10 mg for the average size horse) in combination with xylazine 0.3 to 0.4 mg/kg (150-200 mg for an average size horse) given IV are suitable for this effect.

Sedation and Anesthesia

SEDATION AND ANESTHESIA OF FOALS LESS THAN 1 WEEK OLD

- Anesthesia in foals less than 1 week of age is mostly for emergency procedures for treatment of conditions such as patent urachus, ruptured bladder, gastrointestinal disorders (intussusception, impaction), musculoskeletal injuries, and sepsis of joints. Foals suffering from any of these conditions are usually ill and to safely anesthetize them, they require a detailed preanesthetic workup, preanesthetic stabilization, and close intraoperative monitoring.

Sedation

- In foals less than 1 week of age, sedation for the purpose of preanesthetic calming is usually not necessary. Sedation may be indicated to facilitate examination and noninvasive procedures such as cast or bandage application.
- Diazepam IV 0.1 to 0.5 mg/kg will provide sedation and muscle relaxation for 15 to 25 minutes. Cardiopulmonary function appears to remain within normal range.
- Xylazine IM 0.3 to 0.6 mg/kg will provide sedation and muscle relaxation for 20 to 40 minutes. As in the adult, xylazine depresses cardiopulmonary function.

KEY POINT

Sedatives should be administered as closely as possible to body weight to avoid overdosing. Heavy and prolonged sedation in very young foals should be avoided because it will lead to hypothermia and significant depression of cardiopulmonary function and may interfere with normal gastrointestinal function.

General Anesthesia

KEY POINT

Sick foals need to be stabilized as much as possible before commencing general anesthesia. This includes correction of dehydration, electrolyte, and blood glucose abnormalities. External heat sources (e.g., heat lamps) should be used to correct hypothermia and/or counteract loss of body temperature.

KEY POINT

In foals up to 2 weeks of age, injectable anesthetics should not be used. Because of the immaturity of the hepatic and renal system, elimination of injectable anesthetic is slow and results in prolonged recovery.

Induction and Maintenance of General Anesthesia. The mare, sedated if necessary, should be allowed to be close to the foal during induction to avoid unduly stressing the foal.

- Halothane or isoflurane can be used for induction and maintenance of general anesthesia. Isoflurane will allow a more rapid induction and recovery, and the response to changes in vaporizer settings may be faster. Isoflurane, in comparison with halothane, has less cardiovascular depressant effects. Therefore, isoflurane is a more suitable inhalant for foals, especially very young ones. Although isoflurane should be the inhalant of choice for sick foals, it is much more expensive than halothane.

KEY POINT

For induction, the inhalant can be administered via face mask or via nasotracheal tube. Unless already recumbent, the foals can be physically restrained in standing position.

- A dog-face mask or any homemade snug-fitting mask is suitable for mask induction. For the nasotracheal method, a lubricated 7 to 9 mm endotracheal tube is passed into the ventral nasal meatus, similarly to placing a stomach tube. The tube can be left in the nasal passage or further advanced into the larynx and trachea. Once placement in the trachea is confirmed, the cuff can be inflated. This allows inhalation induction with minimal gas wastage and hence minimal exposure of personnel to anesthetic gases. Induction via nasotracheal tube also is faster than mask induction. Once the foal is anesthetized,

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an orotracheal tube can be placed in similar fashion as for the adult.
- Inhalation induction should be started by first administering oxygen alone for a short period (2-3 min) and then adding the inhalant by increasing the vaporizer setting up to the maximum for the vaporizer.
- Although nitrous oxide can be used, it is best to limit its use to the period of induction, because young foals are prone to hypoxemia. Therefore, if nitrous oxide is used during maintenance of anesthesia, it should not exceed 50% of the total fresh gas flow. Nitrous oxide also may cause gaseous distension of the intestine, and especially following short anesthetics, foals may show signs of colic postoperatively.

SEDATION AND ANESTHESIA IN FOALS OLDER THAN 1 WEEK OLD

Sedation

- The need for preanesthetic sedation in foals of this age group depends on temperament and physical status. Foals that resent handling and restraint should be sedated to reduce stress. In sick foals, induction of general anesthesia can be achieved without previous sedation.
- If sedation is needed, acepromazine IV 0.02 to 0.03 mg/kg, xylazine IV 0.3 mg/kg or IM 0.6 mg/kg, or diazepam IV 0.1 to 0.5 mg/kg can be used. Both acepromazine and xylazine depress cardiovascular function, and overdosing has to be avoided.

General Anesthesia

Induction. Inhalation induction via face mask or nasotracheal tube is the most common induction method in this age group. If a foal is difficult to restrain, even after sedation, injectable anesthetic induction may be a better choice than inhalation.

The following IV induction regimens can be used:

- Guaifenesin 50 to 100 mg/kg to effect, followed by ketamine 1.0 to 2.2 mg/kg
- Guaifenesin 50 to 100 mg/kg to effect, followed by thiopentone 5 to 8 mg/kg
- Propofol 2.0 mg/kg
- After premedication with xylazine IV 0.3 mg/kg, ketamine 2.2 mg/kg and diazepam 0.1 to 0.2 mg/kg mixed in the same syringe, given slowly

KEY POINT

The dose of injectable anesthetics needs to be calculated accurately. Estimation of dose will more often than not lead to overdose. The calculated volume of guaifenesin can be drawn up into 50-mL syringes instead of trying to estimate the dose from a 500-mL bottle.

Maintenance

For very short procedures, an induction dose of an injectable anesthetic may provide sufficient time of anesthesia. Prolonging anesthesia using injectable anesthetics is not recommended in foals of this age group because of prolonged recoveries. Maintaining anesthesia with injectable anesthetics should be avoided in sick foals. Halothane and isoflurane in oxygen are best used for maintenance of general anesthesia in foals less than 1 month old. Isoflurane should be used preferentially to halothane in sick foals.

Supportive Care

IV Fluids. In foals with normal hydration status, normal blood glucose, and normal serum/plasma electrolytes, a balanced electrolyte solution should be given at a rate of 5 to 10 mL/kg/min for any anesthetic of more than 30 minutes duration. In hypoglycemic foals, 5% dextrose or 2.5% dextrose in 0.45% NaCl at 5 to 10 mL/kg/min should be used. This solution should also be used in young normoglycemic foals (less than 1 month old) during prolonged anesthesia to avoid accidental hypoglycemia.

Body Temperature. Foals, especially young ones, will become hypothermic even during short periods of anesthesia. Body temperature therefore should be continually measured, and a means of maintaining or at least reducing loss of body heat should be applied.

Monitoring

Principles of intraoperative monitoring as described for adult horses also apply to foals. The intensity of monitoring depends on age and condition of the foal.

Depth of Anesthesia. Assessment is similar to adult horses. It is noteworthy that foals when lightly anesthetized may respond to a surgical stimulus much more vigorously than adult horses. Continual assessment of the patient is necessary to prevent the foal from becoming too lightly or too deeply anesthetized.

Cardiorespiratory Monitoring. In healthy foals older than 2 weeks and anesthetized for short periods of time (up to 1 hour) and for simple

surgical procedures, monitoring heart rate, pulse rate, pulse quality, mucous membrane color, capillary refill time, respiratory rate, and tidal volume (extent of movement of rebreathing bag) may be sufficient. In foals less than 1 to 2 weeks of age and in sick foals, the ECG and pulse pressure, directly via arterial catheter or indirectly via a cuff (placed on tail), and Doppler or oscillometer should be monitored. Pulse oximetry is a valuable method for cardiopulmonary monitoring. The clamp-like probe can be placed on the tongue. Many pulse oximeters measure and display both the degree of saturation of hemoglobin with oxygen in the tissue enclosed by the probe and the pulse rate. Oxyhemoglobin saturation should not fall below 90%. Unfortunately, pulse oximeter readings are susceptible to artifacts, and readings may not always be reliable. Arterial blood gases, determined from samples collected from the facial or great metatarsal artery, provide accurate information (from $Paco_2$) on pulmonary ventilation and (from Pao_2) on oxygen delivery to the arterial blood. Values for measurements that can be made in awake and anesthetised foals is presented in Table 19-5.

KEY POINT

The goal of intraoperative monitoring of cardiorespiratory function is to maintain values as close to normal as possible. In the immature cardiovascular system of foals, heart rate plays a major role in the regulation of blood flow to tissues and therefore should not be allowed to deviate greatly from normal. A decrease in respiratory rate commonly is seen in anesthetized foals and may be left uncorrected as long as the tidal volume is good.

Blood Glucose. In foals less than 2 weeks of age and in sick foals blood glucose should be continually assessed. Blood glucose should be corrected if values are less than 60 mg/dL (3 mmol).

Trouble Shooting

Hypotension. If pulse pressure in a peripheral artery (e.g., facial or great metatarsal artery) feels weak and the artery is difficult to palpate, if mean arterial blood pressure falls below 60 mm Hg, or if systolic pressure falls below 80 mm Hg:

- Make sure that the heart rate is within the desirable range.
- Ensure that the foal is not too deeply anesthetized and decrease anesthetic depth if possible.
- Start IV fluids if the foal is not already on fluids. A bolus of 10 mL/kg can be given. It is important not to overload the circulation with fluids.
- **Dobutamine 3 to 5 (xg/kg/min** can be administered. The immature cardiovascular system of foals often will respond with an increase in heart rate rather than with an increase in blood pressure. Especially in young foals, response and/or correction of heart rate rather than of blood pressure should be used as the end point of dobutamine therapy. The dose rate of 5 |xg/kg/min should not be exceeded.

Hypoventilation. If respiratory rate is less than 8 breaths/min, if tidal volume (i.e., small excur-

TABLE 19-5. Values for Cardiopulmonary, Hematologic and Arterial Blood Gas Variables in Awake and Anesthetized Foals 1-8 Weeks of Age

Variable	Awake	Desirable Values During Anesthesia
Heart Rate (beats/minute)	60-80	60-100
RR (breaths/minute)	30-40	>15
Tidal Volume		10-15
MAP (mm Hg)		>60
SAP (mm Hg)		>80 (100)
PCV (L/L)	6.35-0.40	>0.25
TP (g/L)	50-70	>50
Glucose (mmol/L)	7-9	>5
$Paco_2$ (mm Hg)		<60 (50)
Pao_2 (mm Hg when receiving 100% O_2)		200-350

MAP, Mean Arterial Pressure; SAP, Systolic Arterial Pressure

sions of the rebreathing bag) is inadequate, or if arterial blood gas analysis shows $Paco_2$ is greater than 65 to 70 mm Hg:

- Ensure that the anesthetic depth is not too great and reduce the concentration of anesthetic delivered by the vaporizer.
- Assist ventilation: close relief (pop off) valve; compress rebreathing bag for 1 to 2 seconds while watching chest and try to mimic a normal breath; if pressure gauge is present in the breathing circuit, do not exceed an inflation pressure of 20 cm H_2O; maintain a respiratory rate of 10 to 12 breaths/min; make sure patient is not getting too deep; if possible, repeat arterial blood gases—$Paco_2$ should be around 50 mm Hg, Pao_2 around 200 to 300 mm Hg.

Recovery

Ideally horses should be recovered in a quiet, dark area lying on a surface with padding, such as a 5-10 cm firm rubber mat, or rubber flooring of similar thickness as the padding. This surface should be nonslippery and kept dry and when using a mat, nonmobile and flat to prevent horses from slipping and tripping when attempting to stand. Once placed in the recovery area, the front leg, which is in the down position in the laterally recumbent patient, should be pulled cranially to minimize postoperative lameness due to nerve damage. Halters should not be put on recumbent patients since they may contribute to facial nerve damage and other injuries, unless they are specifically designed for that purpose, i.e., they are padded and have no hardware in places where trauma can occur. A source of oxygen should be available to allow for insufflation as long as the horse is recumbent.

The time of recumbency in recovery varies with the duration of anesthesia. There are two desirable scenarios for duration of recumbency: (1) the patient should remain recumbent until such a point that he/she can actually stand when attempting to do so or (2) recumbency and especially lateral recumbency is as short as possible to minimize the the risk of myopathy/neuropathy. As a general guideline for this conflict, horses can be left undisturbed for the first 30 minutes of the recovery period, then they should be assessed. If they appear to have sufficiently recovered, they can be encouraged to rise or at least go into sternal recumbency. If this is not the case they can be left for another 30 minutes after which time they should definitely be encouraged to stand. If needed, horses can be assisted to stand. A halter is placed on the patient and then support is provided by lifting the head and the tail up while the patient is attempting to get up.

When recovering horses in the field, the same principles apply. The horses' eyes should be covered to decrease CNS stimulation and hence premature attempts to stand. Since a halter often needs to be left on the patient, halter-related problems can be minimized by applying padding, e.g., a folded or rolled-up towel, between the halter and patient face on the down side of the head. Since field anesthesia is most commonly produced with parenteral (IV) anesthetics and is of limited duration, horses usually recover to standing within about 30 minutes.

Following inhalation anesthesia or any anesthesia during which the trachea was intubated, the endotracheal tube should be left in place until a spontaneous swallowing reflex has returned. Oxygen at a flow of 15 L/min can be administered via small-bore tubing placed well into the endotracheal tube. Once the endotracheal tube has been removed, make sure that the patient's airway is not obstructed (e.g., entrapped soft palate) and that breathing is effortless. In the case of congestion of the nasal passages, a 14-16 mm endotracheal tube or a similar type of tubing should be placed into one nasal passage to at least the level of the larynx and taped firmly in place (to avoid aspiration of the tube). This tube can be removed once the horse has recovered and is standing. Oxygen insufflation should be continued even after the endotracheal tube has been removed.

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Anthelmintics

David R. Hodgson and
Reuben J. Rose

A wide range of anthelmintics are available for the treatment of internal parasites. Most are now available in paste or gel form, which can be administered by owners and trainers. Unfortunately, these are sometimes administered incorrectly, and because of this, some larger horse farms have returned to the administration of anthelmintics by the veterinarian using nasogastric intubation.

KEY POINT

Details of the various anthelmintics that are available for oral (PO) use or nasogastric administration are presented in Table 19-6.

Many of these drugs are effective against large and small strongyles (cyathastomes), but only a few are effective in treatment of *Gastrophilus* spp., *Parascaris equorum, Habronema muscae, Onchocerca* spp., and tapeworms. Many available anthelmintics for horses are part of the benzimidazole group (oxibendazole, oxfendazole, fenbendazole, mebendazole), for which there is variable resistance among the cyathostomes.

KEY POINT

The most important clinical problems arising from parasite infestation are cyathostomiasis in the large bowel and to a lesser extent verminous arteritis.

Cyathostomiasis results from the larvae of cyathostomes maturing in the mucosa of the large colon. These larvae may cause local inflammation, and if there are sufficient numbers, colitis may result. This may be manifest as weight loss and diarrhea. In addition, the larvae may encyst or become hypobiotic in the mucosa in the winter or in response to routine anthelmintic therapy only to unencyst en masse in the spring, thereby resulting in colitis. Encysted larvae also have increased resistance to anthelmintics, and effective therapy may require repeated dosing with either fenbendazole (10 mg/kg/day for 5 days PO or 60 mg/kg PO once) or ivermectin at twice the normal dose rate (800 μg/kg PO). Moxidectin is also reputed to have improved efficacy against encysted larvae. With any of these regimens, only 50 to 70% of encysted larvae are likely to be killed; therefore, repeat dosing in approximately 14 days is often necessary. Some practitioners also recommend dosing horses with prednisolone (1 mg/kg q12h PO) before and during the therapy to assist in reducing the colonic inflammation that may result from killing of large numbers of encysted larvae.

Verminous arteritis results from the migrating stages of larvae of *Strongylus vulgaris,* which cause variable damage to the arterial intestinal supply, particularly the cranial mesenteric artery (see Chapter 7). Clinical signs vary from mild to severe colic and, occasionally, diarrhea due to intestinal damage during larval migration. Some anthelmintics have some larvicidal effects, the most commonly used being ivermectin, moxidectin, and fenbendazole. Because of this and the wide range of parasites that are sensitive to ivermectin and moxidectin, these have become the most widely used of the anthelmintics. As for cyathostomes, some benzimidazole agents are also larvicidal at higher dose rates but usually require dosages over several days and may produce diarrhea. The same dosage schedules may be used as for cyathostomiasis.

Antibiotic Therapy

Reuben J. Rose and Daria N. Love

Antibiotics are probably the group of drugs most widely misused in equine practice.

KEY POINT

Inappropriate antibiotic selection, incorrect dose rates, prolonged intervals between doses, and incorrect duration of therapy are a few of the common misuses.

TABLE 19-6. Dose Rates, Formulations, and Indications for Various Anthelmintics Used in Horses

Drug	Trade Name	Formulation and Dose Rate	Range of Parasites Treated
Oxibendazole	Anthelcide (SmithKline Beecham), Equipar (Coopers)	Paste or suspension, 10-15 mg/kg	*Strongylus equinus, S. edentatus, S. vulgaris,* small strongyles, *Parascaris equorum, Oxyuris equi, Strongyloides westeri*
Oxfendazole	Benzelmin (Syntex)	Powder, paste, suspension, 10 mg/kg	*S. equinus, S. edentatus, S. vulgaris,* small strongyles, *P. equorum, O. equi*
Oxfendazole + trichlorfon	Benzelmin Plus (Syntex)	Paste, 2.5 mg/kg oxfendazole, 40 mg/kg trichlorfon	Same as oxfendazole plus bots {*Gastrophilus* spp.)
Febantel + trichlorfon	Combotel (Haver/ Diamond Scientific), Negabot-Plus (Cutter)	Paste, 6 mg/kg febantel, 40 mg/ kg trichlorfon	*S. equinus, S. edentatus, S. vulgaris,* small strongyles, *P. equorum, O. equi, Gastrophilus* spp.
Febantel	Cutter Paste Wormer (Cutter), Rintal (Haver/Diamond Scientific)	Paste, liquid, 6 mg/kg febantel	*S. equinus, S. edentatus, S. vulgaris,* small strongyles, *P. equorum, O. equi*
Ivermectin	Eqvalan (MSD-Agvet)	Paste, liquid, 0.2 mg/kg	*S. equinus, S. edentatus, S. vulgaris,* small strongyles, *P. equorum, O. equi, Habronema muscae, Onchocerca* spp., *Gastrophilus* spp., *Dictyocaulus arnfeldi, S. westeri*
Fenbendazole	Panacur (Hoechst-Roussel), Safe-Guard (Hoechst-Roussel)	Paste, granules, liquid, 5 mg/kg	*S. equinus, S. edentatus, S. vulgaris,* small strongyles, *P. equorum, O. equi*
Pyrantel	Strongid (Pfizer), Imathal Equine (SmithKline Beecham)	Paste, liquid, 6.6 mg/kg of base	*S. equinus, S. edentatus, S. vulgaris,* small strongyles, *P. equorum, O. equi,* Cestodes (tapeworms)
Mebendazole	Telmin (Pitman-Moore)	Paste, liquid, 9 mg/kg	*S. equinus, S. edentatus, S. vulgaris,* small strongyles, *O. equi*
Moxidectin*	Quest gel (Fort Dodge)	Paste/gel, 0.4 mg/kg	*S. equinus, S. edentatus, S. vulgaris,* small strongyles, *P. equorum, O. equi, H. muscae, Onchocerca* spp., *Gastrophilus* spp., *S. westeri, Trichostrongylus axei*
Trichlorfon	Combot (Haver/ Diamond Scientific)	Liquid, 40 mg/kg	*Gastrophilus* spp., *O. equi, P. equorum*
Dichlorvos	Cutter Dichlorvos Horse Wormer (Cutter)	Powder, 170 mg/kg	*S. equinus, S. edentatus, S. vulgaris,* small strongyles, *P. equorum, O. equi, Gastrophilus* spp.
Piperazine		Powder, 67 mg/kg	Efficacy 100% against *P. equorum*

*Label claim of efficacy against larval/immature forms of large *(S. equinus, S. edentatus, S. vulgaris)* and small strongyles. Small strongyles = *Cyathostomum* sp., *Cylicocyclus* sp., *Cylicodontophorus* sp., *Cylicostephanus* sp., *Gyalocephalus* sp.

Antimicrobial therapy can kill off the normal bacterial flora and may allow some organisms to multiply unchecked by either the antibiotic or by competition from the normal bacteria. Resistance of microorganisms to antibiotics is also becoming an increasing problem. Whatever the ultimate mechanism of such acquired resistance, the prevalence of antibiotic-resistant strains is generally proportional to the extent of use of any particular antibiotic. A particular antibiotic acts as a powerful selection factor in the spread of resistant bacteria, and restriction of its use should reduce the proportion of resistant strains.

A more careful approach to antibiotic use is required by equine practitioners. Therapy should be based on bacteriologic information, which, if not known in the individual case, is based on likely causes and published information. Too often, broad-spectrum agents are used without thought for their potential adverse effects. If bacteriologic samples are to be taken, they should not be taken after days or weeks of antibiotics that have been unsuccessful but rather before therapy is commenced. Some of these issues are discussed in Chapter 17.

STRATEGY OF ANTIBIOTIC THERAPY

The ideal management of patients with infections in which it is intended to use antibiotics is

- Establish a clinical diagnosis.
- Confirm the nature of this infection by isolation of the causative organism.
- Prescribe the antibiotic as directed by sensitivity testing.
- Carry out follow-up examinations so that clinical and biologic cures can be checked.

It is usually only possible and practical to follow such a strategic plan in a minority of cases in hospital and even fewer in a practice setting.

Most horses diagnosed as suffering from bacterial infections are treated with antibiotics on an empirical basis. Generally, the results of such treatment are good, in large part because the natural defenses of the body are adequate to overcome the infection. Antibiotics merely assist these mechanisms rather than being the principal reason for recovery. Many infectious diseases do not require antibiotics for a successful outcome, although it is a difficult clinical decision to withhold such therapy when it is anticipated by clients.

In serious infections, horses will require chemotherapy urgently as a life-saving measure. Although every effort should be made to isolate the causal organism and examine its specific antibacterial sensitivities, therapy must be started at the earliest possible moment on the basis of experience in treatment of such infections.

KEY POINT

Wherever possible, a specimen for laboratory examination should be collected from the patient before the start of therapy.

If material cannot be obtained from the animal before therapy is begun, the isolation of microorganisms from tissues and circulating body fluids after antimicrobial therapy has begun is much more difficult. Even when only one dose of antibiotic has been given, it may make it difficult to see and/or culture the causal organism.

KEY POINT

There are many situations in equine practice where examination of simple smear preparations (e.g., from transtracheal aspirate, pleural fluid, abscess aspiration) will allow a presumptive diagnosis and rational antibiotic therapy to be instituted (e.g., streptococcal infections, actinomyces, and corynebacteria are uniformly sensitive to benzylpenicillin).

In other circumstances (unless imminently life threatening), therapy should be withheld until a correctly performed antibiotic sensitivity assay is available. The result of this will determine the therapy to be given, which should be given for sufficient time to enable resolution of the infection. Changing antibiotic medication after a few days "because it does not appear to be working" is not recommended.

