Illustrated Guide to Equine Diseases

1 Diseases of the Gastrointestinal Tract and Liver

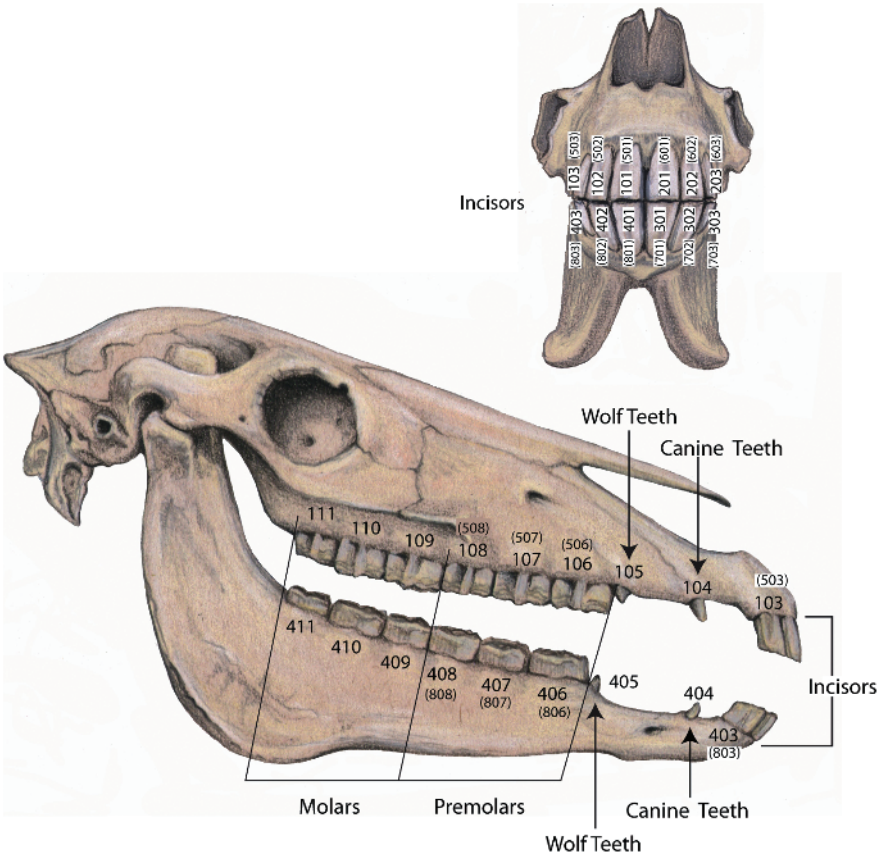


Figure 1.1 Illustration for the Triadan numbering system for equine dentition. The permanent dentition is described by 1–400s while the deciduous dentition is described by the 5–800s.

Wave Malocclusion

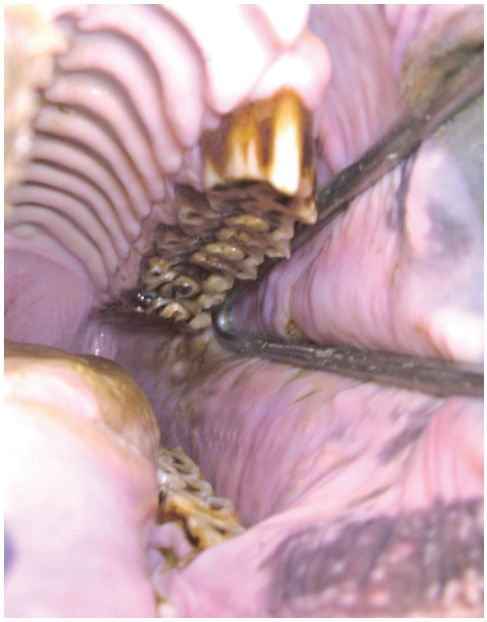


Figure 1.2a Wave malocclusion involving the 200 and 300 arcades in a middle-aged patient. The 206 is overlong. The 207 and 208 exhibit progressively shorter clinical crown to the 208/9 junction. Note that the gingival margin is displaced dorsally at this point and dips ventrally again at the 209/10 junction where the 210 is overlong. This involvement of the gingival margin is an indicator of chronicity and signals probable bony remodeling. Patient age and amount of clinical crown and gingival margin/bony changes collectively determine the amount of correction possible at a single session. Some wave malocclusions cannot be normalized but are best maintained to minimize progression and deterioration.