Treatment of Clostridial Infection in Muscle

A common sequela to intramuscular injections administered by clients with unsterile syringes or needles is infection with *Clostridium* spp. This infection is extremely serious, and steps should be taken to begin appropriate therapy immediately. Any horse that shows local swelling, fever, depression, and inappetence 12 to 24 hours after an intramuscular injection should be regarded as being a likely candidate for a clostridial infection. With some clostridial species there will be local gas production, but others may show only swelling and pain. Where possible, the area should be scanned using ultrasound, and samples should be collected from the center of the swelling by aspirating with a needle and syringe. Fluid and/or

purulent material in a capped syringe should be submitted for Gram stain and appropriate culture (see Chapter 17), after which antibiotic therapy is begun. Either sodium or potassium benzyl penicillin (Treatment Nos. 85 and 86) should be given intravenously at a dose rate of 40,000 IU/kg q6h until there is clinical improvement. Surgical drainage of the area should be carried out, allowing the area exposure to the air, which will inhibit growth of the Clostridia and allow drainage of purulent material.

APPROACH TO ANTIBIOTIC THERAPY

The most important consideration in initiating antibiotic therapy is to decide if the horse has a definite bacterial infection. Antibiotics are often given on the basis of elevated body temperature, where there may be no bacterial infection. In our clinic, we have found that many horses will have a transient increase in temperature that resolves spontaneously in 12 to 24 hours.

KEY POINT

Many clinicians use nonsteroidal anti-inflammatory drugs to decrease the temperature, but as a routine approach, this may not be ideal.

Monitoring a temperature rise is useful in evaluating progress of an infection and response to therapy, and in most cases, antipyretic drugs should not be given. The following steps should be considered before starting antibiotic therapy.

1. Make a Diagnosis

Various diagnostic techniques should be considered to establish a bacteriologic cause of the disease. Techniques such as blood culture, transtracheal aspiration, thoracocentesis, abscess aspiration, and joint fluid collection may be useful, depending on the particular problem. Worse than no bacteriology is inappropriate bacteriology.

KEY POINT

For example, collecting bacteriologic samples from discharging wounds is never indicated.

Such samples will invariably result in growth of a mixed bacterial flora and give no indication of the causative organism. It is important that contamination of the sample for bacteriology does not occur during the collection procedure. The most common sources of contamination are from the hair or skin of the horse and occasionally from the person taking the sample. Contamination can be minimized by attention to skin disinfection and protection of the operator using sterile gloves and, in certain situations, a cap and mask. If possible, it is best to submit an aspirate in a sterile syringe rather than a bacteriologic swab. Unfortunately, a number of laboratories resist receiving samples in sterile syringes, and therefore arrangements should be made in advance and an explanation given.

Many sites, such as the respiratory and reproductive tracts, have a normal bacterial flora and thus the mere finding of bacteria on culture does not necessarily indicate an infection. Note should be made of the cytologic findings and the degree of bacterial growth (for details, see Chapter 17).

2. Use a Bactericidal Antibiotic if Possible

Bactericidal antibiotics are preferable to bacteriostatic antibiotics because in many situations (e.g., septic arthritis, pleuritis) there is compromised host phagocytic activity, and therefore bacteriostatic antibiotics may not eliminate the infection.

KEY POINT

Bactericidal antibiotics include benzylpenicillin, semisynthetic penicillins (amoxycillin, ampicillin), isoxazolyl penicillins (cloxacillin, flucloxacillin), cephalosporins (ceftiofur), and the aminoglycosides (streptomycin, neomycin, amikacin, kanamycin, gentamicin).

Bacteriostatic antibiotics include chloramphenicol, erythromycin, tetracyclines, and sulphonamides.

3. Use a Narrow-Spectrum Antibiotic

Although broad-spectrum antibiotics are often favored by clinicians, it is better therapeutic practice to use a narrow-spectrum agent (e.g., penicillin) to avoid complications such as killing off the normal bacterial flora, superinfection, and bacterial resistance. A failure of therapy with a narrow-spectrum agent provides an indication of the likely range of bacteria causing the infection and allows more rational adjustment of the antibiotic regimen.

4. Maintain Effective Blood Levels of Antibiotics

It has been established that maintenance of therapeutic blood concentrations of antibiotics is not

necessary to successfully treat an infection. This is so because intermittent antibiotic "peaks" (particularly with bactericidal antibiotics) can successfully eliminate an infection. Nevertheless, most bacterial disease responds best to maintenance of effective antibiotic levels in the bloodstream. Because it is difficult to determine the concentrations of antibiotics at the site of infection, maintenance of effective blood concentrations for a period of treatment is usual. It is generally accepted that the concentration that should be maintained is two to four times the minimum inhibitory concentration (MIC) of antibiotic for the particular bacteria. With most antibiotics, this requires treatment two to four times daily. As a generalization, the so-called long-acting antibiotics only maintain adequate concentrations against the most sensitive bacteria for the 2 to 3 days claimed by the manufacturer. In most cases, these antibiotics do not maintain effective blood concentrations of antibiotics.

5. Choose an Antibiotic Likely to Be Effective Against Equine Bacterial Pathogens

Consider the following points when making a decision to use a certain antibiotic:

Amoxicillin and Ampicillin. Most gram-negative equine pathogens are resistant to the semisynthetic penicillins because of (3-lactamase production by bacteria. Thus, these antibiotics are not cost effective, given that they are less active but considerably more expensive than penicillin G against gram-positive species.

Chloramphenicol. Whether given intravenously, intramuscularly, or orally, chloramphenicol has such a short half-life that it is not a useful antibiotic for treating horses. Furthermore, with the horse being used as a food-producing animal, there is also a public health risk.

Streptomycin. Streptomycin is still used in some practices in combination with penicillin for the treatment of infections in horses. However, most gram-negative pathogens are resistant to streptomycin, and therefore, unless sensitivity tests indicate otherwise, it should not be used in horses.

Oral Antibiotics. Oral antibiotics may not provide reliable blood and tissue concentrations in adult horses. Problems include variable absorption and diarrhea, particularly with penicillins. However, trimethoprim-sulfadiazine combinations are often useful in either paste form or powder administered in the feed.

6. Decide on the Duration of Therapy

The length of treatment should be decided by the specific problem and response to therapy.

> . . .. KEY POINT.
> *As a general rule, for antibiotics that are bactericidal, the time required for treatment of acute infections should be equivalent to the time that the animal has been clinically ill plus 3 days.*

Acute infections usually require therapy for 5 to 7 days.

> KEY POINT
> *However, it may be necessary to treat some staphylococcal infections and others that are intracellular survivors for several weeks to ensure that the organisms that have been phagocytosed are killed as they emerge from phagocytes.*

It is imperative that organisms that are intracellular survivors and multipliers are treated with bactericidal antibiotics for a quick resolution to the infection. It may be necessary to treat and monitor these infections for many months if they require bacteriostatic antibiotics. Some guides to the duration of antibiotic therapy are as follows:

- Surgical prophylaxis—12 to 24 hours
- Acute infection—5 to 7 days
- Septic arthritis—10 to 21 days
- Urinary tract infection—14 to 28 days
- Chronic pneumonia (e.g., *Rhodococcus equi* in foals)—1 to 3 months

7. Decide if Antibiotics Should Be Used Prophylactically

It is important to be sure of a bacterial infection before chemoprophylaxis is used. Unless a problem has been identified (e.g., a particular neonatal foal infection on a stud farm), prophylactic antibiotic use is of no value. Routine antibiotic prophylaxis (e.g., before and after surgery) is often used whether or not it is necessary. Important considerations in chemoprophylaxis associated with surgery include the site (i.e., whether contaminated or not), operating time, degree of tissue trauma, and likelihood of contamination.

Antibiotics have been used as agents in chemoprophylaxis for four purposes:

- To protect healthy animals against invasions by

a microorganism to which they have been exposed.
- To prevent secondary bacterial infection in individual animals acutely ill with disease, especially viruses, for which antibiotics are of no direct help.
- To reduce the risk of infection in animals with various types of chronic illness.
- To inhibit the spread of disease from areas of localized infection or to prevent infection in patients subjected to accidental or surgical trauma.

The following generalizations have been found to apply when antibiotics are administered prophylactically:

KEY POINT

When a single effective drug is used when bacterial infection is present but not clinically evident, the results are highly successful.

- If the aim is to prevent colonization and/or infection by any and all microorganisms that may be present in the internal or external environment, results are often unsatisfactory.
- The incidence of superinfection is directly related to the time of exposure to broad-spectrum antibiotics. To reduce the likelihood of superinfection, chemoprophylaxis should be used for only a short period.
- One of the great controversies in chemoprophylaxis is antibiotic use in viral respiratory disease. There have been no clinical trials to support or oppose the use of antibiotics in this situation. However, in principle, it is difficult to advocate the prophylactic administration of antibiotics for treatment of viral respiratory disease.

8. Antibiotic Dose Rates

The dose rates for various commonly used antibiotics, together with some specific considerations in their use, are presented in Table 19-7. Antibiotics used in foals are discussed in Chapter 9.

REASONS FOR FAILURE OF ANTIBIOTIC THERAPY

Failure of antibiotic therapy often leads the clinician to blame the antibiotic rather than looking carefully at likely reasons why the therapy has been unsuccessful. Some of the common reasons for failure of antibiotic therapy are discussed below.

Treatment of Untreatable Infections

One of the most common reasons for antibiotic failure is their use to treat viral infections. Treatment either fails or is deemed to be successful because when given for a sufficient period of time, the horse eventually recovers by natural defense mechanisms.

Improper Dosage or Duration of Therapy

Too Much. May be harmful in instances, especially where impaired elimination is present. This is particularly the case with the aminoglycosides, which can cause renal toxicity, especially in cases of shock and dehydration.

Too Little or Too Short a Time. Is probably the most common area of antibiotic misuse. Underestimating the body weight and providing treatment only once daily can result in failure of therapy.

Too Long. It seems general practice to treat horses for too long, especially for acute infections. In most infections, if there has not been clinical improvement after 5 days, you should be looking for an underlying problem that is making the animal refractory to treatment. It may be that you did not have the correct diagnosis and treatment in the first place or that the organism has become resistant to the antibiotic during the course of the treatment. It also may be a problem that will not respond to antibiotic therapy alone (e.g., infected tooth root, discharging sinus, septic arthritis).

Reliance on Chemotherapy with Omission of Surgical Drainage

This often places a demand on antibiotics they cannot always satisfy (e.g., lesions where there are appreciable quantities of purulent exudate or necrotic or avascular infected tissues). Where substantial quantities of pus, necrotic tissue, or a foreign body is a problem, the most effective treatment is a combination of antibiotic given in adequate dose plus a properly performed surgical procedure to provide effective drainage.

Certain drugs may be inactivated by microbial or other enzymes. Some drugs will not be as effective at low Eh (oxidation-reduction potential) or at low pH (situations likely to be encountered in abscesses). In some situations, the organisms may not be multiplying fast enough so that antibiotics directed against bacterial cell-wall synthesis are not effective in inactivating the organism. Likewise, phagocytic cells will not be able to function effectively in such an environment. This will be of particular consequence for antibiotics that depend for ultimate elimination of the organism on phagocytosis.

TABLE 19-7. Dose Rates of Antibiotics for Use in Adult Horses

Antibiotic	Dose Rate (per kg)	Interval	Route	Aspects of Treatment
Na or K benzylpenicillin	20,000-40,000 IU	q6h	IM. IV	Blood levels maintained for longer after IM than IV injection. May use for routine IV therapy or to achieve high circulating and tissue levels at start of therapy with procaine penicillin G.
Procaine penicillin	15,000-20,000 IU or 15-20 mg	q12h	IM only	Most useful antibiotic in equine practice for gram-positive pathogens. Excitement reactions may be seen after injection and occasionally anaphylaxis.
Long-acting penicillin (e.g., benzathine)	15,000-25,000 IU	q24-48h	IM	*Do not use except where the organisms concerned have very low MICs as low blood levels of penicillin maintained.*
Na amoxicillin or ampicillin	15-40 mg	q8h	IM, IV	These drugs have a similar spectrum of activity but have less activity than penicillin against susceptible organisms. Little efficacy against gram-negative pathogens in horses.
Na cloxacillin or oxacillin	30 mg	q8h	IM, IV	Used exclusively to treat beta-lactamase-producing *Staphylococcus aureus* infections, particularly in septic arthritis.
Cephalothin or cephalopirin	10-20 mg	q8h	IM, IV	Should not be used unless there is microbiologic evidence for efficacy. Use may result in diarrhea.
Ceftiofur	2-4 mg	q12h	IM, IV	Useful for acute bacterial conditions, especially pneumonias. Reported to cause diarrhea, particularly at higher dose rates.
Kanamycin	5 mg	q8h	IV, IM	Useful to treat some gram-negative infections. However, because only a small range of bacteria are sensitive, there should be culture and sensitivity evidence of potential efficacy.
Gentamicin	6.6 mg	q24h (may consider 3.3 mg/kg q12h in foals)	IV, IM	The most common aminoglycoside used. Effective against a wide range of gram-negative pathogens. Best to use IV because IM administration causes localized myositis.
Amikacin	7 mg	q12h or q8h	IV, IM	Should not be used unless sensitivity tests show no other aminoglycoside that is effective. Gentamicin should be the primary aminoglycoside used.
Oxytetracycline hydrochloride	5 mg	q12h	IV	Because it is a bacteriostatic drug, should only be used if bacteriologic tests indicate sensitivity. May cause diarrhea.
Trimethoprim plus one of the sulfonamides	15-20 mg of combined drugs	q12h	IV, PO	Higher dose with oral use of drug. Useful to treat a wide range of equine bacterial pathogens. At high dose rates may cause diarrhea.
Metronidazole	15-mg loading dose, then 7-15 mg	q6h	PO	Useful to treat anaerobic infections, particularly some cases of pleuritis caused by *Bacteroides fragilis* infections.

Lack of Adequate Bacteriologic Information

In human medicine, it has been documented that for hospitalized patients, half the courses of antimicrobial therapy are administered in the absence of support from microbiology. The great bulk of drugs used is based on clinical judgment alone. A high proportion of the use is for chemoprophylaxis of questionable value.

Bacterial samples are often inappropriately taken, and culture reports are therefore inaccurate and/or inappropriate. Bacterial cultures are obtained too infrequently, and results, where available, may be disregarded by the clinician when selecting the antibiotic.

KEY POINT

Routine use of drug combinations is a cover for diagnostic imprecision. The agents selected are more likely to be those of habit rather than for special indications, and the dosages used are routine.

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Anti-Inflammatory Therapy

Reuben J. Rose and
David R. Hodgson

The anti-inflammatory drugs used in equine practice can be classed in two major groups:

- Nonsteroidal anti-inflammatory drugs (NSAIDs)
- Corticosteroids

NONSTEROIDAL ANTI-INFLAMMATORY DRUGS

There are two major subdivisions of the NSAIDs that can be made:

- Enolic acids—the major drug in this group is phenylbutazone.
- Carboxylic acids—flunixin, meclofenamic acid, ketoprofen, and naproxen are in this group.

A third drug that is gaining more acceptance in equine practice is aspirin, which is also classified with the NSAIDs.

The NSAIDs block a part of the cyclo-oxygenase pathway and suppress synthesis of several chemical mediators of inflammation, such as thromboxane, prostacyclin, and prostaglandins. The NSAIDs reduce prostaglandin-induced heat, swelling, and pain in inflamed tissues. They are widely used for the treatment of skeletal and soft-tissue injuries and for reducing body temperature.

Although all NSAIDs have similar modes of action, clinical experience has dictated the use of different drugs for the range of clinical conditions encountered in practice. The two most widely used of the NSAIDs are phenylbutazone and flunixin meglumine. More recently, ketoprofen (Treatment No. 66) has become available and is efficacious in treating musculoskeletal and abdominal pain. Aspirin, despite its short half-life, is becoming more widely used in equine practice.

Phenylbutazone (Treatment No. 89)

- Phenylbutazone is by far the most commonly used NSAID and has been described as "the equine practitioner's best friend."
- Phenylbutazone is the least expensive of the NSAIDs and has been found to be effective in the treatment of a wide range of conditions.
- Most consistent responses have been found in the treatment of soft-tissue injuries and musculoskeletal problems, particularly low-grade lameness.
- Phenylbutazone also has an antipyretic effect and will reduce body temperature when there is a fever, which may be a problem if temperature is being monitored as a guide to possible infection. However, at low dose rates of phenylbutazone (2.2 mg/kg daily), there appears to be little effect on body temperature.

KEY POINT

We use phenylbutazone routinely after surgery for 3 to 5 days to reduce the extent of soft-tissue swelling and find that it is also useful

after suturing lacerations because the degree of swelling, and therefore tension on sutures, is minimized.

- Because the NSAIDs do not inhibit the cellular inflammatory response, they may be used without delaying wound healing.
- Phenylbutazone is available in oral formulations as tablets, granules, and paste, as well as a solution (200 mg/mL) for intravenous injection (see Treatment No. 89).
- The recommended dose rate for phenylbutazone is a loading dose of 4.4 mg/kg on the first day of administration, followed by 2.2 mg/kg twice daily for up to 4 days, and then 2.2 mg/kg for up to 7 days. Higher doses can be used if necessary to obtain the degree of pain relief required, but the horse should be monitored closely for any adverse effects.

Flunixin Meglumine (Treatment No. 52)

- Flunixin is considerably more expensive than phenylbutazone, but experience by most equine practitioners indicates that it is more effective than phenylbutazone in the treatment of visceral pain (colic) and in the symptomatic control of endotoxemia.
- Oral formulations include granules and paste, and flunixin is also available as a solution for intramuscular or intravenous (preferred route) injection.
- A dose rate of 1 mg/kg has been recommended for treatment of visceral pain, but the dose for control of endotoxic manifestations is as little as 0.2 mg/kg.

Ketoprofen (Treatment No. 66)

- Ketoprofen is an NSAID in the propionic acid group (similar to naproxen) that has been available for a few years and appears to be valuable in the treatment of musculoskeletal disorders. Some clinical experience has shown that it is also useful for the treatment of colic and uveitis. Ketoprofen has some effect on signs of endotoxemia but is not as effective as flunixin in relieving abdominal pain. One advantage of ketoprofen is that no adverse effects on gastrointestinal function have been reported even at five times the normal dose rate. It has a relatively long half-life, so treatment is only required every 24 hours.
- Ketoprofen is only available in a solution for intravenous injection. The concentration of the solution is 100 mg/mL.
- The recommended dose rate is 1 to 2 mg/kg given IV every 24 hours.

Aspirin (Treatment No. 9)

- Aspirin recently has become a popular drug with equine practitioners because it appears to have a more prolonged clinical effect, even though its half-life is short.
- An intravenous form of aspirin is available (Aspri-Ject Injection, Vedco, St. Joseph, MO), and the recommended dose rate is 35 mg/kg, given every 6 to 8 hours.
- The most common oral form is a large tablet or bolus containing 15.6 g aspirin (Aspirin Boluses, Butler, Dublin, OH; Farmtech, Kansas City, MO; Vedco, Agri Laboratories, Phoenix, St. Joseph, MO; rX Veterinary Products, Porterville, CA).
- Aspirin has a prolonged effect on clotting time, and this should be remembered if the drug is given before surgery.
- We have found aspirin useful for controlling minor abdominal pain, for low-grade lameness, and as an antipyretic agent. Aspirin is especially useful in the treatment of immune-mediated uveitis after the initial inflammatory response has been managed. It is relatively inexpensive and is available as a bolus that can be administered with a balling gun or ground up to permit a paste to be made using yogurt or corn syrup or the powder added to the feed.
- Because of its antithrombotic effects, aspirin also has been used for attempted prevention of laminitis and exercise-induced pulmonary hemorrhage.
- Other NSAIDs that have been used in horses include meclofenamic acid, indomethacin, and naproxen, but none is commonly used in equine practice. There is less clinical experience with these NSAIDs, which appear to produce a less reliable result than phenylbutazone, flunixin, or aspirin. Furthermore, most of the NSAIDs are considerably more expensive than phenylbutazone.

Nonsteroidal Anti-Inflammatory Drug Toxicity

KEY POINT

Phenylbutazone has been used widely in equine practice for the last 30 years, with few reports of ill effects.

However, during the last 10 years, reports of toxicity, particularly affecting the gastrointestinal tract and to a lesser extent the kidneys, have appeared

in the scientific literature. Ponies have been thought to be particularly susceptible to the toxic effects of phenylbutazone, but this is probably related to relative overdosage because of overestimation of body weight. Our experience indicates that because the granule or tablet formulations are in 1-g lots, many small ponies are given 2 g/day, which can translate into dose rates around 12 to 15 mg/kg, well above the upper limit of acceptable therapeutic range, which is less than 8 mg/kg.

The more difficult clinical situation is the horse with severe laminitis that may require large doses of phenylbutazone for prolonged periods to provide analgesia. Although the doses of phenylbutazone required (4-6 mg/kg daily) may produce signs of gastrointestinal dysfunction, lower doses will not result in sufficient analgesia, and the horse may suffer great discomfort.

Signs of toxicity relate to gastrointestinal dysfunction, with ulceration a common finding. Clinical signs include anorexia, colic, lethargy, weight loss, diarrhea, melena, and, terminally, death from shock. There is invariably a protein-losing enteropathy in cases of phenylbutazone toxicity because of the gastrointestinal effects of these agents. Toxicity is likely with the other NSAIDs if dosages above those recommended are given. Care must be taken with concurrent administration of NSAIDs and aminoglycoside antibiotics because they may increase the nephrotoxic effects of aminoglycosides.

KEY POINT

Because nearly all NSAIDs used in equine practice exert their effects by a common mechanism, the effects of these drugs are likely to be additive if used in combination.

Phenylbutazone is also very irritant if injected perivascularly.

KEY POINT

If large doses of phenylbutazone are required, the possibility of toxicity can be monitored by measurement of plasma or serum TP concentration. This will decrease in direct response to the toxicity.

Before any clinical abnormalities are found, blood biochemistry will show a decrease in TP. Therefore, intermittent blood sampling can be used to determine whether phenylbutazone is producing adverse effects on gastrointestinal function if prolonged or high dosage is required.

Toxicity appears to be less of a problem with the other anti-inflammatory drugs, although adverse signs have been reported with high doses of meclofenamic acid. Flunixin has been administered at dose rates of 3.3 mg/kg daily for 10 days without signs of toxicity or adverse effects on blood or urinary values. However, foals may be more susceptible, and therefore flunixin should be used with care.

Dose Rates

Recommended dose rates for NSAIDs are provided in Table 19-8. These dose rates are only a guide, but care should be taken if higher dose rates than those recommended are used.