Figure 1.2b Same patient in fig. 1.2a. View of 300 arcade "wave" abnormality. Note that the distal 308 is the tallest point in the 300 arcade and the 309 is not visible at all.

Rostral Hook

Figure 1.3 Mesial portion of 206 is overlong due to malocclusion with 306. Commonly referred to as a "rostral hook," this abnormality is often seen in class 2 malocclusions commonly known as "parrot mouth." Early recognition and reduction of the excessive crown is recommended to avoid large or staged reductions.

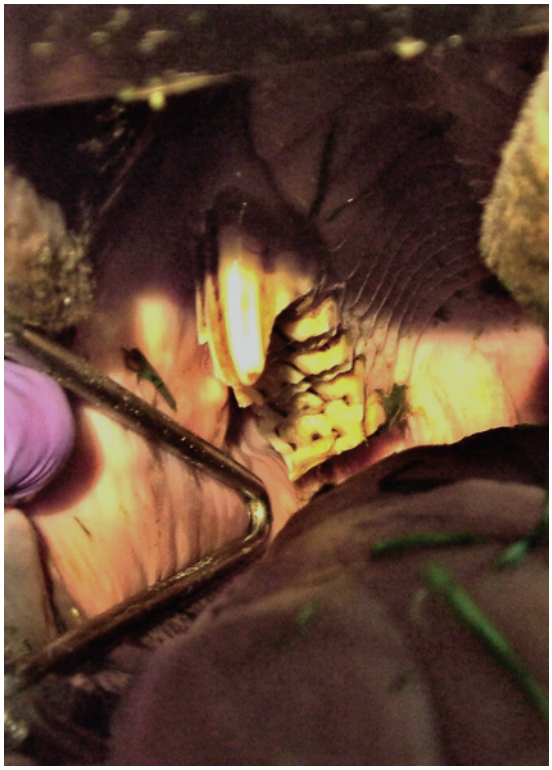


Figure 1.4 Large amount of excessive crown at the mesial portion of 106 in a 10-year-old quarter horse stallion. This abnormality is common in class 2 malocclusions (parrot mouth) although this patient has normal incisor occlusion. Commonly referred to as a "rostral hook," it is progressive, can traumatize soft tissue, may exacerbate malocclusions elsewhere in the mouth, and may interfere with normal masticatory function. Overlong crown of this magnitude requires staged reductions to avoid pulpar exposure or thermal injury.

Caudal Hooks or Ramps

Figure 1.5 Overlong crown at distal 311 due to malocclusion with 211. Commonly known as "caudal hooks or ramps," these abnormalities are progressive, can injure soft tissue, predispose to other malocclusions and periodontal disease, and may interfere with normal masticatory motion. Commonly, though not exclusively, seen in class 2 malocclusions (parrot mouth).

Stepped Tooth

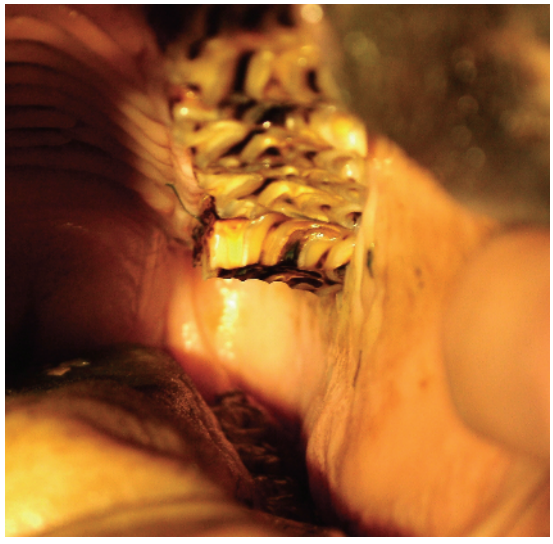


Figure 1.6 Overlong 209 due to missing 309. Commonly referred to as a "stepped tooth." Regular crown reductions may be necessary to maintain normal rostral/caudal mandibular movement.