DIMETHYLSULFOXIDE (Treatment No. 34)

- Although physiologic effects of dimethylsulfoxide (DMSO) are incompletely understood, the clinical usefulness of DMSO is well established in the treatment of acute musculoskeletal injuries, acute traumatic and inflammatory disorders of the CNS, and septic arthritis.
- DMSO appears to be of most use in acute inflammatory conditions and has little or no effect in chronic conditions.
- The anti-inflammatory effect of DMSO is associated with its role as a scavenger of free radicals, together with inhibition of the influx of inflammatory cells into the sites of inflammation.
- Although toxicity of DMSO is low, rapid IV administration can induce seizures, and hemolysis may be observed with high concentrations because of the high osmolality of DMSO. Topical DMSO may produce local skin irritation and "blistering" because of histamine release.
- DMSO is also used as a carrier in combination with other drugs (particularly corticosteroids) because it increases absorption through the skin. Because of this, care should be taken when using DMSO with any other drugs and also in avoiding contact of DMSO with the skin of the person applying the product. Preferably, local application should be performed using gloves.
- DMSO enhances the skin and blood-brain barrier penetration of nonionized molecules of low molecular weight, such as corticosteroids and some antibiotics.
- In horses, a 20% solution of DMSO in normal saline can be given safely as an intravenous infusion at a dose rate of 0.5 to 1.0 g/kg for the treatment of cranial or spinal cord trauma and some septic conditions.

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TABLE 19-8. Dose Rates and Routes of Administration of Nonsteroidal Anti-Inflammatory Drugs in the Horse

Drug	Form	Route	Recommended Dose and Use
Phenylbutazone	Tablets, paste, granules, powder	PO	Loading dose on first day of 4.4 mg/kg followed by 2.2 mg/kg q12h for 4 days and maintenance dose of 2.2 mg/kg daily thereafter. Used for musculoskeletal problems and perioperatively to minimize the extent of swelling.
Phenylbutazone	Solution	IV	Used to establish initial therapeutic blood levels. Dose rate varies from 2.2 to 4.4 mg/kg.
Flunixin meglumine	Solution, granules, paste	IV, IM, or PO	Highest recommended dose rate is 1.1 mg/kg. This is the dose used in colic cases. For endotoxemia, dose is 0.2-0.3 mg/kg given q6-8h.
Meclofenamic acid	Granules	PO	2.2 mg/kg daily for 5-7 days, and if further treatment needed, 2.2 mg/kg every second day. Most useful for horses with chronic laminitis. More expensive than phenylbutazone.
Naproxen	Powder	PO	10 mg/kg daily. Few clinical indications and seldom used.
Dipyrone monohydrate	Solution	IM or IV	10-20 mg/kg given once or twice only. Useful as a spasmolytic agent in some colic cases and as an antipyretic.
Phenylbutazone and isopyrin	Solution	IV only	4 mg/kg of phenylbutazone component of preparation, given once daily. Combination with isopyrin more than doubles the plasma half-life. Must be given very slowly (30-60 s) IV, because rapid injection causes excitement and ataxia. Useful to achieve prolonged response.
Aspirin	Solution and tablets	IV or PO	30-100 mg/kg q8h or q6h. Useful for some musculoskeletal problems, uveitis, laminitis, and mild abdominal pain.
Ketoprofen	Solution	IV	1-2 mg/kg q24h. Useful for musculoskeletal problems and endotoxemia.

KEY POINT

More recently, a 20% solution of DMSO also has been found useful in the local treatment of septic arthritis.

- After placement of indwelling drains under general anesthesia, 4 to 5 L of DMSO solution is infused into the joint, followed by 5 to 6 L of a povidone-iodine solution (see Disinfectants).

CORTICOSTEROIDS (Treatment Nos. 12, 13, 29, 30, 74, 93, 107)

Mode of Action

- Two classes of corticosteroid esters are available for parenteral administration. Prednisolone is the most widely used corticosteroid for oral administration.
- The short-acting succinate and phosphate preparations are used to attain high plasma and tissue concentrations, but the effect is short lasting. Conditions in which short-acting corticosteroids can be considered include shock, anaphylaxis, and allergic reactions.
- The insoluble esters such as acetate are absorbed and excreted more slowly, thus producing prolonged clinical effects. These "depot" type corticosteroids are used widely in equine practice, particularly for intra-articular medication.
- The corticosteroids act by stabilizing cellular, lysosomal, and mitochondrial membranes. They also inhibit phospholipase A_2, which is involved in the formation of arachidonic acid.

KEY POINT

The corticosteroids have a profound negative effect on wound healing, unlike the NSAIDs, and therefore should not be used perioperatively.

- Clinical experience with horses that required

arthrotomies within 1 to 2 months of administration of a long-acting corticosteroid intra-articularly is that wound breakdown was common. With the advent of arthroscopic surgery, this has become less of a problem.

Indications for Corticosteroid Use

- Corticosteroids act nonspecifically, and there are many undesirable side effects, particularly delayed healing, increased susceptibility to infection, and in joints some cartilage degradation. These possible adverse effects have to be weighed against the therapeutic benefits.

KEY POINT

Particular care must be taken in decisions about topical *corticosteroid use in ocular disease (possibility of corneal rupture if an ulcer is present), pulmonary disease (may worsen a low-grade infection), and joint disease (may aggravate preexisting cartilage damage).*

- Corticosteroids are useful for the treatment of various autoimmune disorders, the most common of which is probably pemphigus foliaceus.
- One of the contentious areas of corticosteroid use is in the treatment of shock. Most cases of shock do not require corticosteroids. However, in severe endotoxic shock, there may be a role for short-acting corticosteroid administration. High dose rates (0.5-1 mg/kg of dexamethasone) have been advocated.
- Corticosteroids are probably of most use in the treatment of some skin disorders (see Chapter 13). Oral prednisolone is inexpensive and effective. The usual dose rate is 1 mg/kg twice daily for the first 2 to 4 days, followed by 1 mg/kg once daily in the morning for 5 to 7 days, after which an every-other-day dosage (in the morning) is given to effect. This regimen has been shown to produce little adverse effect on adrenal function.
- Intra-articular use of corticosteroids is valuable where there is an acute synovitis (see Chapter 4). The main drugs used are methylprednisolone acetate (Treatment No. 74) and the combination of betamethasone acetate and betamethasone phosphate (Treatment Nos. 12 and 13). The possible negative effects of corticosteroids on joint function have probably been overemphasized, and clinical experience with the corticosteroids indicates that few adverse effects are encountered.

KEY POINT

In states with controlled medication in performance horses, it should be remembered that when used intra-articularly, the long-acting corticosteroids can remain detectable for up to 6 weeks.

- Other conditions where corticosteroids can be considered are rhabdomyolysis, some ocular conditions, some CNS diseases, and some chronic pulmonary diseases. Chapters on individual body systems should be consulted for further details.

Adverse Effects of Corticosteroids

- Inhibition of fibroblasts and increase in collagen breakdown, resulting in detrimental effects on wound healing.
- Inhibition of bone growth, matrix formation, and calcification can occur.
- Suppression of the hypothalamic-pituitary-adrenal system. Soluble forms of the corticosteroids result in adrenal suppression for 1 to 2 days. In contrast, the long-acting corticosteroids may produce adrenal suppression for up to 1 month.
- Great care ensuring aseptic technique when injecting corticosteroids intra-articularly is essential. Introduction of infective agents (usually bacteria) at the time of injection of corticosteroids may result in septic arthritis with severe long-term consequences for joint function, even if the infection is treated successfully.

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Figure 19-2. Foot bandage. Application of a poultice to encourage drainage of a subsolar abscess.

Bandaging and Casting

Reuben J. Rose and
David R. Hodgson

Bandaging of the distal limbs is necessary for restriction of movement, support, and to prevent swelling after surgery or after suturing wounds. If additional support or immobilization is required, the use of a large supporting bandage such as a Robert Jones bandage or a cast is required.

BANDAGING

A variety of bandages can be used but are usually based around three principles:

- Nonadherent dressings such as paraffin gauze to prevent the dressings adhering to the wound.
- Some form of padding such as sheet cotton to restrict movement, absorb any exudate, and allow pressure to be maintained evenly along the limb.
- A conforming bandage and/or elastic adhesive bandage to maintain the padding in position as a compressive layer.

Three main types of bandages are demonstrated in this section.

Foot Bandage

- Bandaging the foot is required after foot surgery to protect the foot and, if necessary, to maintain a poultice in position to aid in provision of drainage (e.g., subsolar abscess).
- After a poultice has been placed over the sole (Fig. 19-2), sheet cotton (cotton wool) is used to maintain the poultice in position (Fig. 19-3) and a cotton conforming bandage is applied as firmly as possible (Fig. 19-4). To prevent the bandage from becoming contaminated or wet, a plastic wrap can be positioned underneath the



Figure 19-3. Foot bandage. After application of a poultice, sheet cotton is applied over the foot.



Figure 19-4. Foot bandage. Cotton conforming bandage is applied firmly to hold the sheet cotton in position.

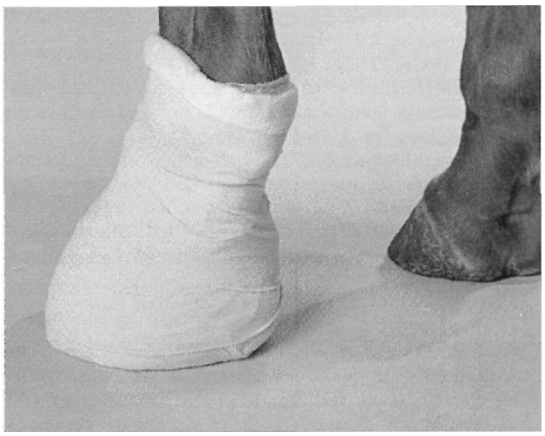


Figure 19-6. Foot bandage. Elastic adhesive bandage is used to complete the bandaging of the foot.

foot (Fig. 19-5) and fixed in position using elastic adhesive bandage (Fig. 19-6).

BANDAGING THE CARPUS OR FETLOCK

- For a light restrictive bandage that is useful to provide support to the carpus or fetlock, if there is a wound, a paraffin gauze dressing is applied (Fig. 19-7).
- This is followed by polypropylene light bandage (Fig. 19-8) and a conforming bandage such as Vetwrap (3M Company, St. Paul, MN). For bandaging the carpus, the bandage is best applied in a figure of 8 format (Figs. 19-9 and 19-10) so that pressure is avoided over the accessory carpal bone. When the fetlock is bandaged, a similar process is used, but a figure of 8 bandage is not necessary.
- An alternative is the use of an elastic adhesive bandage applied instead of the Vetwrap.

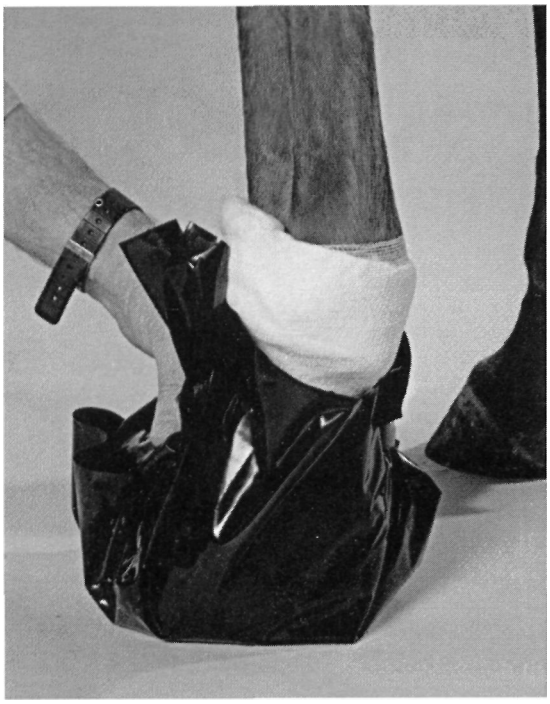


Figure 19-5. Foot bandage. Thick plastic is applied over the foot to prevent contamination of the bandage with urine and feces.

KEY POINT

With elastic adhesive bandages, pressure over the accessory carpal bone is released by cutting through the elastic adhesive bandage with a scalpel blade (Fig. 19-11).

ROBERT JONES BANDAGE

KEY POINT

For more extensive support and immobilization (e.g., in cases of severe lacerations or temporary fracture support), a Robert Jones bandage is extremely useful.

- This is a bandage that consists mainly of sheet cotton fixed in place with cotton conforming bandages.
- A full-limb Robert Jones bandage commences with rolls of 10-cm (4-inch) elastic adhesive bandage applied to the cranial and caudal surfaces of the limb (Fig. 19-12). These serve to prevent the bandage from slipping down the leg. Sheet cotton (four to five 500 g [1-lb] rolls) is

Text continued on page 745



Figure 19-7. Bandaging of the carpus. Paraffin-gauze dressing applied over a wound on the dorsum of the carpus.



Figure 19-8. Bandaging of the carpus. Application of a light polypropylene bandage to the carpus.

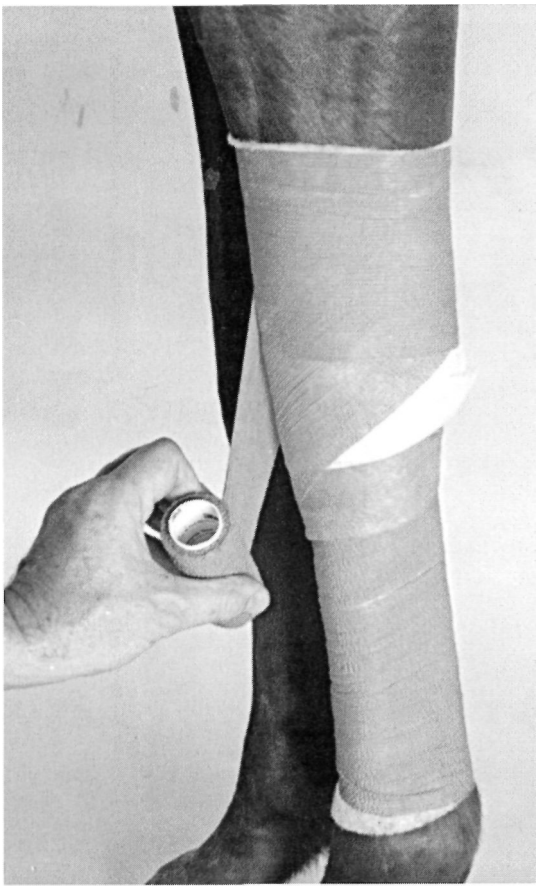


Figure 19-9. Bandaging of the carpus. A figure-of-8 bandage is used to avoid pressure over the accessory carpal bone.

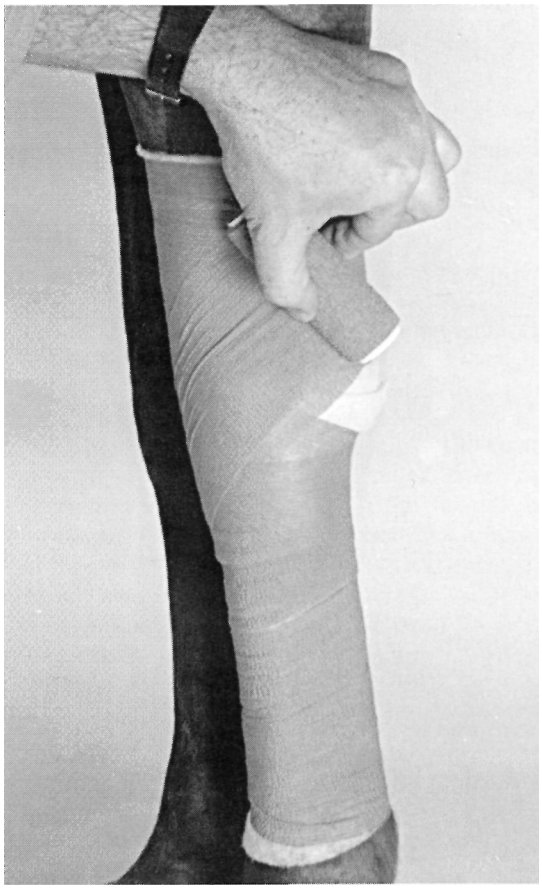


Figure 19-10. Bandaging of the carpus. The bandage used is Vetwrap (3M Company), which is a self-adhesive conforming bandage.

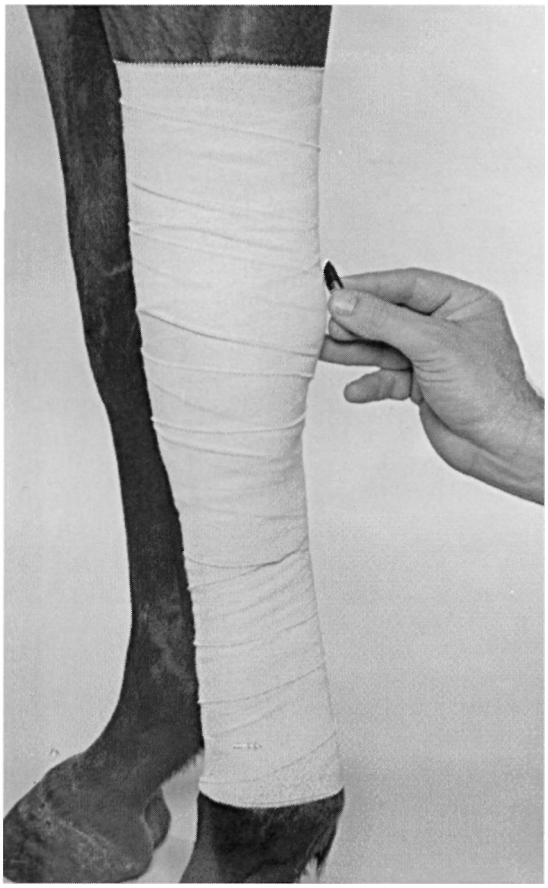


Figure 19-11. Bandaging of the carpus. An alternative to a figure-of-8 bandage is an elastic adhesive bandage that is applied normally over the carpus. After bandaging, pressure over the accessory carpal bone is released by cutting the bandage over the accessory carpal bone.



Figure 19-12. Robert Jones bandage. The bandage begins with application of elastic adhesive bandage to the front and back of the limb. Although this is not essential, it is useful to prevent the bandage from slipping down.



Figure 19-13. Robert Jones bandage. Sheet cotton rolls are applied to the limb from the foot up to the proximal forearm. For a full-limb Robert Jones bandage, four to five 500-g (1 -lb) rolls of sheet cotton are used.



Figure 19-14. Robert Jones bandage. After the sheet cotton is in place, 10-cm (4-inch) cotton conforming bandages are applied as tightly as possible.



Figure 19-15. Robert Jones bandage. The elastic adhesive bandages, applied initially to the skin, are taken back over the front and back of the cotton conforming bandage layer so that the adhesive side is to the outside.

then applied to the leg (Fig. 19-13), after which 10-cm (4-inch) cotton conforming bandages are applied as tightly as possible. For a full-limb bandage, about six to eight cotton conforming bandages are required (Fig. 19-14). After the cotton conforming bandages are in place, the original elastic adhesive bandages are taken up over the front and back of the bandage (Fig. 19-15), after which the bandage is completed using elastic adhesive bandages applied over the outside (Fig. 19-16). Flicking the bandage with the fingers should result in a sound similar to a tight drum.

TEMPORARY SPLINTING OF FRACTURES

R. Christopher Whitton

• The successful repair of a fractured limb depends on proper care of the limb after the fracture and during transportation to an adequately equipped equine hospital. Proper splinting of fractures prevents further damage to the soft tissues and bone ends, prevents closed fractures becoming open, prevents further contamination of open fractures, and allows the horse some ability to ambulate on the limb while reducing pain and anxiety. The type of splint bandage used depends on the site of the fracture. The most commonly used splint material is wood, but other solid materials have been used successfully.

Fractures distal to the distal third of the metacarpus are stabilized using a splint applied to the dorsal surface of the limb from the foot to immediately below the carpus. The limb is held just proximal to the carpus and the lower limb allowed to hang. One or two layers of cotton wool are applied from the foot to the carpus and the splint applied to the dorsal aspect of the limb and taped on. It is important not to use too



Figure 19-16. Robert Jones bandage. The bandage is completed by application of 10-cm (4-inch) elastic adhesive bandages.

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much padding because this will compress and result in instability. Fiberglass cast material is then placed over the splint to maintain alignment. Commercial splints that perform a similar function are also available.

For fractures of the midmetacarpus to the distal radius, a Robert Jones bandage using multiple layers of padding and conforming gauze is applied from the elbow to the foot. The bandage should be approximately three times the diameter of the limb. Two splints running the full length of the bandage are then applied with nonelastic adhesive tape laterally and caudally. For fractures of the middle and proximal radius, the lateral splint should be extended up the lateral aspect of the chest to prevent abduction of the limb. More proximal fractures cannot be splinted but are generally well supported by the surrounding musculature. Splinting the carpus in extension will help stabilize these during transport.

Fractures of the distal hindlimb are treated as for the forelimb except the splint is applied to the plantar aspect of the limb. For more proximal fractures of the metatarsus, a less bulky Robert Jones bandage is applied and the limb splinted laterally and plantarly from the calcaneal tuber to the foot. For fractures of the tarsus and tibia, a single lateral splint is applied over a Robert Jones bandage and extended to the ilium. A wide board is best for this purpose. It is not possible to splint fractures of the femur.

CASTING

R. Christopher Whitton,
Reuben J. Rose, and
David R. Hodgson

Materials

- A range of casting techniques has been used with a variety of materials, including plaster of Paris, thermoplastics, and fiberglass.

KEY POINT

Plaster of Paris is the easiest material to work with but has less strength than the other materials and is extremely heavy.

- The other problem with plaster of Paris is that it does not reach its full strength for approximately 24 hours after application, whereas the time that the major stress is on the cast is during recovery from anesthesia.

Fiberglass is now the most widely used casting material, and its advantages are that it is strong, light, and impervious to water. The major disadvantage of fiberglass is that it is extremely rigid and unforgiving and can result in pressure sores if applied with uneven pressure.

Casts are most commonly used for the treatment of severe wounds and for limb immobilization after fracture repair. Fiberglass casting tape is widely used due to its strength, light weight, and ability to set rapidly. Except tube casts for angular limb deformities in foals, the foot should always be included. Full-limb casts are applied to the level of the proximal radius or tibia, half-limb casts to the level of the proximal metacarpus/metatarsus, and short casts to the level of the proximal phalanx. Short casts are primarily used for severe wounds of the heels or the coronary band and help prevent the excessive granulation tissue that often complicates healing of these wounds. These casts are applied

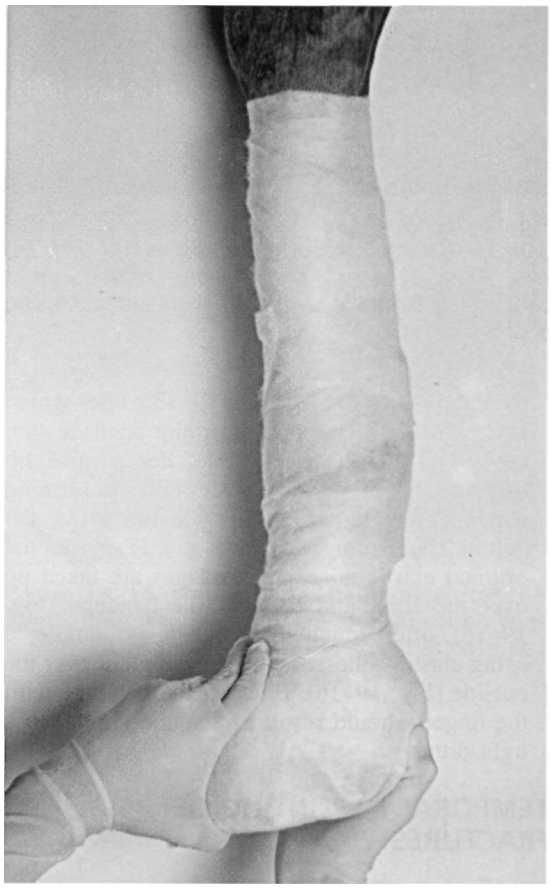


Figure 19-17. Application of a half-length cast. Casting begins with application of light padding using a polypropylene bandage.