Step Mouth

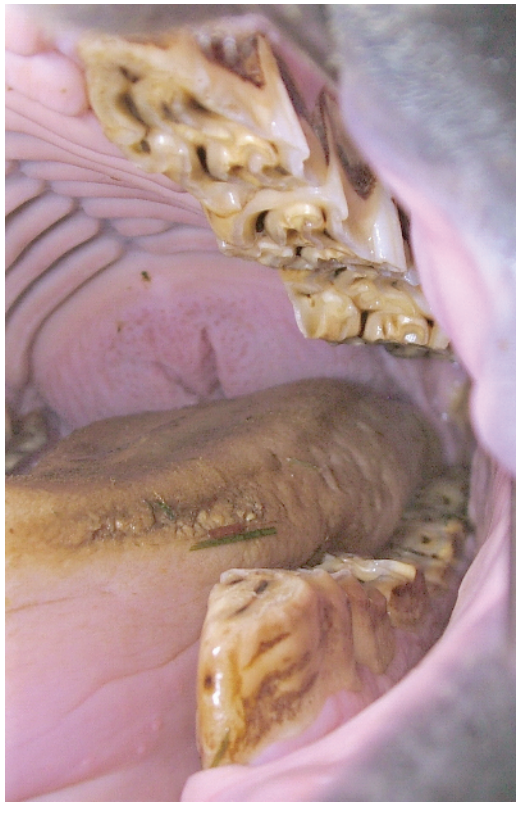


Figure 1.7 Abrupt, severe changes in crown height along an entire arcade pair is commonly known as a "step mouth." Normal mastication is significantly compromised with such malocclusions. Severe cases require serial crown reductions for safe correction.

Hooks or Ramps

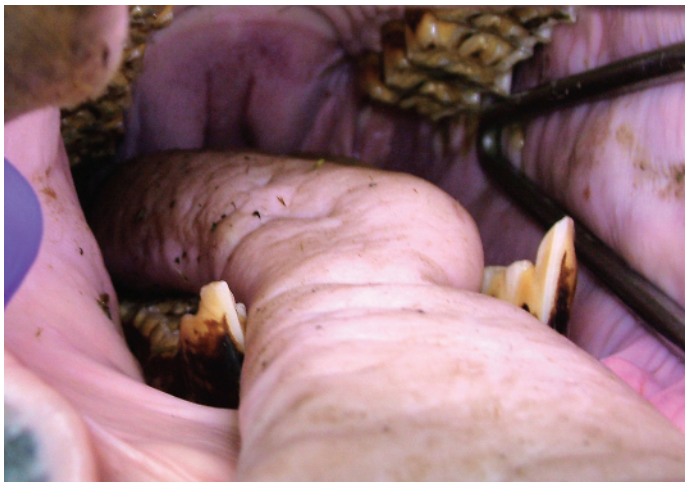


Figure 1.8 The 306 and 406 with excessive mesial crown commonly referred to as hooks or ramps. They are caused by malocclusion with the maxillary 6s. They are progressive and can cause soft tissue injury, biting pain, periodontal disease, and abnormal mastication.