Figure 19-18. Application of a half-length cast. Stockinette (10 cm diameter) is applied over the padding.

after adequate debridement and lavage of the wound, and this may be done with the horse standing and sedated with regional anesthesia or under general anesthesia. Half-limb casts are used for wounds of the pastern, fetlock, and distal metacarpal/metatarsal areas or for stabilization of fractures distal to the midmetacarpus/metatarsus generally in combination with some form of internal fixation. Full-limb casts are used for wounds of the proximal metacarpus/metatarsus, carpus or tarsus, and distal antebrachium or crus. Tube casts may be used in young foals with incomplete ossification of the cuboidal bones of the carpus or tarsus. These are applied from immediately proximal to the fetlock joint up to the proximal radius or tibia.

Cast Application

- With the exception of short casts, most casts are applied under anesthesia. The shoe on the limb to be cast should be removed and the foot trimmed. Casts should be applied with the leg in extension. This is usually achieved by an assistant holding the limb while the casting material is applied. Wire threaded through holes in the hoof wall can be used to apply traction to the limb.
- If there is a wound, a sterile dressing is applied over the affected area. This is usually a nonstick dressing of limited thickness. Padding should then be applied over the whole area to which the casting material is to be applied. It is always a compromise between good immobilization (the least amount of padding possible) and prevention of pressure sores (extra padding). We prefer the least amount of padding possible and use a single roll of polypropylene padding (Fig. 19-17) with a 10-cm-diameter stockinette (Figs. 19-18 and 19-19) applied over it. Foam rubber (Reston foam, 3M Company) can be used to prevent pressure sores at the top of the cast and over areas such as the accessory carpal bone, sesamoids, and calcaneus (Fig. 19-20).

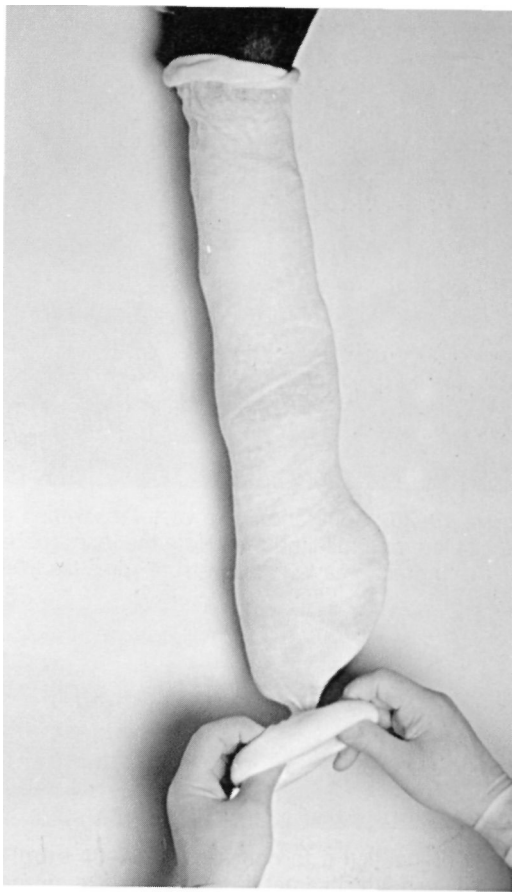


Figure 19-19. Application of a half-length cast. Stockinette in position.

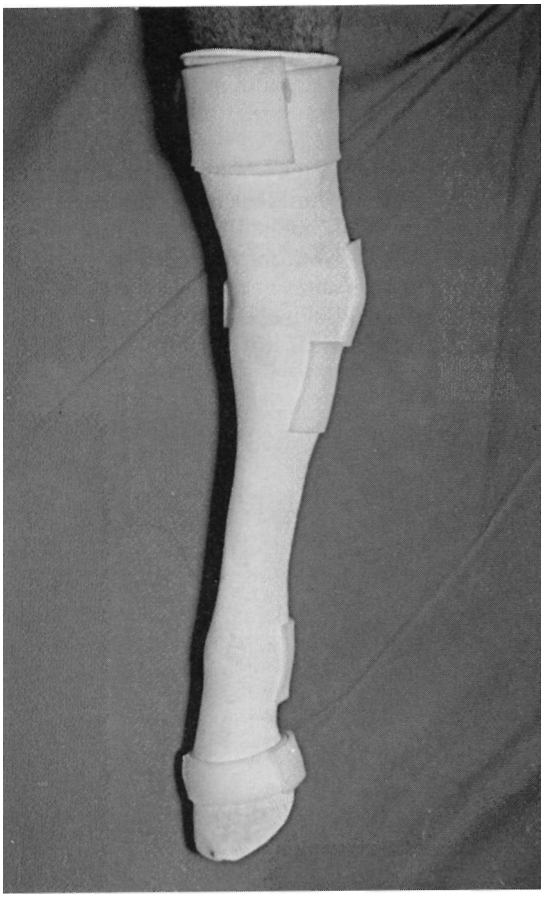


Figure 19-20. Application of a cast. Preparation of the hindleg for a full-limb cast. Note the foam rubber used to protect areas where pressure sores are likely to occur.

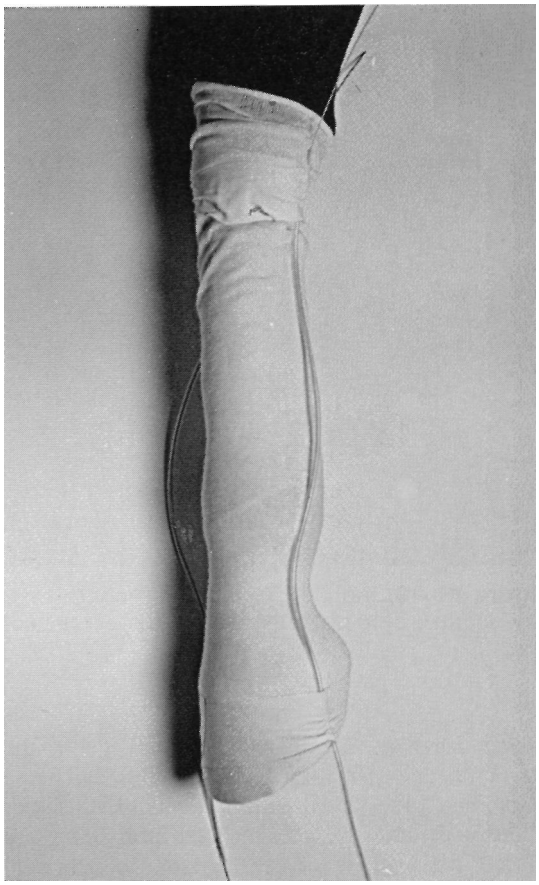


Figure 19-21. Application of a half-length cast. Before the casting material is applied, embryotomy wire may be applied inside drip-set tubing to provide a simple mechanism for later removal.

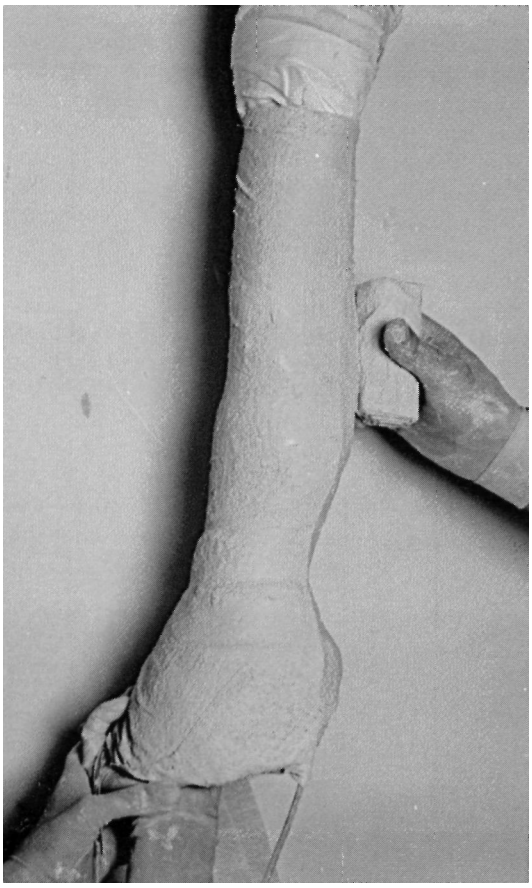


Figure 19-22. Application of a half-length cast. The cast is begun using 10-cm (4-inch) rolls of plaster of Paris bandage.

KEY POINT

To aid in easy cast removal, we include embryotomy wire underneath the cast.

To prevent rusting and wire breakage, the wire is placed inside suitable material such as drip-set tubing and fixed on the medial and lateral sides of the limb with elastic adhesive bandage (Fig. 19-21).

We begin the cast with one to two layers (two to three rolls) of 10-cm (4-inch) plaster of Paris bandage (Fig. 19-22), which can help to avoid pressure sores that may result when fiberglass is applied directly. It is possible to use fiberglass alone without the plaster of Paris. For application of plaster of Paris, we use hot water to hasten the setting time. After this, a roll of 10-cm plaster of Paris is used underneath the heel of the foot to ensure a level surface underneath the cast on which the horse can walk (Fig. 19-23). An alternative is the use of a metal walking bar. Fiberglass rolls (10-cm-diameter; Vetcast, 3M Company) are then applied after immersion in lukewarm water. These rolls should not have the excess water squeezed out, unlike plaster, but rather the water should be allowed to drip from the bandage before its application. The rolls are applied beginning at the foot, overlapping each layer by about two-thirds of the diameter of the bandage (Fig. 19-24). Extra layers should be applied over regions where the cast is more likely to break, such as the pastern, fetlock, carpus, and tarsus. For a half-length cast, about six rolls of 10-cm-diameter fiberglass are needed, and for a full-length cast, 10 to 12 rolls of fiberglass are needed. To prevent a sharp edge at the top of the cast, the stockinette can be pulled distally immediately after fiberglass application to ensure a smooth edge to the cast. The embryotomy wires within the drip-set tubing are taped to the cast to complete it and some tire innertube applied over the sole for waterproofing and protection (Fig. 19-25).

To remove the cast, it is simple to attach handles to the embryotomy wire and saw through the medial and lateral sides of the cast (Fig. 19-26).



Figure 19-23. Application of a half-length cast. To ensure a level surface underneath the cast, a 10-cm (4-inch) roll of plaster of Paris can be placed at the heel of the cast.

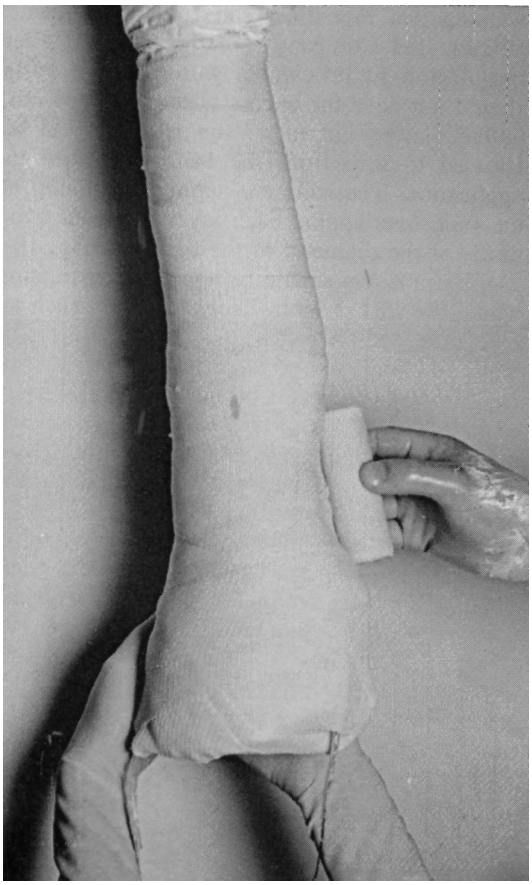


Figure 19-24. Application of a half-length cast. After the plaster of Paris bandages are applied, 10-cm (4-inch) fiberglass bandages are used to provide the reinforcement necessary for the cast.

KEY POINT

It is critical that a cast be changed at the first sign of any sudden lameness or obvious irritation, with the horse biting at the cast.

- Daily inspection should be made for signs of discharge and cracks, and the cast should be changed if a crack appears. The length of time a cast can stay on varies greatly but rarely exceeds 6 weeks. While the horse is comfortable and there are no complications with the cast, there is no need to change it. The exception is casts on the limbs of young growing foals that should be changed every 2 weeks.

Cast Removal

- If the cast is not to be replaced, it should be removed standing under sedation. If a new cast is to be applied, the cast is removed under general anesthesia. The most convenient method of cast removal is to use an oscillating saw to cut both sides and the base of the cast. Cast spreaders are extremely useful to separate the two halves. If an oscillating saw is not available, two pieces of embryotomy wire can be incorporated on either side of the cast when it is applied. The wire is positioned between the padding and the casting material within tubing such as that from a fluid administration set. The ends of the wire are folded against the cast and covered with adhesive tape. When the cast is removed, handles are attached to the wires and each side of the cast sawed through in turn.

Transfixation Casts

- In cases with fractures that have poor axial stability, a cast incorporating transfixation pins

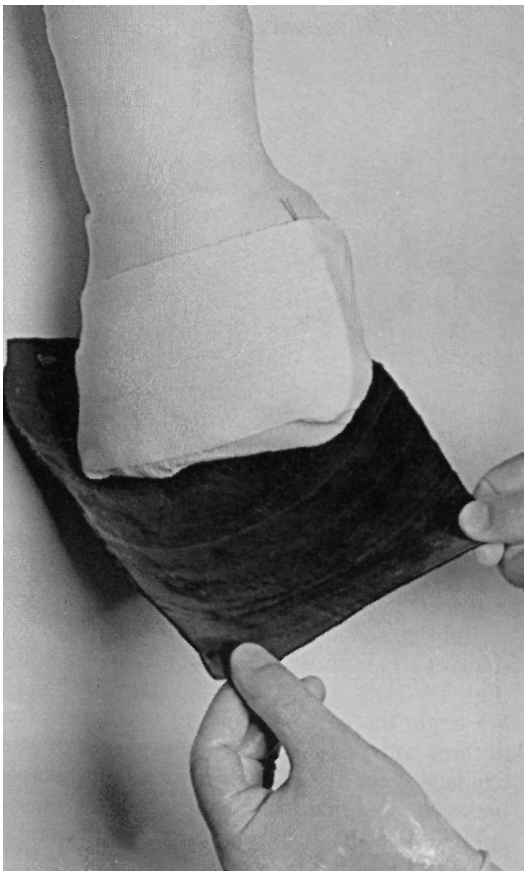


Figure 19-25. Application of a half-length cast. The cast is completed by taping the embryotomy wire to the cast and using a piece of rubber inner tube applied to the bottom of the cast. This is to prevent the cast from slipping when the horse places weight on it.

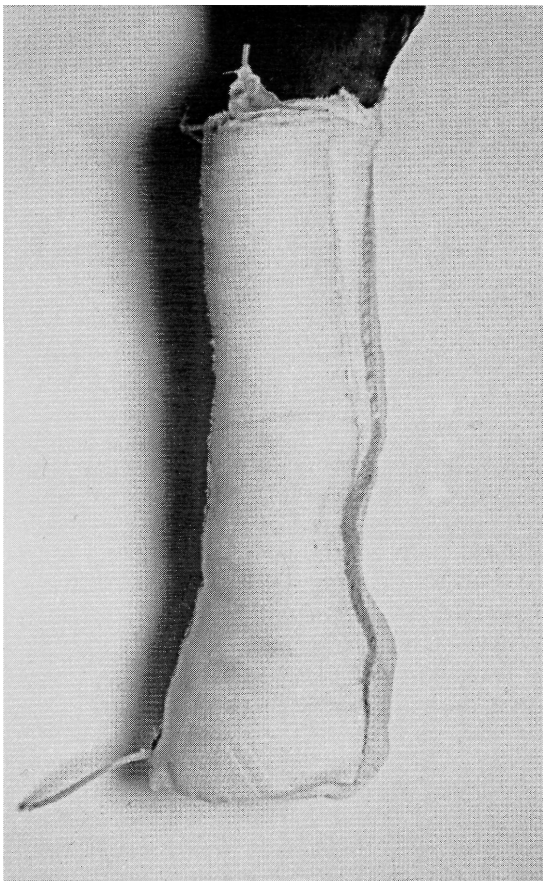


Figure 19-26. Cast removal. The cast is easily removed by attaching handles to the embryotomy wire and sawing through the medial and lateral sides of the cast.

through the bone proximal to the fracture site can be used to prevent collapse. Transfixation casts are most commonly used for highly comminuted fractures of the pastern, where the pins are placed in the distal third metacarpal or metatarsal bone. It is important that the pins are placed distally in the bone and the cast extended to the proximal end of the bone to reduce the risk of fracture at the pins.

- The cast is applied under general anesthetic. Generally, two quarter-inch or 6-mm-smooth Steinmann pins are placed aseptically through stab incisions. The holes should be predrilled with an appropriate-sized drill bit. The first pin is positioned from lateral to medial 4 cm proximal to the fetlock joint and the second pin 3 cm proximally. The pins are cut so that 3 to 4 cm is protruding on either side and the cast is applied as previously described. The fiberglass cast tape is cut to allow it to be placed over the pins. The pins are then cut flush with the cast and another layer of casting tape applied over the top.
- After about 6 weeks, transfixation casts are removed under general anesthesia and replaced with a normal cast for a further 6 weeks. An oscillating saw is necessary to free the pins from the cast. Smooth pins generally loosen by this time and are easy to remove. To prevent pin loosening, threaded pins have been recommended.

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Disinfectants

Reuben J. Rose and David R. Hodgson

Disinfection is the elimination of some or all pathogenic microorganisms from an object so that infection is prevented. Disinfectants are used widely in equine practice for some instruments and where sterilization is not possible or impractical. Disinfectants are also used in the treatment of some wounds and irrigation of abscesses.

KEY POINT

However, many disinfectants are inactivated by organic materials such as pus and blood.

More important, the disinfectants will cause considerable tissue irritation and should be used with great care around wounds or mucous membranes. Too often, disinfectants are splashed into a bucket until a color is achieved that the practitioner believes is appropriate. Accurate dilution is required to achieve the correct concentration for efficacy without tissue irritation. Some major disinfectants available are presented in Table 19-9. A wide variety of other disinfectants are available, and readers are referred to the *Compendium of Veterinary Products* (North American Compendiums, Port Huron, MI) for further details.

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TABLE 19-9. Disinfectants: Effective Concentrations and Use

Disinfectant	Group	Trade Name	Use	Concentration	Effective Concentration
Hexachlorophane	Phenols	pHisoHex (Winthrop)	Skin	3%	Concentrate (3%) is used on skin
Chlorhexidine	Phenols	Hibiclens (Stuart)	General	4%	0.5% solution for general use (10 mL in 100 mL water) and 0.05% (1 mL in 100 mL) solution for wound cleansing
Iodine	Halogens	Tincture of iodine (mitis)	Skin		2.5% iodine and 2.5% KI in 90% ethanol
Povidone-iodine	Halogens	Betadine (Purdue-Frederick)	Skin and mucous membranes	10% povidone-iodine	10% iodine needed (equivalent to 1% available iodine)
Ethyl alcohol	Volatile solvent		Skin		70% alcohol is the most active form. To make up appropriate concentration using surgical spirits, take 815 mL and make up to 1 L with water
Benzalkonium chloride	Quaternary ammonium	Zephiran (Winthrop)	General	17%	0.05%-0.2% solution (5-20 mL in 1 L water)
Dialdehyde	Aldehydes	Cidex (Surgikos)	Instruments	2%	Place instruments in activated solution for 10 minutes

Medication Administration

Reuben J. Rose and
David R. Hodgson

Medication is administered to horses via three main routes: PO or via a nasogastric tube, IM, and IV. A variety of preparations such as antibiotics, anti-inflammatory agents, vitamins, minerals, and anthelmintics are available in paste form, and these products are easily administered by depositing on the back of the tongue via a syringe placed in the interdental space. Nasogastric tubing is also used for fluid and electrolyte administration and for a range of other medications, such as mineral oil and anthelmintics. Details of the technique for nasogastric tubing are given in Chapter 7.

Intramuscular Injection

- Intramuscular (IM) injections can be given in four main sites: the neck, pectorals, rump, and hindleg.

KEY POINT

We *use 18- or 19-gauge, 3.75-cm (1.5-inch) needles for all IM injections, and the needle should always be inserted right up to the needle hub.*

- If the needle is inserted only part of the way, it is possible that as the injection is being given, the needle may be pushed into a vessel, and therefore some of the drug may be administered intravascularly.
- Skin cleansing and disinfection are done using a suitable solution such as 70% alcohol. For IM

injections in the neck, after a few taps with the back of the hand, the needle is inserted about 7.5 cm cranial to the line of the shoulder, in the middle of the neck (Fig. 19-27). Note that further ventral there is a risk of striking the transverse processes of the cervical vertebrae and dorsally the injection will be made into the ligamentum nuchae, where it will be poorly absorbed.

KEY POINT

The pectoral site is a good one because horses tolerate injections in this site (Fig. 19-28).

- The only disadvantage is that localized edema often is seen in the days after the injection, and this may be considered a problem by the client.
- The middle gluteal muscle in the rump is a useful injection site because it is one of the largest muscles in the body. It is located by placing the thumb along the tuber coxa and spreading the hand so that where the small finger lies, the site is close to the center of the middle gluteal muscle. The needle is inserted at this site (Fig. 19-29).
- Injections into the semitendinosis and/or semimembranosis muscles are useful sites in foals and also may be used in adults (Fig. 19-30).
- Before administration of the medication, aspiration should be performed with the syringe to ensure that the needle is not lying within a vessel. If IM injections are required several times a day for several days or weeks, it is wise to alternate the injection sites and to use the left and right sides of the horse so that the degree of inflammatory reaction is minimized at each of the locations.

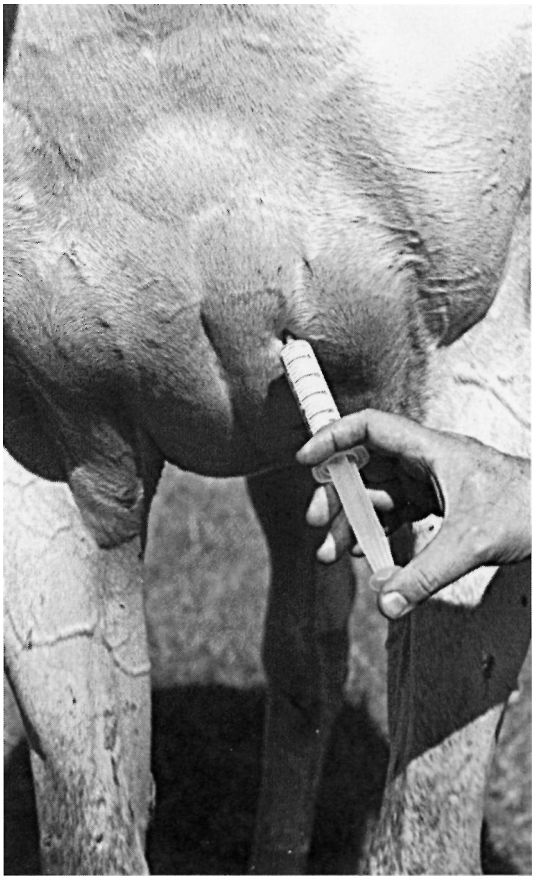


Figure 19-28. Intramuscular injection. Position of needle for injections in the pectoral muscles.

Intravenous Injection and Catheterization

- IV injections are necessary for administering a wide range of medications.

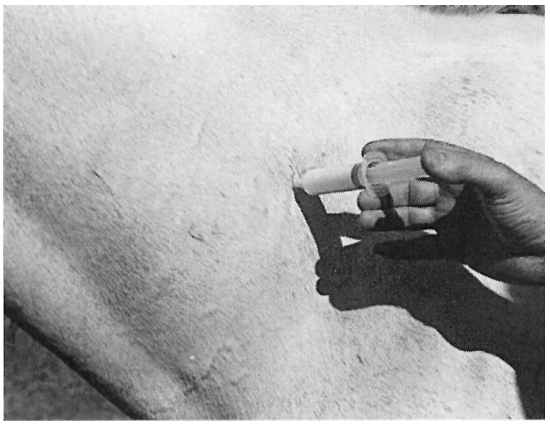


Figure 19-27. Intramuscular injection. Position of needle for injections in the neck.

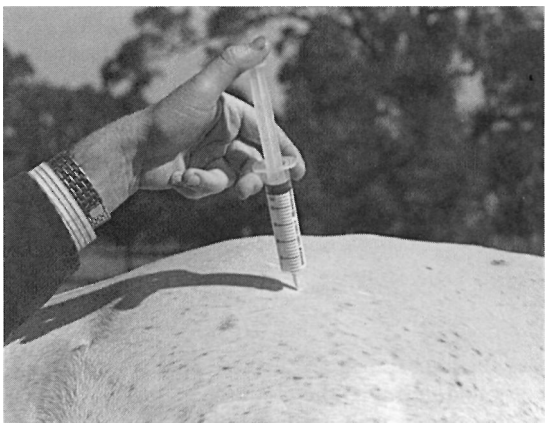


Figure 19-29. Intramuscular injection. Position of needle for injections in the middle gluteal muscle.

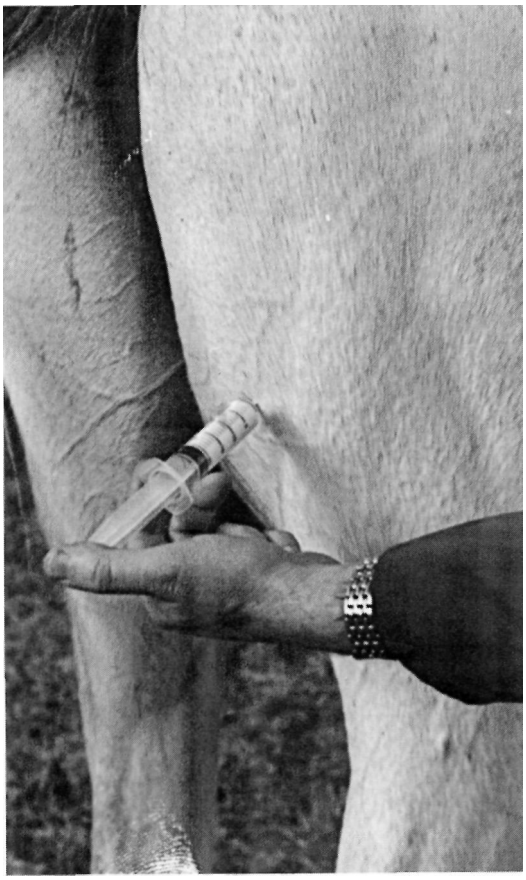


Figure 19-30. Intramuscular injection. Position of needle for injections in the semitendinosis muscle.

Many drugs given are irritating if injected perivascularly, and therefore care must be taken to ensure that the needle is wholly within the vein. Although a range of veins are used for IV catheterization, most injections are given via the jugular vein. After the skin over the vein is disinfected, the vein is distended by maintaining digital pressure on the vein in the lower third of the neck, and the needle is inserted (Fig. 19-31). We prefer 19-gauge, 3.75-mm (1.5-inch) needles for all IV injections.

KEY POINT

Catheterization must be done with care given to sterile technique.

Complications such as phlebitis and septicemia can arise from inadequate skin disinfection and trauma during catheterization.

The jugular vein is commonly used for IV catheterization, and the following technique is used. Hair over the vein should be clipped and shaved,



Figure 19-31. Intravenous injection. The needle is inserted at a slight angle to the jugular vein, and after blood flows from the needle, it is straightened and inserted up the lumen of the vein until the hub is reached.

after which the skin is given three 1-minute scrubs with povidone-iodine scrub (Treatment No. 91), interspersed with application of 70% alcohol. To complete disinfection, povidone-iodine solution (1% available iodine) is sprayed on the skin. Local anesthetic (0.5-1.0 mL of 2% lidocaine, mepivicaine, or prilocaine) is injected intradermally over the center of the jugular vein (Fig. 19-32), after which the skin is disinfected with povidone-iodine.

Sterile gloves are worn when inserting either a 12- or 14-gauge catheter, 8 to 30 cm (3-12 inch) long. The longer catheter is used if long-term fluid therapy is planned, and a 12-gauge catheter is selected if rapid fluid infusion is required. The catheter can be inserted either up or down

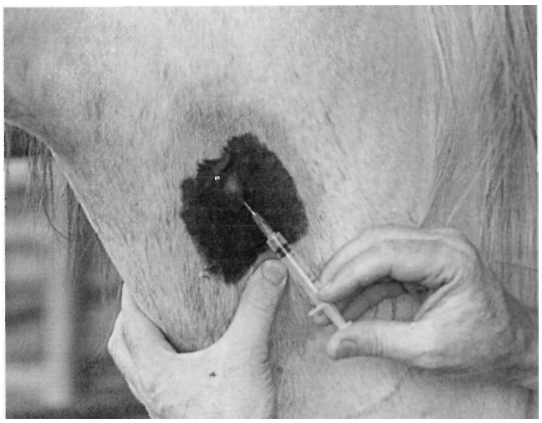


Figure 19-32. Intravenous catheterization. After skin disinfection, a bleb of local anesthetic is injected over the middle of the jugular vein.



Figure 19-33. Intravenous catheterization. With the jugular vein distended, the catheter is inserted at an angle to the vein until blood appears at the end of the stylet.



Figure 19-35. Intravenous catheterization. The catheter and extension tubing can be fixed in position with a rapidly acting glue such as Superglue or Krazy Glue.

the vein, and for longer-term fluid administration, it may be better to insert the catheter down the vein. A 12-gauge catheter is too large to insert without a stab incision in the skin. The catheter should be inserted at a 30-degree angle to the horizontal plane of the skin (Fig. 19-33), until blood is visible in the catheter stylet, indicating that the tip of the stylet is within the vein. The catheter and stylet are straightened toward a zero angle with the skin and advanced 2 to 3 cm (1 inch). The stylet is then withdrawn about 1.25 cm (0.5 inches), and the catheter is advanced up to the hub of the catheter (Fig. 19-34). Extension tubing, which has previously been filled with heparinized saline (10 IU heparin/L), is connected to the catheter, and the hub of the catheter and extension tube are fixed in place with a few drops of a rapid-setting glue such as Superglue or Krazy Glue (Fig. 19-35).

- Extendable plastic or rubber tubing is connected to the extension tubing. This extendable tubing allows the horse to move around freely within its box stall (Fig. 19-36).

KEY POINT

We find that the use of the Gyro Fluid Hanger and Stat Large Animal IV Set (International Win, Ltd., Cary, NC), shown in Figure 19-36, is helpful in the administration of large volumes of fluid with minimal supervision.

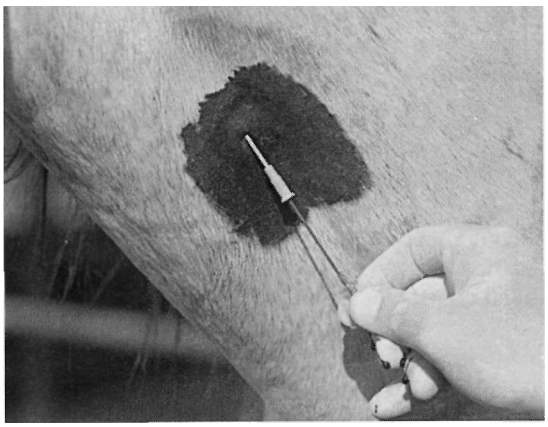


Figure 19-34. Intravenous catheterization. After the catheter and stylet are within the lumen of the vein, they are advanced for 2-3 cm (1 inch), and the stylet is withdrawn 1 cm (0.5 inches). The catheter and stylet are then inserted up the vein.

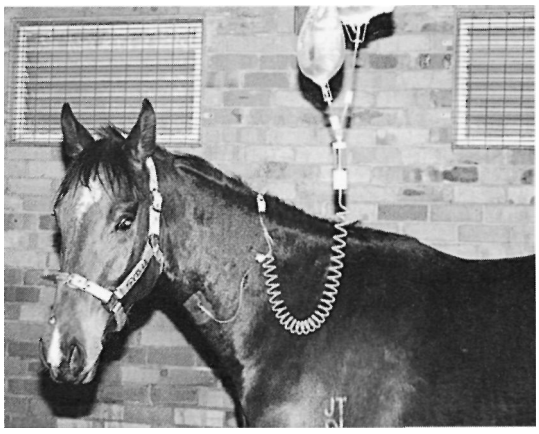


Figure 19-36. Intravenous catheterization. Connection of the extension tubing to extendable rubber or plastic tubing allows the horse to receive fluids while moving around its box stall.

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Figure 19-37. Intravenous catheterization. An alternative site to the jugular vein for intravenous catheterization is the cephalic vein. This site is particularly useful for intravenous fluid administration to foals.

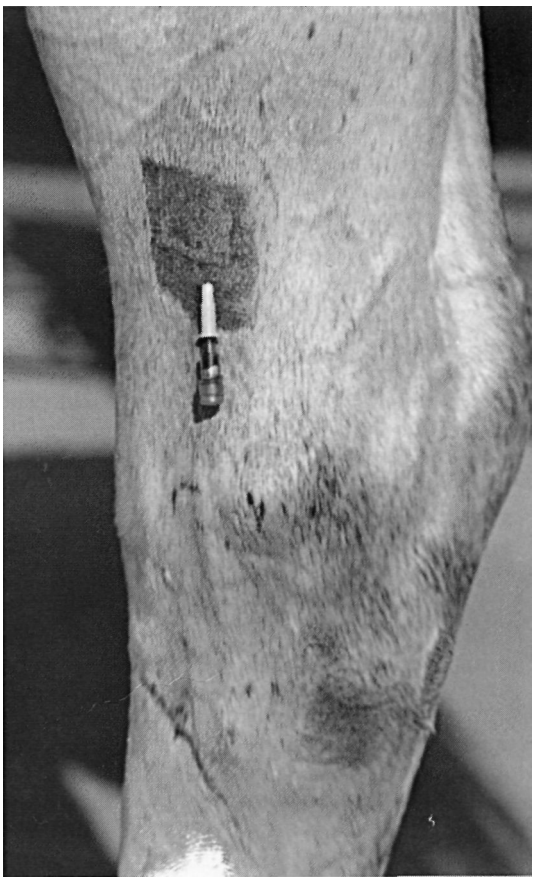


Figure 19-38. Intravenous catheterization. An alternative site to the jugular vein for intravenous catheterization is the saphenous vein. This site is more difficult and dangerous to catheterize than other available sites.

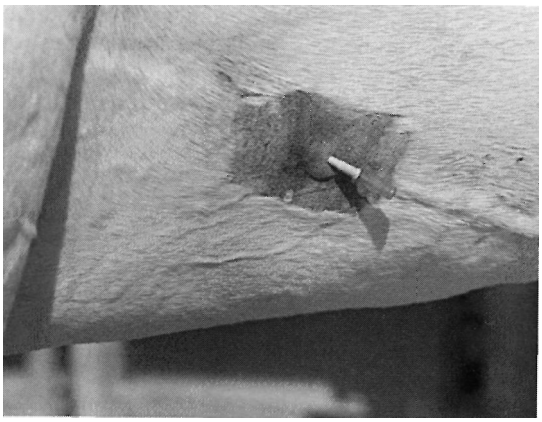


Figure 19-39. Intravenous catheterization. An alternative site to the jugular vein for intravenous catheterization is the lateral thoracic vein. Because this is a large-diameter vein, a 10- or 12-gauge catheter can be inserted if needed. The vein is located deeper than the other veins used for catheterization.

- Other veins that can be used for fluid administration include the cephalic (Fig. 19-37), saphenous (Fig. 19-38), and superficial (lateral) thoracic (Fig. 19-39) veins.

Fluid and Electrolyte Therapy

Reuben J. Rose and
David R. Hodgson

Many clinicians regard the old saying "the horse's kidney is smarter than the smartest equine clinician" as indicating that they can give whatever fluid-electrolyte solution they like and the horse will sort it all out. Although there is a little truth in this, selection of the correct fluid and route and rate of administration and deciding whether or not bicarbonate is required are critical decisions that need to be made affecting the outcome of treatment. To make these decisions, laboratory data are required, together with an accurate clinical assessment of the case. A few principles are helpful when instituting fluid therapy:

KEY POINT
For IV fluids, think in big volumes in adult horses, that is, 20 to 50 L is the usual range.

- Use a balanced electrolyte solution, usually with similar composition to plasma.
- If the peripheral pulse is weak or absent and there is prolonged capillary refill time, rapid infusion of large volumes (>20 L in 1-2 hours) of polyionic isotonic solutions is necessary.
- Do not use bicarbonate solutions unless measurements are made and plasma values are less than 15 mmol/L (mEq/L).

KEY POINT
Consider fluid administration by nasogastric tube if gastrointestinal function is normal.

Assessment of Fluid and Electrolyte Balance

Clinical assessment of hydration state can be gained from clinical examination; pulse rate and quality, capillary refill time, heart rate, and skin turgor provide a guide to the extent of fluid loss.

KEY POINT
Skin turgor is best assessed by pulling up a fold of skin over the point of the shoulder to determine how quickly the skin moves back into a normal position. This site provides more reliable results than when the skin is assessed over the neck.

Five percent dehydration is the minimum degree of dehydration that can be detected, and there are few changes in clinical signs. Moderate dehydration (7-8%) results in a thready pulse, prolonged capillary refill time to 3 to 4 seconds, and a decrease in skin turgor. Severe dehydration (10-12%) is evidenced by dry mucous membranes, prolonged capillary refill time (4-5 seconds), weak or no detectable pulse, and marked decrease in skin turgor. Laboratory aids that are available in practice and are useful in assessing fluid and electrolyte deficits and deciding on therapy are as follows:

Hematocrit (PCV) and Plasma TP. These are simply measured using the microhematocrit technique (PCV) and refractometry (TP) and provide a guide to extracellular fluid (ECF) balance. Although interpretation of increases in PCV always should be guarded as extra red cells are released if the horse is excited or in pain, a high PCV (>0.45 L/L or 45%) generally indicates reduction in ECF volume and sodium loss. The TP is unaffected by excitement and therefore, is often a better guide to ECF volume depletion, with values greater than 75 g/L (7.5 g/dL) indicating sodium and water loss. However, it should be remembered that in conditions where there is protein loss (e.g.,

diarrhea, renal disease), a horse may be dehydrated yet have a normal to low TP. Similarly, if there is a chronic infection with increases in fibrinogen and gammaglobulins, the TP may be elevated without dehydration being present. In general, TP values less than 75 g/L (7.5 g/dL) indicate less than 5% dehydration. TP values in the range 75 to 85 g/L (7.5-8.5. g/dL) indicate 5% to 8% dehydration, and values between 85 and 95 g/L (8.5-9.5 g/dL) signify 8% to 10% dehydration.

Serum or Plasma Electrolytes. These are essential measurements if accurate estimations of electrolyte losses are to be made. We prefer plasma samples to be collected because an estimate of bicarbonate concentration or total carbon dioxide concentration (TCO_2) can be performed if samples are collected into Vacutainer tubes containing lithium heparin as an anticoagulant. Electrolytes that should be measured include sodium, potassium, chloride, and bicarbonate.

Significance of Fluid and Electrolyte Alterations

The total body water (60-70% of body weight) is divided between two main compartments, the intracellular fluid (ICF) and the extracellular fluid (ECF), which vary considerably in electrolyte composition. The ECF has high sodium and low potassium concentrations, whereas the ICF has low sodium and high potassium concentrations. It should be remembered that water moves freely between the ECF and ICF

SODIUM

The average 450- to 500-kg horse has a total exchangeable sodium (Na) of approximately 14,000 mmol (mEq) (average Na 140 mmol/L and ECF volume 100 L), nearly all of which is located in the ECF.

KEY POINT

Plasma or serum sodium values do not indicate sodium deficits or excesses but are affected by water movement and changes in sodium or potassium concentrations.

This relationship has been represented by the following equation (Edelman et al., 1958):

Serum Na (mmol/L H_20 or mEq/L H_20) = (exchangeable Na + exchangeable K)/(total body water).

This equation can provide a guide to the sodium and potassium deficits and the distributions of fluid losses between compartments if the serum or plasma sodium is known and an estimate of the total body water can be made. Accurate measurement of body weight is of great value in assessing the response to therapy because fluid retention (liters) approximately equals increase in body weight (kg).

POTASSIUM

The ECF contains only 400 mmol (mEq) or less than 2% of the total body potassium (approximately 28,000 mmol), and therefore plasma and serum potassium values do not indicate total body potassium status. Although hyperkalemia (serum or plasma K values > 4.5 mmol/L) in the horse is rare, hypokalemia (serum or plasma K values < 3.0 mmol/L) is more common, particularly in horses with diarrhea.

KEY POINT

Serum or plasma potassium values greater than 4.5 mmol/L may be due to problems with sample handling or processing rather than to a pathologic process.

Delays in processing or storage of samples in the heat can result in potassium movement out of the erythrocytes or hemolysis with resulting increases in plasma potassium. Therefore, high plasma or serum potassium values should be regarded initially as suspicious of a laboratory error rather than of a disease state. A further blood sample should be collected to confirm hyperkalemia.

KEY POINT

Hypokalemia usually indicates total body potassium depletion, but the extent of depletion cannot be quantified from the extent of hypokalemia found.

For example, horses with diarrhea often have plasma or serum potassium values less than 2.0 mmol/L (mEq/L), but the extent of potassium loss may be less than half that of a horse that has had food and water deprivation for several days and has a plasma potassium value of 3.0 mmol/L (mEq/L). Plasma potassium values are also affected by acid-base status, with acidosis increasing and alkalosis decreasing plasma potassium. However, the extent of change is quite small in most clinical problems, usually less than 1.0 mmol/L.

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CHLORIDE AND BICARBONATE

Chloride and bicarbonate are the principal anions of the ECF. Chloride is located in the ECF, and changes in plasma or serum concentrations reflect changes in whole body status. Changes tend to occur in conditions where there is chloride loss, such as diarrhea, and in substantial sweat losses, such as occur during prolonged exercise. Plasma chloride and bicarbonate concentrations are inversely related.

KEY POINT

In metabolic acidosis, plasma bicarbonate concentration is decreased and chloride increased, whereas the reverse is true in metabolic alkalosis.

Bicarbonate values are most commonly measured as total CO_2 in plasma using autoanalyzers. Alternatively, true bicarbonate can be measured from the pH and Pco_2 using a blood gas machine. The total CO_2 represents the bicarbonate plus dissolved CO_2, and in most situations the bicarbonate is approximately 5% lower than the total CO_2. If blood samples are collected into lithium heparin Vacutainer tubes and kept refrigerated, bicarbonate concentrations will remain stable for up to 3 days after blood collection. Because primary respiratory acid-base alterations are rare in the conscious horse, increased total CO_2 indicates metabolic alkalosis, whereas decreased CO, is evidence of metabolic acidosis.

KEY POINT

Metabolic acidosis occurs when there is bicarbonate loss (e.g., diarrhea) or increased lactate production (e.g., shock, high-intensity exercise). In contrast, metabolic alkalosis is rarely found but may occur where there is excessive loss of gastric secretions such as in anterior enteritis, where there are extensive losses of chloride in the sweat, or where horses have been administered high doses of bicarbonate before racing.

Calculating Fluid and Electrolyte Losses

KEY POINT

A guide to the electrolyte and fluid deficits can be obtained using the equation of Edelman et al. (1958), with measurement of serum or plasma sodium and assessment of total body water.

An example of calculations that can be made of fluid and electrolyte deficits is set out below. The symbol / refers to initial value and *D* refers to dehydrated values.

A 500-kg horse is assessed from clinical findings as being 8% dehydrated due to severe diarrhea. The assessment is made on the basis of increased skin turgor, dry mucous membranes, and increased capillary refill time. Plasma sodium is 130 mmol/L (mEq/L) and plasma potassium is 3.0 mEq/L. Assumptions are that the plasma sodium value before dehydration was the mean of the normal range (134-142 mmol/L), that is, 138 mmol/L, and that the total body water is 60% of the body weight, that is, 300 L before dehydration.