Shear Mouth

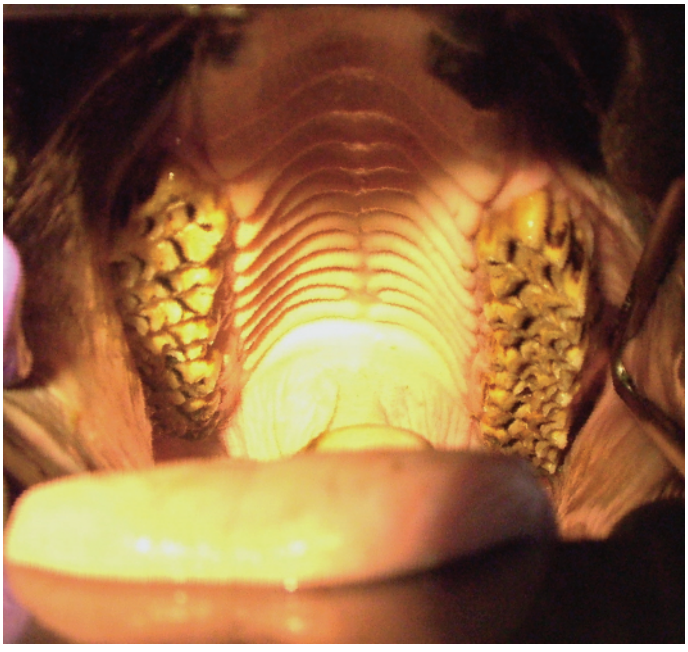


Figure 1.9 "Shear mouth" in an 8-year-old quarter horse mare. Note the slightly steeper table angle of the 200 arcade in comparison to the 100 arcade. Gradual reduction of the steep table angle can be helpful. If left unchecked, many cases will worsen to the point of abnormal mastication.

Overlong Distal Portion of the Third Incisor

1

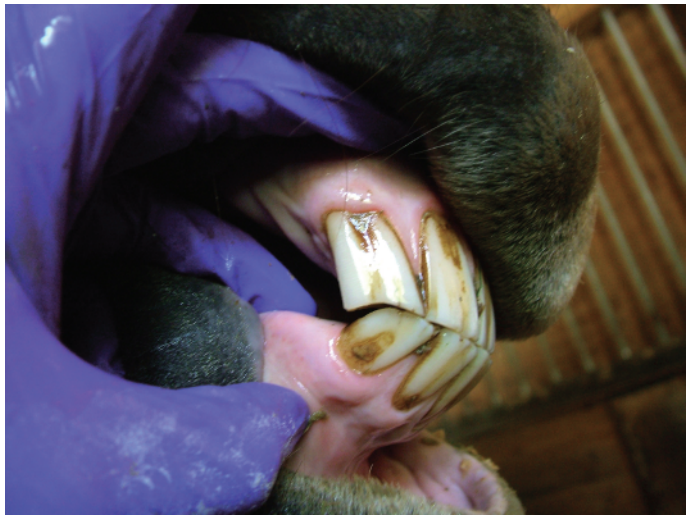


Figure 1.10a Distal portion of 103 is overlong caused by a malocclusion with 403. Such areas of excessive crown are progressive and can cause interference with normal lateral excursion of the mandible and thereby affect functional occlusion of the cheek teeth.