If the horse was 500 kg before diarrhea, 8% dehydration would result in a loss of approximately 40 kg. We assume that 90% of the body weight loss is due to water loss, and therefore, the water loss is 36L:

Water deficit

= weight loss X 0.9

= 40 kg X 0.9

= 36 L

Total body water (TBW) before dehydration

= 300 L (60% body weight)

Total body water after dehydration

= 264 L

Serum or plasma sodium times TBW equals exchangeable Na (Na_e) plus exchangeable K (K_e) (Edelman et al., 1958), that is,

$$140 \times 300 = Na_e + K_e\ (/)$$

$$Na_e + K_e\ (/) = 42{,}000 \text{ mmol (mEq)}$$

$$Na_e + K_e\ (D) = 264 \times 130$$

$$= 34{,}320 \text{ mmol/L (mEq)}$$

Total deficit of Na + K

= 42,000 - 34,320

= 7680 mmol (mEq)

How can this deficit be apportioned between Na and K? In diarrhea, approximately 70% of Na + K loss is Na. In food and/or water deprivation, approximately 10% to 15% of Na + K loss is Na. Thus, the 7680 Na + K deficit can be apportioned as follows:

Na deficit = 5376 mmol (mEq)

K deficit = 2304 mmol (mEq)

Because virtually all exchangeable Na is in the ECF and all exchangeable K in the ICF, some calculations can be made concerning compartmental fluid distributions:

$$\text{Na content}_D = 14{,}000 \text{ mmol (mEq)} (140 \text{ X } 100 \text{ L of ECF}) — 5376 \text{ mmol (mEq)}$$

$$= 8624 \text{ mmol (mEq)}$$

$$\text{ECF}_D(\text{L}) = (\text{Na content}_D)/(\text{Na mmol/L (mEq/L)})_D$$

$$= 8624/130$$

$$= 66 \text{ L}$$

$$\text{ICF}_D = \text{TBW}_D - \text{ECF}_D$$

$$= 264 \text{ L} - 66 \text{ L}$$

$$= 198 \text{ L}$$

OVERALL DEFICITS

TBW deficit	=	40 kg X 0.9 = 36 L
ECF deficit	=	100 - 66 L = 34 L
ICF deficit	=	200 - 198 L = 2 L
Na deficit	=	5376 mmol (mEq)
K deficit	=	2304 mmol (mEq)

KEY POINT

Thus, quite substantial electrolyte deficits can exist in the face of relatively normal plasma/serum values.

- Administration of 36 L of a balanced polyionic solution (e.g., Multisol, Normosol R, Sanofi, Overland Park, KS) would provide 5040 mmol (mEq) and almost completely replace the sodium debt, whereas only 180 mmol (mEq), or less than 10%, of the potassium debt would be corrected. This is not a major problem in the short term, but if fluid therapy is required over several days and there is little potassium replacement, substantial deficits can develop that can affect cardiac, neuromuscular, and gastrointestinal function.

KEY POINT

Of major importance is the sodium deficit, because sodium controls the ECF volume.

- With substantial sodium deficits, such as in the case illustrated above, there will be decreased peripheral perfusion and signs of shock. The sodium and volume deficit correction are the chief concerns when initiating fluid and electrolyte therapy.

KEY POINT

It also should be remembered that the equine kidney has adapted to a diet rich in potassium by excreting large amounts of potassium in the urine.

- Therefore, potassium deficits may occur when a horse is not eating or does not have access to green feed. In these circumstances, the oral or nasogastric administration of potassium chloride for several days can be effective in restoring potassium balance, because this is impossible using IV fluids. Usual doses of oral potassium chloride are 50 to 100 g (2-3.5 oz), equivalent to 675 to 1350 mmol (mEq) of potassium. These doses are usually administered in several liters of water by nasogastric tube.

Route of Fluid Administration

If rapid blood volume expansion is not needed and if the gastrointestinal tract is functioning normally, oral fluids or fluids given by nasogastric tube should be considered. In horses with diarrhea, electrolyte solutions administered by nasogastric tube are well absorbed and are useful for maintaining hydration status in addition to potassium balance.

KEY POINT

Fluids administered by nasogastric tube should not be used if the horse has ileus or positive nasogastric reflux.

We have found the use of glucose-glycine-electrolyte mixtures such as Re-Sorb (SmithKline Beecham, Exton, PA), Hy-Sorb (Sanofi, Overland Park, KS), Hydra-Lyte and Isotone A (Vet-A-Mix, Shenandoah, IA), and Revive (Fermenta, Kansas City, MO), to be effective for oral or nasogastric fluid administration. The glucose and glycine enhance the uptake of fluid and electrolytes from the small intestine. Some of these mixtures were formulated for calf diarrhea but are useful for fluid and electrolyte therapy in horses when mixed with an appropriate volume of water to make an isotonic solution. Most horses will not drink these mixtures, but in adult horses, 8 to 10 L can be administered by nasogastric tube every 30 to 60 minutes until the estimated fluid loss is replaced. Fluids administered by nasogastric tube are useful for maintenance fluid therapy.

KEY POINT

Hypertonic solutions administered by nasogastric tube should be avoided in horses with hypovolemia because water will move out of other spaces into the gastrointestinal tract and worsen the hypovolemia.

Type and Volume of Fluid

Three main fluid types should be considered:

Replacement Fluids. Have a sodium composition similar to plasma. The major fluids used are polyionic, and the typical composition (mmol/L or mEq/L) would be Na 140 mmol/L, K 5 mmol/L, Mg 3 mmol/L, Cl 98 mmol/L, and bicarbonate precursors (usually acetate and gluconate) 50 mmol/L. In the past, normal saline (0.9% sodium chloride) has been used, but the main disadvantages are a high chloride content (154 mmol/L), lack of bicarbonate precursors, and higher than desired sodium (154 mmol/L).

KEY POINT

Replacement fluids should be used in situations where there has been sodium loss (hypovolemia) or where the horse is in shock.

Sterile replacement fluids are available in 3-L bags (Normosol R or Lactated Ringer's Solution Rx, Sanofi, Overland Park, KS). Alternatively, concentrated electrolyte solutions are available that can be added to sterile water to constitute 3 L of a replacement electrolyte solution. Products that are available include Lactated Ringer's Solution, Multisol-R 3X, and Equi-Lyte Concentrate (Sanofi, Overland Park, KS).

Maintenance Fluids. Have a much lower sodium but higher potassium concentration than those in replacement fluids. The typical composition (mmol/L or mEq/L) would be Na 40 mmol/L, K 13 mmol/L, Cl 40 mmol/L, acetate 16 mmol/L, and Mg 3 mmol/L, in 5% dextrose. If maintenance with intravenous fluids is required over several days, a high sodium concentration in the fluid is contraindicated. Use of a replacement fluid in these situations may not correct dehydration, because of the excessive sodium load being excreted, together with water.

Hypertonic Fluids. In horses with severe hypovolemic shock (e.g., blood loss, bowel torsion), the intravenous administration of small volumes of fluids with a high concentration of sodium (e.g., 5% NaCl) can rapidly correct the hypovolemia. This is only a short-term measure to improve cardiac output and arterial blood pressure and should not be relied on to correct the fluid deficits. A 7% saline solution is available commercially (Hyper Saline-7, Butler, Dublin, OH).

The volume and type of fluid to be administered varies with the condition requiring treatment, and some guidelines are presented in Table 19-10. In general, the flow rate of intravenous fluids should not exceed 2 L/h unless the horse is in shock. Higher flow rates will result in increased urinary excretion of fluids with poor retention.

BICARBONATE USE

Sodium bicarbonate is used to aid in treatment of conditions that result in severe metabolic acidosis.

KEY POINT

Mild to moderate metabolic acidosis itself causes little adverse effects; rather, it is the problem that caused the acidosis that should be treated.

Thus, in shock or ischemic colic, metabolic acidosis is the result of reduced tissue perfusion, with a resulting increase in lactate production. In such cases, therapy should focus on expansion of ECF volume rather than bicarbonate administration.

KEY POINT

Bicarbonate therapy is useful in cases where there has been substantial bicarbonate loss (e.g., diarrhea) or where metabolic acidosis is severe (HCO_3 < 15 mmol/L).

If bicarbonate is administered, it should be used sparingly because an equal number of milliequivalents of sodium will also be given, which may lead to additional excretion of water.

To calculate the amount of bicarbonate to be used, the following formula is generally used:

Bicarbonate required (mEq or mmol) = 0.3 X body weight (kg) X base deficit (mEq/L or mmol/L)

The amount of bicarbonate calculated is based on a deficit in the extracellular fluid (approximately 30% of body weight), which is why 0.3 is included in the formula. The base deficit is calculated by subtracting the plasma bicarbonate or total carbon dioxide from 25 mmol/L (mEq/L), which is the lower end of the normal range (25-33 mmol/L). In severe acidosis, the plasma bicarbonate concentration may fall to 10 mEq/L. Thus, the calculated bicarbonate requirement in a 500-kg

TABLE 19-10. Likely Fluid and Electrolyte Deficits and Alterations in Laboratory Data with Conditions that Cause Fluid and Electrolyte Disturbances in a Typical Adult (500-kg) Horse

	Estimated Deficits			Likely Changes in Laboratory Datat						
Clinical Problem	H_20 (L)	Na (mmol*)	K (mmol*)	PCV	TP	Na	K	Cl	$HC0_3$	Suggested Type and Amount of Fluid
Diarrhea—mild, horse drinking	15-25	800-1200	300-450	N	N				Nor	15-20 L by stomach tube of a glucose-glycine-electrolyte mix (Treatment No. 57) with added KC1 (50 g/10 L H_20)
Diarrhea—severe, horse not drinking	40-60	2000-4000	800-1600			Nor				30-40 L IV replacement fluid. 3 L of 5% $NaHC0_3$. Possibly IV plasma
Food and water deprivation 24-48 h	10-25	200-500	2000-3000				N		N	10-15 L of IV maintenance fluid supplemented by 10 L glucose-glycine-electrolyte mix by stomach tube with 75 g KC1
Colic—LI impaction for 2-3 days	15-30	500-1500	2000-3000				N	N	N	No fluids by stomach tube until some gut motility. Large volumes (50-80 L) of IV fluids, preferably maintenance, to soften mass
Colic—torsion, intussusception	20-50	2000-5000	1000-2000			N	N	N		IV replacement fluid to effect. Bicarbonate not needed in most cases. 20-40 L IV; monitor TP; slow fluids if TP <50 g/L (5 g/dL)
Postexercise dehydration (fast exercise), 3-10 km	5-10	500-1000	400-800				N	N		5-10 L of glucose-glycine-electrolyte mixture by stomach tube
Postexercise dehydration 80-160 km (endurance)	20-50	2000-5000	1000-2500			Nor			Nor	15-20 L replacement fluids IV if signs of decreased perfusion. If normal gut activity, fluids can then be given by stomach tube

LI, Large intestine; PCV, packed cell volume; TP, plasma total protein.
*mmol is the same as mEq.
tN, within the normal range; , slightly decreased; substantially decreased; , slightly increased; , substantially increased.

horse would be 0.3 X 500 X (25 - 10) = 2250 mmol (mEq). Half the calculated deficit is usually replaced so that overcorrection of the acidosis does not occur, and a further blood sample is taken for measurement of bicarbonate concentrations. Thus, in the case above, 1125 mmol (mEq) of sodium bicarbonate could be administered, which is approximately 2 L of a *5%* solution.

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Appendix 1

Dose Rates, Use, and Route of Administration of Some Drugs Commonly Used in Equine Practice

Name of Drug	Main Use	Dose and Route
Acepromazine maleate	Tranquilization	0.04-0.08 mg/kg IV or IM
Acetazolamide	Management of HYPP	2.2 mg/kg PO q6-12h
Alpha-tocopherol	White muscle disease	1.5-4.5 mg/kg POq24h
Altrenogest (allyl trenbolone)	Transitional estrus Estrus synchronization	0.044 mg/kg (1 mL/50 kg body weight) PO
Amikacin sulfate	Antibiotic: gram-negative bacteria	4-8 mg/kg IV or IM q8-12h
Aminophylline	Bronchodilatation	4-7 mg/kg PO q8h
Amoxicillin trihydrate	Antibiotic—mostly for gram-positive bacteria	10-22 mg/kg IM q12h; 20 mg/kg q6h PO
Ampicillin sodium	Antibiotic—mostly for gram-positive bacteria	10-50 mg/kg IV or IM q8h
Aspirin	Anti-inflammatory, analgesic, and antipyretic	15-100 mg/kg PO or IV q24h
Bismuth subsalicylate suspension (1.75%)	Foal diarrhea	0.5 mL/kg PO q4-6h
Boldenone undecyclenate	Anabolic (debility, anemia)	1 mg/kg IM repeated at 3-week intervals
Bromhexine	Respiratory disease	3 mg/kg PO q12h
Butorphanol tartrate	Analgesia, colic pain	0.02-0.1 mg/kg IV
Captan	Ringworm	2%-3% solution topical
Ceftiofur	Some bacterial respiratory infections	2.2-4.4 mg/kg IM q24h
Cephalothin sodium	Antibiotic—gram-positive bacteria; to be used if renal function is normal	11-18 mg/kg IM or IV q8h
Cimetidine HCl	Gastroduodenal ulcer disease	6-7 mg/kg PO, IM or IV q6h or 1000 mg q8h
Clenbuterol	Bronchodilatation—respiratory diseases	0.8 µg/kg IV, by nebulizer, or PO q12h
Cloxacillin sodium	Antibiotic for beta-lactamase staphylococci	30 mg/kg IM or IV q6-8h
Detomidine HCl	Sedative, analgesic	0.02-0.04 mg/kg IV or IM
Dexamethasone	Corticosteroid—shock, CNS injury, immune-mediated disorders	0.04-0.2 mg/kg IV, IM or PO q24h
Diazepam	Tranquilizer, seizures	Foals: 0.05-0.4 mg/kg IV; adults 25-50 mg IV (for seizures, repeat within 30 min if necessary)
Digoxin	Heart failure	0.06-0.08 mg/kg PO q8h for 5-6 doses for digitilization, then 0.01-0.02 mg/kg for maintenance

Table continued on following page

Name of Drug	Main Use	Dose and Route
Dimethylsulfoxide (DMSO)	CNS trauma, gastrointestinal disorders, topical application for local inflammation, septic arthritis	Available as 90% solution (9 g/mL). 1 g/kg as a 10-20% solution in saline IV q24h for up to 3 days. May also be used as an intraarticular infusion for septic arthritis
Dioctyl sodium sulfosuccinate	Large bowel impactions	10-20 mg/kg PO q24h
Dipyrone	Analgesic (colic) and antipyretic	11-22 mg/kg IV or IM q24h
Dobutamine	Hypotension during anesthesia	2-10 μg/kg/min IV
Epinephrine 1:1000 (Adrenaline)	For treatment of anaphylaxis	3-5 mL/450 kg IM or SC
Erythromycin lactobionate	Antibiotic, broad spectrum but bacteriostatic	3-5 mg/kg IV or IM q6-8h
Erythromycin estolate	For treatment of *R. equi* in combination with rifampin	25 mg/kg PO q8h
Erythromycin phosphate	For treatment of *R. equi* in combination with rifampin	37.5 mg/kg PO q12h
Fenbendazole	Anthelmintic	5 mg/kg PO or 10 mg/kg q24h for 5 days for *S. vulgaris* in foals. 60 mg/kg PO once for encysted cyathostomes
Flunixin meglumine	Abdominal and musculoskeletal pain, endotoxemia	0.25 mg/kg IV, IM or PO q6h or 1.1 mg/kg q12-24h
Fluprostenol	Luteolytic agent, induction of parturition, synchronize estrus	1100 μg/500 kg IM for parturition; 250 μg to synchronize estrus (repeat after 16 days)
Furosemide	Edema, renal failure, EIPH	0.25-1 mg/kg IV or IM
Gentamicin	Antibiotic—gram-negative bacteria	6.6 mg/kg q24h
Glycosaminoglycan (polysulfated)	Degenerative joint disease	250 mg intra-articular per joint per week for 3 weeks or 500 mg IM
Griseofulvin	Ringworm	10 mg/kg q24h PO for 7 days
Heparin	Abdominal surgery, laminitis, DIC	80-100 IU/kg IV q6-12h; 25-40 IU/kg q8h for low-grade DIC
hCG	Induction of ovulation	2000-3000 IU/450 kg IV
Hyaluronate sodium	Traumatic and degenerative joint disease	10-60 mg intra-articular
Hydrocortisone sodium succinate	Shock	1-4 mg/kg slow IV infusion q6-12h
Iodochlorhydroxyquin	Chronic diarrhea	20 mg/kg PO q24h for 3 days; reduce dosing frequency thereafter
Isoxsuprine hydrochloride	Navicular disease	0.6-1.2 mg/kg PO q12h
Ivermectin	Internal parasites, ectoparasites	0.2 mg/kg PO once for internal parasites or 0.2 mg/kg q96h for lice and mange
Kanamycin	Antibiotic for gram-negative infections	5 mg/kg IV q8h
Ketamine	Anesthesia induction	2.2 mg/kg IV after xylazine 1.1 mg/kg IV
Ketoprofen	Anti-inflammatory, abdominal pain, endotoxemia (for ketoprofen uses)	1.1-2.2 mg/kg IV or IM q24h
Levothyroxine	Hypothyroidism	10 mg in 70 mL of corn syrup PO q24h

Name of Drug	Main Use	Dose and Route
Lidocaine, 2% solution	Local anesthesia, treatment of arrhythmias	As required for local anesthesia; for epidural, 5-8 mL; for arrhythmias, 1-1.5 mg/kg SC or IV
Malathion	Ectoparasites	0.5% topical wash
Mannitol	CNS trauma, renal failure	0.25-2 g/kg as a 20% solution IV (slowly)
Mebendazole	Internal parasites and lungworm	9 mg/kg PO
Meclofenamic acid	Laminitis and musculoskeletal pain	2.2 mg/kg PO ql2h
Meperidine	Analgesia	2.2-4 mg/kg 1M or IV q8-12h
Methadone	Analgesia only use in combination with sedatives such as xylazine or detomidine HCl	0.05-0.15 mg/kg IV after prior sedation
Methicillin	Antibiotic for beta-lactamase staphylococci	25 mg/kg IV or IM q6h
Methylprednisolone acetate	Synovitis, degenerative joint disease	40-160 mg intra-articular per joint
Methylprednisolone sodium succinate	Shock, anti-inflammatory, urticaria	10-20 mg/kg IV
Metoclopramide	Ileus	0.02-0.1 mg/kg IV q6-8h
Metronidazole	Antibiotic for anaerobic infections	15 mg/kg PO q6h
Mineral oil	Gastrointestinal lubrication or treatment of laminitis	5-10 mL/kg PO q12-24h
Morphine	Analgesic; only use in combination with sedatives such as xylazine and detomidine	0.2-0.6 mg/kg IV (slowly)
Moxidectin	Anthelmintic	0.4 mg/kg PO
Naproxen	Musculoskeletal pain	10 mg/kgPO or IV q12-24h
Neomycin sulfate	Antibiotic—gram-negative bacteria	4-7.5 mg/kg IV or IM q12h
Neostigmine	Ileus	0.02 mg/kg SC q30min
Omeprazole	Gastroduodenal ulcer disease	0.7-1.4 mg/kg POq24h
Oxacillin sodium	Antibiotic for beta-lactamase staphylococci	25-50 mg/kg IV or IM q12h
Oxfendazole	Anthelmintic	10 mg/kg PO
Oxibendazole	Anthelmintic	10-15 mg/kg PO
Oxytetracycline	Bacteriostatic antibiotic; use where sensitivity tests indicate only effective drug	5-10 mg/kg IV q12h
Oxytocin	Induction of parturition; retained placenta	20-40 IU IV for induction of parturition; or 80-100 IU in 500 mL normal saline IV
Penicillin G, procaine	Antibiotics—gram-positive infections	15,000 lU/kg or 15 mg/kg IM only q12h
Penicillin G, Na or K	Antibiotics—gram-positive infections	20,000 IU/kg or 12 mg/kg IM or IV q6h
Pentazocine	Analgesic, only use in combination with sedatives, such as xylazine or detomidine HC1	0.4-0.8 mg/kg IV
Pentosan sulfate	Degenerative joint disease	250 mg intra-articular q7-14d
Phenoxybenzamine hydrochloride	Diarrhea, laminitis; should not be used if already hypotensive	0.66 mg/kg in 1500 mL normal saline IV

Table continued on following page

Name of Drug	Main Use	Dose and Route
Phenylbutazone	Anti-inflammatory, musculoskeletal problems, prevention of postoperative swelling	2.2-4.4 mg/kg PO or IV q12h or q24h. Dose of 4.4 mg/kg q12h should not be used for more than 1 day
Phenytoin sodium	Chronic low-grade rhabdomyolysis, stringhalt	10-12 mg/kg PO q12h for 3-4 days, then 10-12 mg/kg q24h for 3-4 days, then 5-6 mg/kg q24h
Piperazine	Anthelmintic, for *Parascaris equorum*	110 mg/kg PO
Potassium chloride	Potassium replacement therapy	30-60 g (400-800 mmol or mEq K) in 4 L water by nasogastric tube
Prednisolone sodium succinate	Anti-inflammatory therapy, shock, purpura	0.2-3 mg/kg IV q6-12h
Prednisolone	Low-grade respiratory disease, dermatitis, pemphigus, autoimmune disorders	Initial dose rate 1 mg/kg PO q12h for 3-4 days, then 1 mg/kg q24h for 3-4 days; maintenance 0.5-1 mg/kg q24h every other day
Promazine	Tranquilization	0.4-1.0 mg/kg IV or PO or 1-2 mg/kg oral form
Propantheline bromide	To reduce rectal straining when undertaking rectal examination	0.06 mg/kg IV
Proparacaine	Topical analgesia for eye problems	0.5% solution, ophthalmic drops
Psyllium mucilloid	Sand impaction of the large bowel	0.5 kg in 6-8 L of water PO q24h
Pyrantel pamoate	Anthelmintic	6.6 mg/kg PO
Quinidine sulfate	Atrial fibrillation	20 mg/kg by nasogastric tube q2-3h until cardioversion (not to exceed 160 mg/kg in 12 h)
Quinidine gluconate	Atrial fibrillation	1.5 mg/kg IV q10min until cardioversion (not to exceed 40 mg/kg)
Ranitidine	Gastroduodenal ulcer disease	0.5 mg/kg PO or IV q12h
Rifampin	Use in combination with erythromycin to treat *R.. equi*	5 mg/kg PO q8h
Romifidine	Sedative	0.08-0.12 mg/kg
Stanozolol	Anabolic—debility and anemia	0.55 mg/kg IM q7-14d
Streptomycin	Antibiotic; few equine pathogens are sensitive	2 mg/kg IM q12h
Sucralfate	Used as a gastric protectant in gastroduodenal ulcer disease	30 mg/kg PO q6-8h
Theophylline	Bronchodilator	1 mg/kg PO q6h
Trichlorfon	Anthelmintic for treatment of *Gastrophilus* spp.	40 mg/kg PO
Trimethoprim-sulfadiazine	Antibiotic; respiratory infections	15-20 mg/kg IV or PO q8-12h
Tripelennamine hydrochloride	Antihistamine	1 mg/kg IM or IV (slowly) q8-12h
Xylazine	Sedative	0.2-1.0 mg/kg IV (repeated as necessary)
Yohimbine	Antagonist for xylazine	0.075 mg/kg IV (slowly) for reversal of xylazine or detomidine HCl sedation

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Appendix 2

Reference Values for Serum or Plasma Biochemical Measurements (see also Table 16-2)

Item*	Reference Range
Plasma biochemistry	
Sodium (mmol/L or mEq/L)	132-142
Potassium (mmol/L or mEq/L)	3.2-4.2
Chloride (mmol/L or mEq/L)	94-104
Bicarbonate or total CO_2 (mmol/L or mEq/L)	26-34
Osmolality (mOsmol/kg)	276-290
Urea nitrogen (mmol/L)	4-8 (11.2-22.4 mg/dL) nitrogen
Creatinine (μmol/L)	100-160(1.1-1.8 mg/dL)
Glucose (mmol/L)	4.1-6.4 (75-115 mg/dL)
Bilirubin (μmol/L)	10-50 (0.6-2.9 mg/dL)
Iron (μmol/L)	13.1-25.1 (73-140 mg/dL)
Total protein (g/L)	55-75 (5.5-7.5 g/dL)
ALP, alkaline phosphatase (U/L)	138-251
AST, aspartate amino transaminase (U/L)	160-412
CK, creatine kinase (U/L)	60-330
GGT, gamma glutamyl transferase (U/L)	10-40
Calcium (mmol/L)	2.7-3.3 (10.8-13.2 mg/dL)
Phosphate (mmol/L)	0.75-1.25 (2.3-3.9 mg/dL)
Cholesterol (mmol/L)	2.1-3.6 (81-139 mg/dL)
Uric acid (μmol/L)	0-60 (0-1 mg/dL)
Triglycerides (mmol/L)	0.06-0.61 (5.3-54.0 mg/dL)
Serum biochemistry†	
Albumin (g/L)	28-36 (2.8-3.6 g/dL)
Alphaglobulin (g/L)	7-17 (0.7-1.7 g/dL)
Betaglobulin (g/L)	6-20 (0.6-2.0 g/dL)
Gammaglobulin (g/L)	8-16 (0.8-1.6 g/dL)

*Using automated techniques (SMAC Autoanalyzer, Technicon, NY).
†Protein electrophoresis.

Appendix 3

Treatment Numbers

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Trade Names and Formulations of Commonly Used Drugs

The treatment numbers in this table refer to numbers given in the text of each of the chapters, where mention of a drug is given. Dose rates for the drugs shown here are provided in Appendix 1.

No.	Drug	Trade Name and Company	Form	Presentation	Concentration	Route
1	Acepromazine maleate	PromAce (Fort Dodge)	Solution	50-mL vial	10 mg/mL	IV, IM, or SC
2	Acetylcysteine	Mucomyst (Bristol)	Solution (10%)	4-, 10-, and 30-mL bottles	100 mg/mL	Aerosol, PO
3	Allyl trenbolone (altrenogest)	Regu-Mate (Hoechst)	Solution (0.22%)	1-L bottles	2.2 mg/mL	PO
4	Amikacin sulfate	Amiglyde-V (Fort Dodge)	Solution	48-mL vials (250 mg/mL) and 50-mL vials (50 mg/mL)	50 or 250 mg/mL	IV, IM, or intrauterine
5	Aminophylline	Aminophylline Inj. (Abbott)	Solution	10- and 20-mL vials	250 mg/mL	IV
6	Ampicillin sodium	Amp-Equine (SK Beecham)	Powder, reconstitute with saline	1- and 3-g vials	300 mg/mL	IV or IM
7	Amphotericin B	Fungizone (Squibb)	Lotion, cream, or ointment	30-mL bottle, 20-g tubes	30 mg/mL or 30 mg/g	Topical
		Fungizone Intravenous (Squibb)	Freeze-dried powder	Vial	50-mg/vial	IV
8	Antibiotic-corticosteroid ointments	Amphoderm Ointment (Aveco-Fort Dodge)	Ointment and solution	7.5- and 15-g tubes; squeeze bottles of 227 g	5 mg/g of each amphomycin and kanamycin and 10 mg/g hydrocortisone	Topical
		AnimaxDerma-4Dermalone (Pharmaderm SKB Vedco)	Ointment	7.5-, 15-, and 30-mL tubes and 240-mL bottles	Nystatin 100,000 U/mL, neomycin 2.5 mg/mL thiostrepton 2500 U/mL triamcinolone 1 mg/mL	Topical

		Forte-Topical (Upjohn)	Suspension	10-mL tubes	Hydrocortisone acetate and succinate 2 and 1.25 mg/mL, plus neomycin 25 mg/mL, penicillin 10,000 IU/mL, polymyxin B 5000 U/mL	Topical
		Kymar Ointment (Schering-Plough)	Ointment	57-g tubes	Neomycin 3.5 mg/g, trypsin-chymotrypsin 10,000 U/g, hydrocortisone 2.5 mg/g	Topical
		Liquichlor (Evsco)	Ointment	10-mL tubes and 12 fl oz (355-mL) bottles	Chloramphenicol 4.2 mg/mL, prednisolone 1.7 mg/mL, tetracaine 4.2 mg/g	Topical
		Neo-Predef Sterile Ointment (Upjohn)	Ointment	3.5- and 5-g tubes	Isoflupredone acetate 1 mg/g, neomycin sulfate 5 mg/g	Topical
		Tritop (Upjohn)	Ointment	10-g tubes	Isoflupredone acetate 1 mg/g, neomycin sulfate 5 mg/g, tetracaine hydrochloride 5 mg/g	Topical
9	Aspirin	Aspirin Boluses (Veterinary)	Bolus/tablet	Boxes of 50 boluses	240 grains (15.6 g) per tablet	PO
		Aspri-Ject Injection (Vedco)	Solution	30-mL vial	100 mg sodium salicylate/mL	IV
10	Atropine sulfate	Atrophate (Schering-Plough)	Ointment	3.5-g tubes	10 mg atropine/g	Ophthalmic
11	Atropine	Atropine Injectable L.A. (Fort Dodge)	Solution	100-mL vial	2 mg/mL	IV, SC
		Atropine L.A. (Butler)	Solution	100-mL vial	15 mg/mL	IV, SC
		Atropine Sulfate (J.A. Webster)	Solution	30- and 100-mL vials	0.5 mg/mL	IV, SC
		Atropine Sulfate Injection L.A. (Vedco)	Solution	100-mL vial	15 mg/mL	IV, SC
12	Betamethasone acetate, betamethasone sodium phosphate	Betavet Soluspan (Schering-Plough)	Suspension	5-mL vial	12 mg/mL betamethasone acetate and 2 mg/mL betamethasone sodium phosphate	IM, IA

Table continued on following page

Trade Names and Formulations of Commonly Used Drugs—*Continued*

No.	Drug	Trade Name and Company	Form	Presentation	Concentration	Route
13	Betamethasone	Betasone (Schering)	Suspension	5-mL vial	5 mg/mL betamethasone as B. diproprionate and 2 mg/mL as B. sodium phosphate	IM, IA
14	Buprenorphine	Buprenex (Norwich Eaton)	Solution	1-mL ampules	0.3 mg/mL	IV
15	Butorphanol tartrate	Torbugesic (Fort Dodge)	Solution	10- and 50-mL vials	10 mg/mL	IV
16	Cefotaxime sodium	Claforan (Hoechst)	Powder	1-, 2-, and 10-g vials		IV
17	Captan	Available from agricultural and garden supply stores (Various manufacturers)	Powder	Make up a 3% solution for topical use in dermatophytosis	45 g/100 g powder	Topical
18	Ceftiofur sodium	Naxcel (Upjohn)	Powder	1- and 4-g vials	Reconstitute solution to 50 mg/mL	IV
19	Cefazolin sodium	Ancef, Kefzol, Zolicef (SKB, Lilly and Bristol)	Powder	0.25-, 0.5-, 1-, 5-, and 10-g vials	Reconstitute with dextrose 5% in water	IV
20	Cephalothin sodium	Kenin (Lilly)	Powder	1-, 2-, and 20-g vials	Reconstitute with dextrose 5% in water	IV
21	Cephapirin sodium	Cefadyl (Bristol)	Powder	500 mg, 1-, 2-, 4-, and 20-g vials	Reconstitute with dextrose 5% in water	IV
22	Chloral hydrate	Activated chloral (Various manufacturers)	Crystals	1- and 5-lb containers	Make up appropriate solution	PO
23	Chlorhexidine citrate	Chlorhex Surgical Scrub (Vedco)	Solution	128-fl oz and 1-gal containers	20 mg/mL	Topical
24	Chlorhexidine diacetate	Chlorhexiderm Shampoo (DVM)	Solution	8-fl oz and 1-gal containers	5 mg/mL	Topical
25	Chorionic gonadotrophin (hCG)	Various manufacturers	Freeze-dried powder	10-mL vial	5000, 10,000 or 20,000 U per vial	IV
26	Cimetidine	Tagamet (SKF)	Tablets and solution	For injection, 150 mg/2 mL	200-, 300-, 400-, and 800-mg tablets; 60-mg/mL solution	PO or IV
27	Clenbuterol	Ventipulmin Granules (Boehringer Ingelheim)	Granules	500-g container	0.016 mg/g	PO
		Ventipulmin Injection (Boehringer Ingelheim)	Solution	50-mL vial	0.03 mg/mL	IV, IM or by nebulizer

28	Detomidine hydrochloride	Dormosedan (SKB)	Solution	5- and 20-mL vials	10 mg/mL	IV or IM
29	Dexamethasone	Various manufacturers (Vedco, Schering, Butler, Durvet, Webster, Lexton, ProLabs)	Powder	30-, 50-, and 100-mL vials	2 mg/mL	IV
30	Dexamethasone sodium phosphate	Various manufacturers (Steris, Webster, Phoenix, Vedco)	Solution	50- and 100-mL vial	4 mg/mL	IV
31	Dialdehyde glutaraldehyde	Cidex (Surgikos)	Solution	Quart, gallon, and 2.5-gal bottles		Instrument disinfection
		Cidex-7 (Surgikos)	Solution	Quart, gallon, and 5-gal bottles	2% solution plus vial of activator	Instrument disinfection
32	Diazepam	Valium Injectable (Roche)	Solution	2-mL ampules and 10-mL vials	5 mg/mL	IV or IM
33	Digoxin	Lanoxin Tablets, Veterinary (Burroughs Wellcome)	Tablet	Bottles of 100 and 1000 tablets	Total mg/tablet: yellow 0.125 and white 0.25	PO
		Lanoxin (Burroughs Wellcome)	Solution	2-mL ampule	0.25 mg/mL	IV
34	Dimethylsulfoxide (DMSO)	Domoso Gel (Syntex)	Gel	60- and 120-g collapsible tubes, and 425-g jars	900 mg/mL	Topical
		Domoso Solution (Syntex)	Solution	4 fl oz with or without sprayer and 1-pt (16-fl oz) and 1-gal (128-fl oz) containers	900 mg/mL	Topical, IV as 10-20% solution, or PO
35	Dioctyl sodium sulfosuccinate (DSS)	Dioctynate (Butler)	Solution	1-gal container	50 mg/mL	Nasogastric tube
36	Dipyrone monohydrate	Dipyrone injection (Various manufacturers—Lextron, RX, Phoenix, Vedco, Butler)	Solution (50%)	100- and 250-mL vial	500 mg/mL	IM or IV
37	Enilconazole	Clinifarm EC (Sterwin)	Solution	750-mL container; to use, dilute 1:100	13.8 mg/mL	Topical
38	Erythromycin estolate	Ilosone (Dista)	Suspension	100-mL bottles	25 or 50 mg/mL	PO
39	Erythromycin ethylsuccinate	Various manufacturers	Suspension	100-, 200-, 480-, and 500-mL bottles	40 or 80 mg/mL	PO
		Various manufacturers	Powder	50-, 60-, 100-, and 200-mL bottles	40 and 80 mg/mL	PO
40	Erythromycin gluceptate	Ilotycin Gluceptate (Dista)	Solution	250-, 500-mg and 1-g vials		IV

Table continued on following page

Trade Names and Formulations of Commonly Used Drugs—*Continued*

No.	Drug	Trade Name and Company	Form	Presentation	Concentration	Route
41	Eye antibiotic: neomycin-bacitracin-polymyxin	Neobacimix (Schering), Trioptic-S **(SKB),** Vetropolycin (Pitman-Moore)	Ointment	3.5-g tube	3.5 mg/g neomycin, 400 U/g bacitracin, 10,000 U polymyxin B sulfate/g	Eye, topical
42	Eye antibiotic: chloramphenicol	Chloromycetin (Parke Davis)	Eye drops and eye ointment	3.5-g tube and 5-mL bottle	1% (ointment) and 5 mg/mL (drops)	Eye, topical
		Chloroptic (Allergan)	Eye drops	2.5- and 7.5-mL bottles	5 mg/mL	Eye, topical
43	Eye antibiotic: gentamicin	Garramycin and Gentacin Ophthalmic (Schering)	Eye drops and eye ointment	3.75-g tubes and 5-mL bottles	3 mg/mL or 3 mg/g	Eye, topical
44	Eye antibiotic: tobramycin	Tobrex (Alcon)	Eye drops and eye ointment	5-mL bottle and 3-g tube	3 mg/mL or 3 mg/g	Eye, topical
45	Eye antifungal: natamycin	Natacyn (Alcon)	Eye drops	15-mL bottle	50 mg/mL	Eye, topical
46	Eye antifungal: miconazole	Monistat-i.v. (Janssen)	Solution	20-mL ampule	10 mg/mL	Eye, topical
47	Eye corticosteroids: dexamethasone	Maxidex (Alcon)	Eye drops	5- and 15-mL bottles	1 mg/mL	Eye, topical
48	Eye nonsteroidal: flurbiprofen sodium	Ocufen (Allergan)	Eye drops	2.5-, 5-, and 10-mL bottles	3 mg/mL	Eye, topical
49	Eye corticosteroids: betamethasone + gentamicin	Gentocin, Durafilm (Schering Plough)	Eye drops	5-mL bottle	Gentamicin 3 mg/mL and 1 mg/mL betamethasone acetate	Eye, topical
50	Eye corticosteroids + chloramphenicol	Chlorasone (Evsco)	Eye ointment	3.5-g tube	2.5 mg/g prednisolone acetate and 10 mg/g chloramphenicol	Eye, topical
51	Fenbendazole	Panacur Granules 22.2% (Hoechst)	Granules	20 X 5.2-g packets per box	222 mg/g	PO
		Panacur Paste 10% (Hoechst)	Paste	25-g syringe	100 mg/g	PO
		Panacur Suspension 10% (Hoechst)	Suspension	1-L bottle	100 mg/mL	PO
52	Flunixin meglumine	Banamine Granules (Schering)	Granules	250-mg in 10-g packets	250mg/10g	PO
		Banamine Paste (Schering)	Paste	30-g syringe	1500 mg/30g	PO
		Banamine solution (Schering)	Solution	50- and 100-mL vials	50 mg/mL	IV or IM

53	Fluprostenol sodium	Equimate (Haver/ Diamond)	Solution	5-mL vial	50 mg/mL	IM
54	Furosemide as monoethanol amine	Disal Injection (Fermenta)	Solution	50- and 100-mL vials	50 mg/mL	IV or IM
55	Furosemide as a diethanolamine salt	Furos-A-Vet (Anthony), Furosemide Injection (Vedco, Webster, Phoenix, ProLabs, Butler, Hoechst)	Solution	30-, 50-, and 100-mL vials	50 mg/mL	IV or IM
56	Gentamicin sulfate	Gentocin Solution (Schering)	Solution	50- and 100-mL vials	50 mg/mL	IV, IM, or intrauterine
57	Glucose-glycine and electrolytes	Hydra-Lyte (Vet-A-Mix)	Soluble powder	5.76-oz (163.4-g) two-compartment foil packets	16 mM glycine, 368 mM and 30, 85, 60 and 45 mEq/L of glucose, K, Na, acetate, and citrate, respectively	PO
		Life-Guard (SKB)	Soluble powder	200-g twin pack	Dextrose 56.76% and glycine 3.12%; total mEq/L when reconstituted: 105 Na, 80 HCO_3, 51 CI, 26 K, 10 Ca, 6 $P0_4$, 6 Mg and 4 $S0_4$	PO
		Re-Sorb (SKB)	Soluble powder	Boxes containing 12 packets	Glucose 44.0 g and glycine 6.36 g and electrolytes	PO
		Revive (Fermenta)	Soluble powder	5.73-oz (162.5-g) packets (cartons of 12 or 50 per pail)	Dextrose and glycine and electrolytes	PO
		Survive (Lextron)	Soluble powder	5.73-oz (162.5-g) packets (48 and 50 packets)	Dextrose and glycine and electrolytes	PO
		Survive (RX)	Soluble powder	5.73-oz (162.5-g) packets (48 packets)	Dextrose and glycine and electrolytes	PO
58	Gonadotropin-releasing hormone-gonadorelin diacetate tetrahydrate	Cystorelin (Ceva)	Solution	Single and multidose 10-mL vials (only registered for use in cattle; probably not effective in horses)	50 (xg/mL	IM or IV
59	Heparin sodium	(Various manufacturers: Upjohn, Winthrop, Organon)	Solution	1- and 10-mL vials	1000, 2000, 5000, 10,000, 20,000 and 40,000 LV niL	IV

Table continued on following page

Trade Names and Formulations of Commonly Used Drugs—*Continued*

No.	Drug	Trade Name and Company	Form	Presentation	Concentration	Route
60	Hyaluronate sodium	Equron (Solvay)	Solution	2-mL glass syringe	5 mg/mL	IA
		Hyalovet (Fort Dodge)	Solution	2-mL syringe or 2-mL vial	10 mg/mL	IA
		Hylartin V (American Equine)	Solution	2-mL glass syringe	10 mg/mL	IA
		Synacid (Schering-Plough)	Solution	5-mL single-dose vial	10 mg/mL	IA
61	Isoxsuprine hydrochloride	Voxuprine (Major)	Tablets	Bottles of 100 tablets	10- and 20-mg tablets	PO
		Vasodilan (Bristol Meyers USP)	Tablets	Bottles of 100, 500, and 1000 tablets	10- and 20-mg tablets	PO
		Circulon (Vetsearch)	Paste	230-mL jars of paste	40 me/mL	PO
62	Ivermectin	Eqvalan Liquid (MSD-AgVet)	Solution	50- and 100-mL bottles	10 mg/mL	PO
		Eqvalan Paste (MSD-AgVet)	Paste	0.21-oz (6.08-g) syringe	1.87%	PO
63	Kanamycin sulfate	Kantrim (Aveco-Fort Dodge)	Solution	50-mL bottle	50 and 200 mg/mL	IV and IM
64	Ketamine hydrochloride	Ketaset (Aveco-Fort Dodge), Vetalar (Park-Davis)	Solution	10-mL vial	100 mg/mL	IV
65	Ketoconazole	Nizoral (Janssen)	Tablets	Bottles of 100	200-mg tablets	PO
66	Ketoprofen	Ketofen (Aveco-Fort Dodge)	Solution	50- and 100-mL bottles	100 mg/mL	IV
67	Lidocaine hydrochloride	Lidocaine 2% Inject. (Various manufacturers: Butler, Pro-Labs, Phoenix, Vedco)	Solution	100-mL vial	20 mg/mL	Epidural, nerve block, infiltration
68	Mannitol	Osmitrol (Baxter)	Solution	250-, 500-, 1000-mL bottles	5%, 10%, 20% solution	IV
69	Meclofenamic acid	Arquel (Fort Dodge)	Granules	10-g packet	50 mg/g	PO
70	Medroxy-progesterone acetate	Depo-Provera (Upjohn)	Suspension	2.5- and 10-mL vials	100 or 400 mg/mL	IA
71	Meperidine hydrochloride	Demerol (Winthrop-Breon)	Solution	Ampules (single dose)	25, 50, 75, and 100 mg	IV

72	Mepivacaine hydrochloride	Carbocaine-V (Upjohn)	Solution	50-mL vial	20 mg/mL	Nerve block, IA, epidural, infiltration
73	Methadone hydrochloride	Dolophine (Lilly)	Solution	1-mL ampule	10 mg/mL	IV
74	Methylprednisolone acetate	Depo-Medrol (Upjohn)	Suspension	20 mg/mL in 10- and 20-mL vials or 40 mg/mL in 5-mL vial	20 or 40 mg/mL	IM and IA
75	Metronidazole	Flagyl (Searle)	Tablets	Bottles of 50, 100, 500, and 1000 tablets	250- or 500-mg tablets	PO
		Metrozole (Metro Med)	Tablets	Bottles of 100 and 250 tablets	250- or 500-mg tablets	PO
		Metryl (Lemmon)	Tablets	Bottles of 100 and 500 tablets	250- or 500-mg tablets	PO
76	Miconazole	Monistat (Janssen)	Solution	20-mL ampule	10 mg/mL	IV
77	Mineral oil	Mineral Oil (Parke-Davis, Fisons, Kremers-Urban)		1-qt and 1-gal container		Nasogastric tube
78	Moxidectin	Quest (Fort Dodge)	Oral gel	Single treatment syringe		
79	Oxymorphone	Numorphan (Dupont)	Solution	1- and 10-mL vials	1 and 1.5 mg/mL	IV
80	Orgotein	Palosein (Coopers)	Powder	Single-dose vials with 2 mL saline ampule for reconstruction	5 mg/vial	IM or IA
81	Oxytetracycline	Various manufacturers: Agricyl (Anthony); Oxy-tet (Anchor); Agrimycin (Agri-Labs); Anaject (Farmtech); Bio-Mycin (Bio-Ceutic); Duramycin (Durvet);	Solution	100-, 250-, and 500-mL bottle	50 and 100 mg/mL	IV
82	Oxytocin	(Various manufacturers: Lextron, Webster, RX, Anthony, Durvet, Phoenix, ProLabs, Vedco)	Solution	30- and 100-mL vials	20 USP U/mL	IV, IM, or SC
83	Penicillin G benzathine, penicillin G procaine	Various manufacturers: Ambi-Pen (Butler); Crystiben (Solvay); Dual-Pen (Fermenta); Durapen (Vedco)	Suspension	100- and 250-mL vial	150,000 U/mL penicillin G benzathine and 150,000 U/mL penicillin G procaine	IM