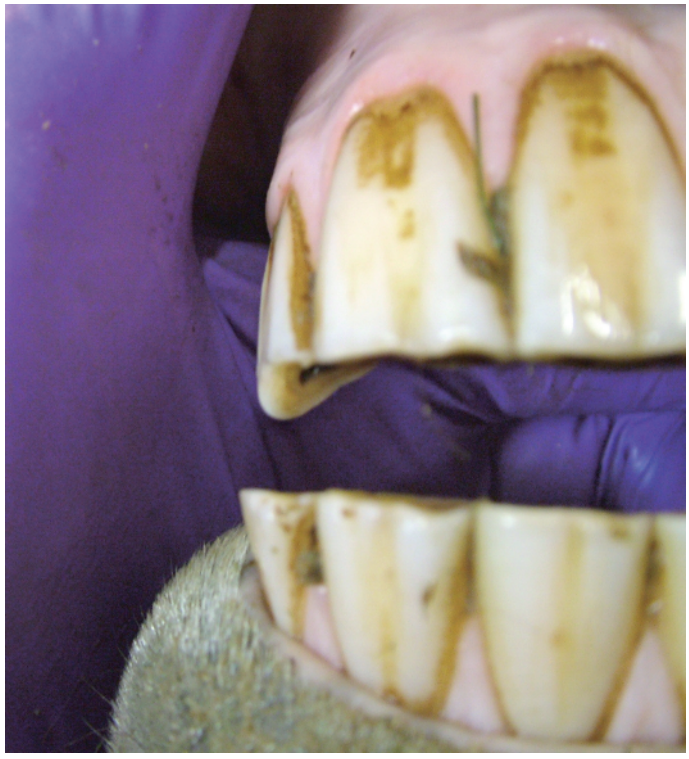


Figure 1.10b Rostral view of overlong distal 103. Same horse in fig. 1.10a

Diagonal Incisor Malocclusion

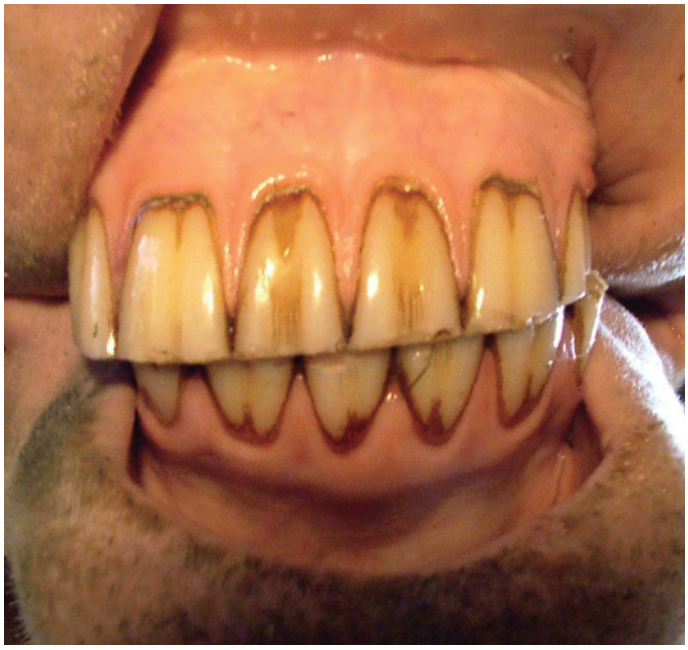


Figure 1.11 Diagonal incisor malocclusion (DGL3) in an aged horse. Note the progressively increasing length of clinical crown from the 203 right to the 103 and from the 403 left to the 303. There is also a mandibular offset to the horse's left. Etiology can be difficult to determine and may be multifactorial. This malocclusion is progressive and early detection and correction are beneficial. Correction in some cases can be harmful. A thorough understanding of equine mastication biomechanics is critical for successful correction and maintenance.

Incisor Curvature

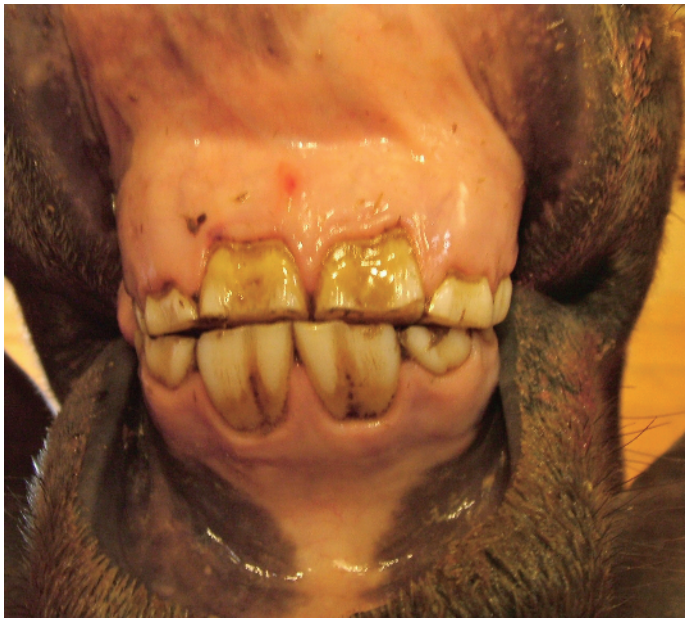


Figure 1.12 Dorsal incisor curvature in a juvenile. Etiology may be asynchronous eruption of the 1s or a cribbing/rubbing habit that is causing selective wear to 101 and 102.