Table continued on following page

Trade Names and Formulations of Commonly Used Drugs—*Continued*

No.	Drug	Trade Name and Company	Form	Presentation	Concentration	Route
84	Penicillin G procaine	Various manufacturers: Agri-Cillin (Agri Laboratories); Aquacillin (Vedco); Pen-Aqueous (Durvet); Fermicillin Aqueous (Fermenta); Penicillin G Procaine (Hanford, Butler, SKB)	Suspension	100-, 250-, and 500-mL vials	300,000 U/mL	IM
85	Penicillin G, potassium	Pfizerpen Injection (Roerig)	Buffered powder	Vials	1,000,000, 5,000,000 or 20,000,000 U	IV or IM
86	Penicillin G, sodium	Various manufacturers		Vials	1 or 5,000,000 U	IV or IM
87	Pentazocine lactate	Talwin-V (Winthrop)	Solution	10-mL vial	30 mg/mL	IV
88	Phenobarbital	Luminal Injection (Winthrop-Breon)	Solution	1-mL ampule	130 mg/mL	IV
		Solubarb (Forest)	Tablets	Bottles of 24 tablets	16-mg tablets (0.25 grains)	PO
89	Phenylbutazone	Butatron Osborne Gel (Sanofi)	Gel	4 g/30g gel in oral syringe	4 g/30 g gel	PO
		Butazolidin Paste (Coopers)	Paste	Syringe	6 g- or 12 g/syringe	PO
		Butazolidin (Coopers)	Tablet or solution	Bottles of 100 tablets or 100-mL vials	1 g/tablet or 200 mg/mL solution	PO or IV
90	Phenytoin sodium	Dylantin (Parke-Davis)	Capsule	Bottles of 1000 capsules	100 mg/capsule	PO
91	Polysulfated glycosamino-glycans	Adequan LA. and Adequan I.M. (Luitpold)	Solution	1-mL glass vial and 5-mL glass vial	250 mg/mL (1 mL vial); 100 mg/mL (5 mL vial)	IA or IM
92	Povidone-iodine	Betadine Aerosol Spray (Purdue Frederick)	Solution	3-fl oz (89-mL) aerosol bottles	50 mg/mL	Topical
		Betadine Solution (Purdue Frederick)	Solution	16- and 32-oz and 1-gal bottles	50 mg/mL	Topical
		Betadine Surgical Scrub (Purdue Frederick)	Solution	16- and 32-oz and 1-gal bottles	75 mg/mL	Topical
		Groom Rite Iodine Shampoo (Pro Vet)	Solution	8-oz and 1-gal bottles	20 mg/mL	Topical
		Iodine Shampoo (Evsco)	Solution	12-fl oz (355-mL) bottle	20 mg/mL	Topical

		Lanodine (Butler)	Solution	8- and 16-oz bottles	100 mg/mL	Topical
		Pro Vet Povidone-Iodine Ointment (Pro Vet)	Ointment	8- and 16-oz jars	100 mg/mL	Topical
		Pro Vet Povidone-Iodine Solution (Pro Vet)	Solution	1-gal plastic jug	100 mg/mL	Topical
		Pro Vet Povidone-Iodine Surgical Scrub (Pro Vet)	Solution	1-gal plastic jug	75 mg/mL	Topical
		Prodine Scrub (Phoenix)	Solution	1-gal bottle	75 mg/mL	Topical
		Prodine Solution (Phoenix)	Solution	1-gal bottle	100 mg/mL	Topical
		Vetadine Scrub (Vedco)	Solution	1-gal bottle	75 mg/mL	Topical
		Vetadine Solution (Vedco)	Solution	1-gal containers	100 mg/mL	Topical
93	Prednisolone	Delta-Cortef (Upjohn)	Tablets	Bottles of 100 and 500 tablets	5 mg tablets	PO
		Meticorten (Schering); Orasone (Reid-Rowell); Deltasone (Upjohn)	Tablets	Bottles of 100 tablets	1-, 5-, 10-, 20-, and 50-mg tablets	PO
94	Prilocaine hydrochloride	Citanest (Astra)	Solution	1.8-mL cartridge	4% solution	Nerve blocks, infiltration, IA
95	Promazine hydrochloride	Promazine Granules (Fort Dodge)	Granules	10.25-oz containers	8 g/10.25 oz	PO
		Tranquazine (Anthony)	Solution	30- and 100-mL bottles	50 mg/mL	IV
96	Propantheline bromide	Pro-Banthine (Searle)	Tablets	100 and 500 tablets per bottle	7.5- or 15-mg/tablet	Oral form only available in US
97	Proparacaine	Ophthaine (Solvay)	Solution (5%)	15-mL bottle with dropper	5 mg/mL	Eye, topical
98	Psyllium mucilloid	Metamucil (Procter and Gamble)	Powder	Jars	7-, 14-, and 21-oz jars	PO
99	Quinidine sulfate	Cin-Quin (Reid Rowell)	Tablets	Bottles of 100 and 1000 tablets	100-, 200-, and 300-mg tablets	PO
		Quinidex Extentabs (Robins)	Tablets	Bottles of 100 and 250 tablets	300-mg tablets	PO
		Quinora (Key)	Tablets	Bottles of 100 and 1000 tablets	300-mg tablets	PO
100	Ranitidine hydrochloride	Zantac Syrup (Glaxo)	Syrup	1-pt bottle	15 mg/mL	PO
		Zantac Tablets (Glaxo)	Tablets	Bottles of 30 or 60 tablets	150- or 300-mg tablets	PO
		Zantac Injection (Glaxo)	Solution	2-, 10-, and 40-mL vials	25 mg/mL	IV
101	Rifampin	Rifadin (Marion)	Capsules	Bottles of 30, 60, and 100 capsules	150- and 300-mg capsules	PO
		Rimactane (Ciba)	Capsules	Bottles of 30, 60, and 100 capsules	300-mg capsules	PO

Table continued on following page

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Trade Names and Formulations of Commonly Used Drugs—*Continued*

No.	Drug	Trade Name and Company	Form	Presentation	Concentration	Route
102	Sucralfate	Carafate (Marion)	Tablets	Bottles of 100	1 g tablets	PO
103	Terbutaline sulfate	Brethine (Geigy)	Solution	1-mL ampule	1 mg/mL	IV
		Brethine (Geigy)	Tablets	Bottles of 100 and 1000 tablets	2.5- and 5-mg tablets	PO
104	Topical insecticides—coumaphos	Co-Ral 1% Shaker Can (Cutter)	Powder	2-lb can	10 mg/g powder; shake on as a dusting powder	Topical
		Co-Ral 25% Wettable Powder (Cutter)	Powder	4-lb bag	10 mg/g powder; make up solution of 125 g (1/4 lb) in 45 L (12 gals) water	Topical
105	Topical insecticides-permefhrin	Atroban 11% EC Insecticide (Coopers)	Solution	1-pt container	110 mg/mL; make up solution of 1 pt/25 gals (1 mL/200 mL)	Topical
		Ectiban EC (Durvet or RX)	Solution	8-oz, 1-qt, and 1-gal containers	57 mg/mL; make up as 1 qt/25 gals (10 mL/L)	Topical
		Permectrin 25% WP (Anchor)	Powder	6-oz containers	250 mg/g powder; make up as 1 oz/3 gals water (2.5 g/L)	Topical
		Permectrin Horse and Stable Spray (Anchor)	Solution	8-oz and 1-qt containers	100 mg/mL; make up as 0.5 oz/3 gals (1.3 mL/L)	Topical
106	Topical insecticides—pyrethrins	Derma-Sect Shampoo (Anchor)	Solution, shampoo	8-oz and 1-gal containers	0.6 mg/mL	Topical

107	Triamcinolone acetate (acetonide)	Vetalog Oral (Solvay)	Powder	10 g in 15-g packet	10 g	PO
		Vetalog Parenteral (Solvay)	Suspension	6 mg/mL in 3-, 5-, and 25-mL vials or 2 mg/mL in 25- and 100-niL vials	2 and 6 mg/mL	IM, SC, or IA
108	Trimethoprim-sulfadiazine	Di-Trim 48% Injection (Syntex)	Solution	100-mL bottle	Trimethoprim 80 mg/mL and sulfadiazine 400 mg/mL	IV
		Di-Trim 400 (Syntex)	Paste	37.5-g Dial-A-Dose syringe	Trimethoprim 67 mg/g paste and sulfadiazine 333 mg/g paste	PO
		Tribrissen 48% Injection (Coopers)	Solution	100-mL bottle	Trimethoprim 80 mg/mL and sulfadiazine 400 mg/mL	IV
		Tribrissen 400 Oral Paste (Coopers)	Paste	37.5-g Dial-A-Dose syringe	Trimethoprim 67 mg/g and sulfadiazine 333 mg/g	PO
109	Xylazine hydrochloride	Various manufacturers: Anased-Horses (Lloyd); Rompun (Haver/Diamond); Xylazine HC1 (Butler, Fermenta)	Solution	50-mL bottle	100 mg/mL	IV or IM

Note: It is recommended to check the manufacturer's directions before a drug is used. IA, intra-articular; IM, intramuscular; IV, intravenous; PO, oral or by nasogastric tube; SC, subcutaneous.

Appendix 4

Suggested Immunization Chart for Horses

Age (mo)	Immunization (Primary Series)	Comments
2-3	Strangles	***Strangles***
3-4	Strangles Influenza Tetanus toxoid Encephalomyelitis Rhinopneumonitis Rabies Potomac horse fever	Use in endemic areas or when imminent risk of infection exists. The use of whole cell vaccine may be more effective. *Yearlings* may benefit from two extra doses, 6 months apart. *Brood mares* should receive a biannual dose with one dose given 4-6 weeks before foaling. Other horses may receive a biannual boost if there is a high risk of infection.
4-5	Strangles Influenza Tetanus toxoid Encephalomyelitis Rhinopneumonitis Rabies Potomac horse fever	***Influenza*** Adequate immunity against influenza is difficult because the antigens of influenza virus are continuously changing. Thus, *foals* should receive at least three doses. Foals from vaccinated mares may have an immune response against the vaccine. *Yearlings* and *performance horses* should receive a boost dose every 3 months. *Brood mares* should receive a biannual dose with one dose given 4-6 weeks before foaling.
5-6	Strangles Influenza Rhinopneumonitis	***Tetanus toxoid*** An annual vaccination for all horses is recommended. *Brood mares* should receive the vaccine 4-6 weeks before foaling in case of foaling-induced trauma and also to provide passive immunity to the foal via colostrum. A boost is warranted if wound or surgery occurs after 6 months since the last dose was given.
6-9	Influenza Rhinopneumonitis	***Encephalomyelitis (EEE, WEE, VEE)*** An annual boost before the mosquito season is recommended.
9-12	Influenza Rhinopneumonitis	*Brood mares* should be vaccinated 4-6 weeks before foaling. In year-round endemic areas, vaccinate every 6 months. Foals born in late spring to vaccinated mares are protected through their first summer. VEE: Only needed if there is high risk of an outbreak.
>12	See comments for details Botulism (see comments)	***Rhinopneumonitis (EHV-1, EHV-4)*** *Yearlings* and *performance horses* should be vaccinated every 3 months. *Brood mares* may receive an inactivated EHV 1 vaccine at fifth, seventh, and ninth month of pregnancy. It is also recommended to vaccinate *mares* with EHV 1 and EHV 4 vaccine prebreeding and 4–6 weeks before foaling. Vaccination reduces the number of abortions, but abortions may still occur in vaccinated mares. ***Rabies*** An annual vaccination is recommended in endemic areas. Brood mares should receive vaccine before breeding. ***Potomac Horse Fever*** Only used in endemic areas. Biannual boost is recommended. *Brood mares* should be vaccinated 4–6 weeks before foaling. ***Botulism*** An annual vaccination is recommended only in endemic areas. *Brood mares* should receive the vaccine 1 month before foaling.

EEE, WEE, VEE: Eastern, Western, and Venezuelan Equine Encephalitis, respectively; EHV-1, EHV-4: equine Herpesvirus types 1 and 4, respectively. Manufacturer's recommendations should be considered before administering any vaccine.

Appendix 5

Vaccines and Antitoxins Currently Available

Disease and Vaccines	Manufacturer	Type of Vaccine (Origin)	Dose and Route	Manufacturer Recommendations for Administration and Comments
Encephalomyelitis				
Bivalent: WEE, EEE				
Encephaloid IM	Fort Dodge, Franklin	ITC	1 mL, IM	*2* doses, 3 wk apart, before mosquito season. Boost annually.
Encephalomyelitis vaccine	Colorado Serum Co.	ICC	1 mL, IM	2 doses, 3-4 wk apart, before mosquito season. Boost annually.
Encevac	Bayer	ITC (chicken)	1 mL, IM	2 doses, 3-4 wk apart, before mosquito season. Boost annually.
Trivalent: WEE, EEE, VEE				
Triple-E	Solvay	ITC	1 mL, IM	2 doses, 3-4 wk apart, before mosquito season. Boost annually.
Cephalovac VEW	Boehringer Ingelheim	ITC (chicken: WEE, EEE; porcine: VEE)	2 mL, IM	2 doses, 3-4 wk apart, before mosquito season. Boost annually.
Rhinopneumonitis				
EHV-1				
Rhinomune	Pfizer	Modified equine cell	1 mL, IM	2 doses, 4—8 wk apart, every 3 mo. Pregnant mares from 2 mo of gestation
Pneumabort-K + lb	Fort Dodge, Franklin	Inactivated	2 mL, IM	Start after weaning, 2 doses, 3-4 wk apart. Repeat after 6 mo and boost annually. Pregnant mares should receive a boost during the 5th, 7th, and 9th mo of gestation. If vaccinated after 5 mo of gestation, revaccinate every 2 mo until foaling. Vaccinate open and maiden mares together with pregnant mares.

Table continued on following page

Vaccines and Antitoxins Currently Available—Continued

Disease and Vaccines	Manufacturer	Type of Vaccine (Origin)	Dose and Route	Manufacturer Recommendations for Administration and Comments
Prodigy	Bayer	ITC	2mL, IM	Start after 6 mo, 3 doses, 4-6 wk apart. Pregnant mares should receive a boost during the 5th, 7th, and 9th mo of gestation. Maiden and barren mares in contact with pregnant mares also should be vaccinated. For respiratory disease: vaccinate annually or when exposure is imminent.
EHV-1 and EHV-2				
Prestige	Bayer	ITC	2 mL, IM	2 doses, 4-6 wk apart. Give boost to foals 6 mo after the 1st dose. Revaccinate annually or when exposure is imminent. Approved for use in pregnant mares, but no claim that prevents abortion.
EHV4,1	Fort Dodge	Inactivated	1 mL, IM	2 doses, 4-6 wk apart. Young horses should receive a 3rd dose, 4 wk after the 2nd dose. Revaccinate annually or when exposure is imminent.
Influenza				
A-equine 1 and 2				
Equicine II	Bayer	ICC (canine)	1 mL, IM	*2* doses, 3-4 wk apart. Boost annually or before likely exposure.
Equi-Flu	Boehringer mgelheim	ICC	1 mL, IM	2 doses, 3-4 wk apart. Boost annually.
Flumune	Pfizer	ICC	1 mL, IM	2 doses, 2-4 wk apart. Boost annually.
Fluvac	Fort Dodge, Franklin	ICC (canine kidney)	1 mL, IM	2 doses, 2-4 wk apart. Boost annually or before likely exposure.
Inflogen3	Solvay	ICC	1 mL, IM	2 doses, 2-4 wk apart. Boost annually or before likely exposure.
Inflogen	Solvey	Inactivated, allantoic fluid (chicken embryo)	1 mL, IM	2 doses, 2-4 wk apart. Boost annually or before likely exposure.
Potomac horse fever				
PHF-VAX	Schering-Plough	Inactivated	1 mL, IM	Start after 3 mo, 2 doses, 3—4 wk apart. Boost every 6 mo in endemic areas. An annual boost with doses before period of peak challenge may improve immunity.
RM Potomavac	Rhone Merieux	Inactivated	1 mL, IM	Start after 3 mo, 2 doses, 3-4 wk apart. Boost annually.
PotomacGuard	Fort Dodge, Franklin	Inactivated	1 mL, IM	2 doses, 3-4 wk apart. Boost annually.
Mystique	Bayer	Inactivated	1 mL, IM	Start after 3 mo, 2 doses, 3-4 wk apart. Boost annually or before likely exposure.

Rabies				
RM Imrab 3	Rhone Merieux	ICC	2mL, IM	Start after 3 mo, 1 dose. Boost annually.
RM Imrab Bovine Plus	Rhone Merieux	ICC	2mL, IM	Start after 3 mo, 1 dose. Boost annually.
Rabvac 3	Solvay	ICC	2mL, IM	Start after 3 mo, 1 dose. Boost annually.
Rabguard-TC	Pfizer	ICC (porcine)	1 mL, IM	Start after 3 mo, 1 dose. Boost annually.
Strangles				
Equibac II	Fort Dodge	Inactivated whole bacteria	2mL, IM	Start after 3 mo, 2 doses, 2-4 wk apart. Boost annually. More than 1 dose per year may increase the risk of anaphylaxis or local reaction. It is contraindicated during incubation period or infection.
Strepguard	Bayer	Inactivated bacterial M-protein extract	1 mL, IM	Start after 3 mo, 2 doses, 3-4 wk apart. Foals vaccinated before 3 mo of age should have a boost at 6 mo or at weaning. Boost annually or when exposure is imminent.
Strep Vax II	Boehringer Ingelheim	Inactivated bacterial M-protein extract	1 mL, IM	Start after 3 mo, 3 doses, 3 wk apart. Foals vaccinated before 3 mo of age should receive a boost at 6 mo or at weaning. Boost annually or before anticipated exposure.
Tetanus				
Toxoid				
Super-Tet	Bayer	Inactivated	1 mL, IM	2 doses, 3-4 wk apart. Boost annually.
Tetanus toxoid	Fort Dodge, Franklin	Inactivated	1 mL, IM	2 doses, 4-8 wk apart. Boost annually. For small horses, use 0.5 mL.
Tetanus toxoid	Colorado Serum Co.	Inactivated	1 mL, IM, SC	2 doses, 4 wk apart. Boost annually.
Tetnogen	Solvay	Inactivated	1 mL, IM, SC	2 doses, 4 wk apart. Boost annually.
Tetguard	Boehringer Ingelheim	Inactivated	1 mL, IM	
Tetmune	Pfizer	Inactivated	1 mL, IM	2 doses, 3-4 wk apart. Boost annually.
Antitoxin				
Tetanus antitoxin	Colorado Serum Co., Sanofi, Bayer		1500 U, IM, SC	Tetanus antitoxin should be administered to unimmunized animals or those with unknown vaccination history that have received sustained injury or undergone surgery. Also to foals born to unvaccinated mares. An antitoxin and toxoid can be administered to these horses, in two separate sites, to provide passive and active immunity. A 2nd dose of toxoid can be administered 4 wk later to secure long-lasting immunity.
	Fort Dodge		1500 U, IM, SC, IV, IP	
Tetnogen-AT	Solvay		1500 U, IM, SC	

Table continued on following page

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Vaccines and Antitoxins Currently Available—Continued

Disease and Vaccines	Manufacturer	Type of Vaccine (Origin)	Dose and Route	Manufacturer Recommendations for Administration and Comments
Viral arteritis				
Arvac	Fort Dodge	Modified cell (equine)	1 mL, IM	Start after 6 wk, 1 dose. Boost annually. Stallions and mares should be vaccinated at least 3 wk before breeding season. Avoid vaccination during the last 2 mo of gestation.
Combined vaccines				
Encephalomyelitis and Tetanus				
EEE, WEE, VEE+ toxoid		All inactivated + toxoid		
Cephalovac VEWT	Boehringer Ingelheim		2mL, IM	Start after 3 mo, 2 doses, 4-8 wk apart. Boost annually.
EEE, WEE + toxoid		All inactivated + toxoid		
Cephalovac EWT	Boehringer Ingelheim		1 mL, IM	2 doses, 4-6 wk apart. Boost annually.
Double-ET	Solvay		1 mL, IM	2 doses, *2-4* wk apart. Boost annually.
Encevac-T	Bayer		1 mL, IM	Start after 3 mo, 2 doses, 3-4 wk apart. Boost annually.
Equiloid	Fort Dodge, Franklin		1 mL, IM	2 doses, 4-8 wk apart. Boost annually.
Triple-ET	Solvay		1 mL, IM	2 doses, 2-4 wk apart. Boost annually.
EWT	Schering-Plough		1 mL, IM	2 doses, 2-4 wk apart. Boost annually.
Tetmune EW	Pfizer		1 mL, IM	2 doses, 3-4 wk apart. Boost annually.
Encephalomyelitis vaccine-tetanus toxoid	Colorado Serum Co.		2 mL, IM	2 doses, 3-4 wk apart. Boost annually.
Encephalomyelitis, Influenza, and Tetanus				
EEE, WEE, influenza Al and A2 + toxoid		All inactivated + toxoid		
Double-EFT	Solvay		1 mL, IM	2 doses, 3-4 wk apart. Boost annually.
Encevac TC-4	Bayer		1 mL, IM	Start after 3 mo with 2 doses, 3-4 wk apart. Boost annually.
Equi-Flu EWT	Boehringer Ingelheim		2 mL, IM	2 doses, 4-6 wk apart. Boost annually.
Fluvac EWT	Fort Dodge, Franklin		1 mL, IM	2 doses, 4-8 wk apart. Boost annually.

Flumune EWT	Pfizer		1 mL, IM	Start after 3 mo with 2 doses, 3 wk apart. Boost annually.
EWTF	Schering-Plough		2mL, IM	2 doses, 2-4 wk apart. Boost annually.
RM Equine EWTF	Rhone Merieux		1 mL, IM	Start after 3 mo with 2 doses, 2-4 wk apart. Boost annually.
EEE, WEE, influenza Al and A2 + toxoid		All inactivated + toxoid		
Triple-EFT	Solvay		1 mL, IM	2 doses, 2-4 wk apart. Boost annually.
Equi-Flu VEWT	Boehringer Ingelheim		2mL, IM	Start after 3 mo with 2 doses, 4-8 wk apart. Boost annually.
Influenza and Herpes		All inactivated		
Fluvac EHV-4/1	Fort Dodge, Franklin		1 mL, IM	Start after 3 mo, 2 doses, 4-6 wk apart. Young horses should receive a 3rd dose 4-6 wk after the 2nd dose. Boost annually or when there is likely exposure.
Rhino-Flu	Pfizer		1 mL, IM	Start after 3 mo, 2 doses, 3 wk apart. Young horses should receive a 3rd dose 4-6 wk after the 2nd dose. Boost annually or when there is likely exposure.
Prestige II	Bayer		1 mL, IM	Start after 3 mo, 2 doses, 4-6 wk apart. Foals should receive a 3rd dose 6 mo after the 2nd dose. Boost annually or when there is likely exposure. No claim for prevention of EHV-1 abortion.
Influenza and Tetanus		Al and A2 inactivated + toxoid		
Inflogen-T	Solvay		1 mL, IM	2 doses, 2-4 wk apart. Boost annually
Influenza, Herpes, and Tetanus		Al and A2 inactivated; EHV-1 modified & toxoid		
Rhino-Flu T	Pfizer		2mL, IM	Start after 3 mo with 2 doses, 3 wk apart. Boost annually.
Influenza, Herpes, Tetanus and Encephalomyelitis		Al and A2 inactivated, EH-1 modified; EEE and WEE inactivated & toxoid		
Rhino-Flu EWT	Pfizer		2mL, IM	Start after 3 mo with 2 doses, 3 wk apart. Boost annually.

ITC, inactivated tissue culture; ICC, inactivated cell culture; EEE, WEE, VEE, Eastern, Western, and Venezuelan equine encephalitis, respectively; EHV-1, EHV-4, equine herpesvirus types 1 and 4, respectively.

Appendix 6

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Boehringer Ingelheim Animal Health Inc.
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Fermenta Animal Health Company
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Marion Merell Dow Inc.
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Mead Johnson Laboratories
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Parke-Davis
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Pfizer Inc.
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Pharmaderm
A Division of Atlanta Inc.
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Phoenix Pharmaceutical Inc.
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Searle & Company
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SmithKline Beecham Animal Health
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I N D E X

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