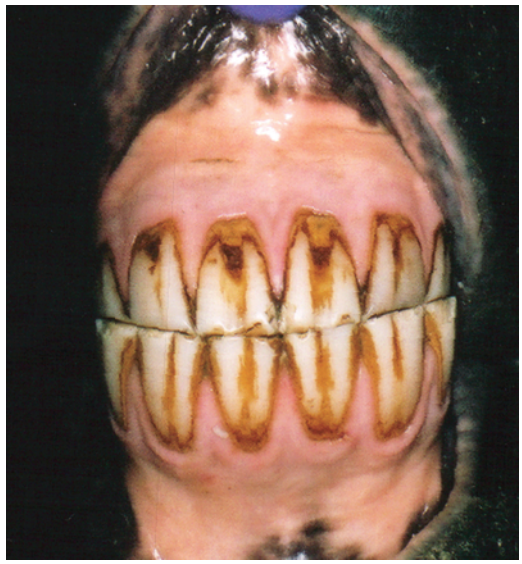


Figure 1.13 Ventral incisor curvature in an aged horse. This malocclusion is usually progressive and can cause abnormal lateral excursion. Overlong incisors should be reduced as necessary to maintain normal lateral excursion and to prevent progression of the malocclusion.

Irregular Incisor Malocclusion

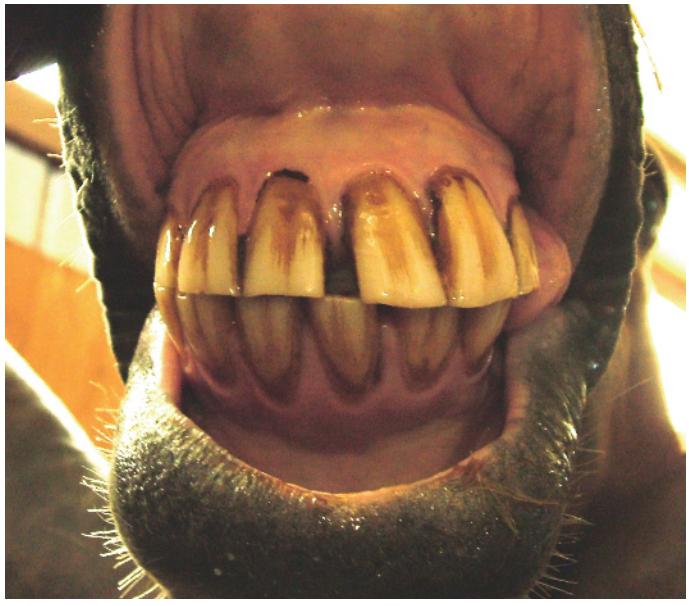


Figure 1.14 Irregular incisor malocclusion in which the occlusal plane undulates in a wave pattern. In this case, probably initiated by the abnormal positions of 101/201. As with any incisor malocclusion, lateral excursion and therefore efficient mastication may be affected.

Supernumerary Incisor

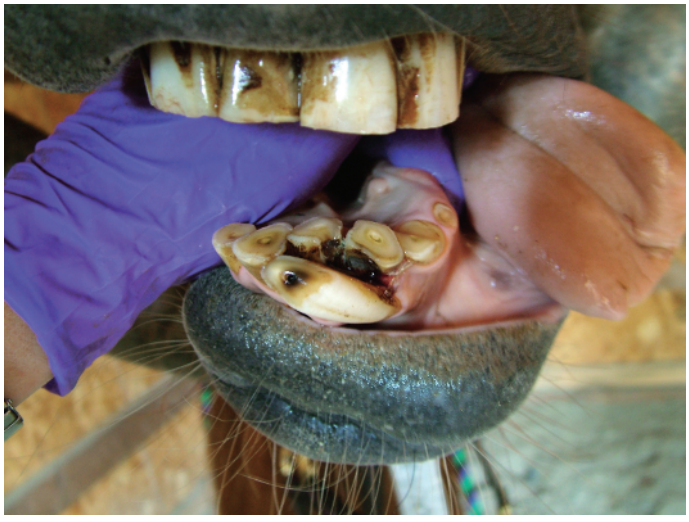


Figure 1.15 A 14-year-old Peruvian Paso mare with supernumerary and displaced incisors. Feed was collecting between the grossly displaced incisor and the ones lingual to it causing periodontal disease and dental decay. Extraction of the displaced incisor and reduction of other overlong incisor crowns to restore normal lateral excursion was beneficial.

Overbite (Parrot Mouth)

Figure 1.16 A 16-year-old Warmblood/Thoroughbred mare with a class 2 malocclusion (parrot mouth). Minimal occlusal contact occurs at the 3s.

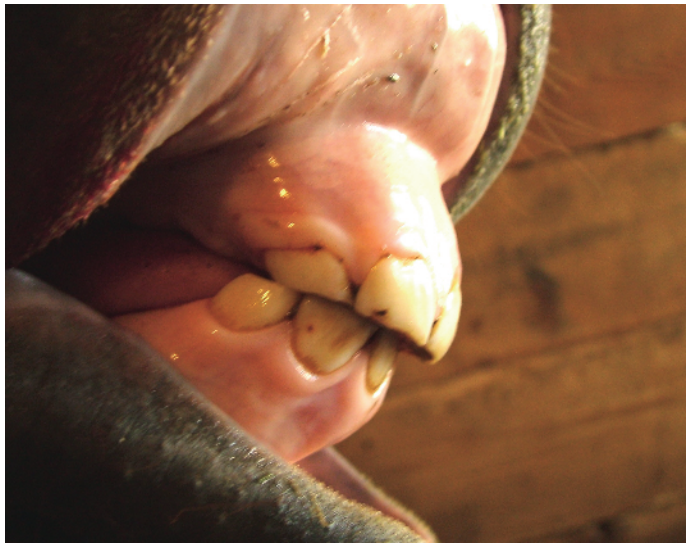


Figure 1.17a Class 2 malocclusion in a yearling. Commonly referred to as “parrot mouth” or “overbite.” Early detection and removal of resultant overlong clinical crowns can be curative in mild to moderate cases. More severe cases may require orthodontic treatment.

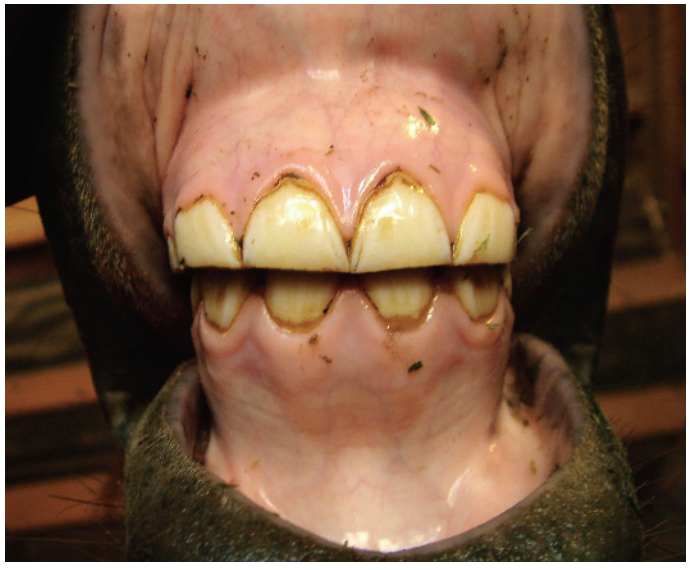


Figure 1.17b Same horse in 1.17a, rostral view of class 2 malocclusion.

Underbite (Sow or Monkey Mouth)

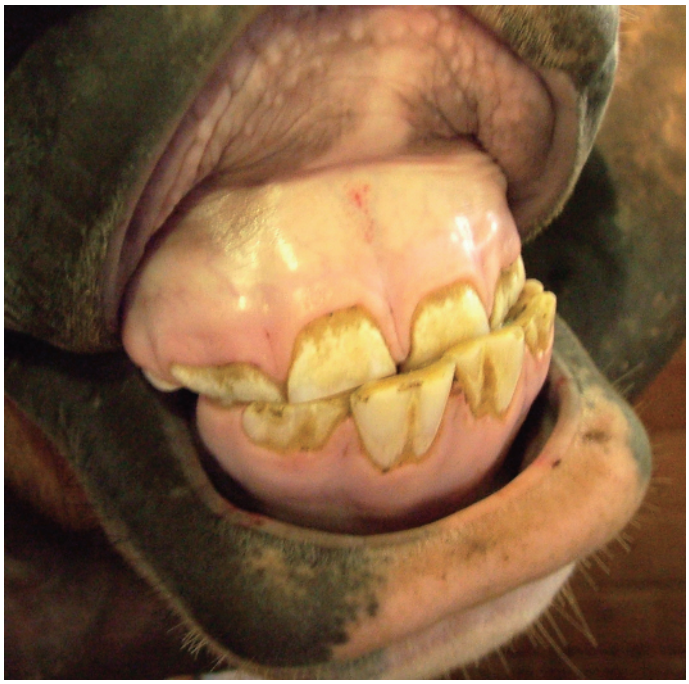


Figure 1.18 Class 3 malocclusion (sow mouth or monkey mouth) in a 2 1/2-year-old. Early recognition and treatment to release the promaxilla from behind the mandible may allow for normal growth and resolution. Advanced cases may not be correctable but benefit greatly from regular reduction of overlong crowns and restoration of normal mastication biomechanics.

Periodontal Disease, Diastema, and Enamel and Cemental Decay

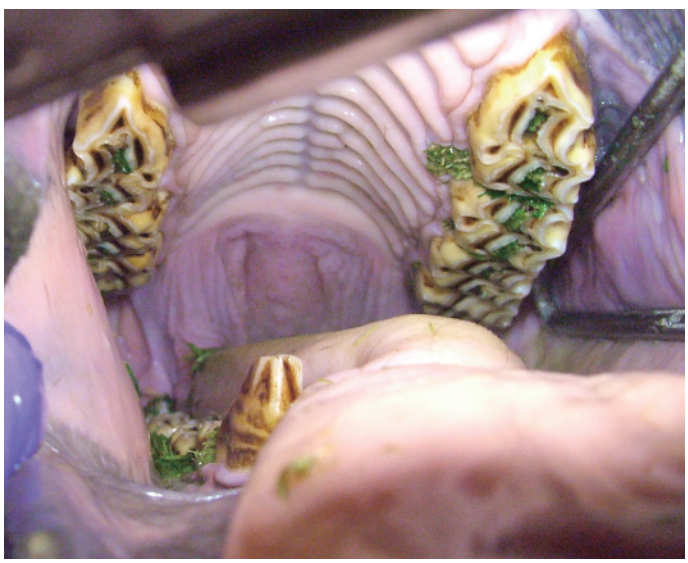


Figure 1.19a A 2-year-old Thoroughbred with feed packed between 506 and 507 and between 806, 807, and 808. This presentation is a strong indicator of periodontal disease and should prompt further examination. The 806 is also overlong due to a missing opposing tooth in the upper right arcade. The overlong crown may be contributing to the feed packing distal to it due to abnormal occlusal forces.

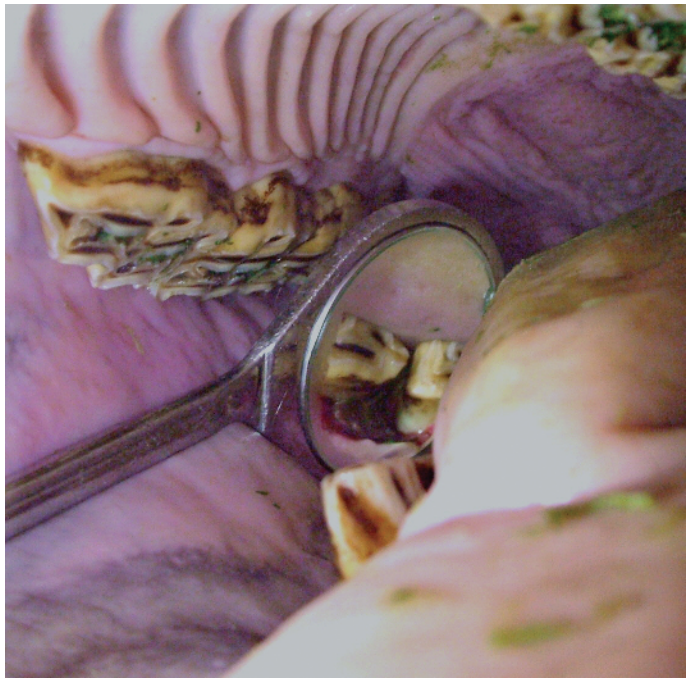


Figure 1.19b Same horse in fig. 1.19a; large periodontal pocket after cleaning trapped feed from interproximal space. Pockets of this size likely indicate bony involvement and radiography is warranted to assess the severity of the disease.

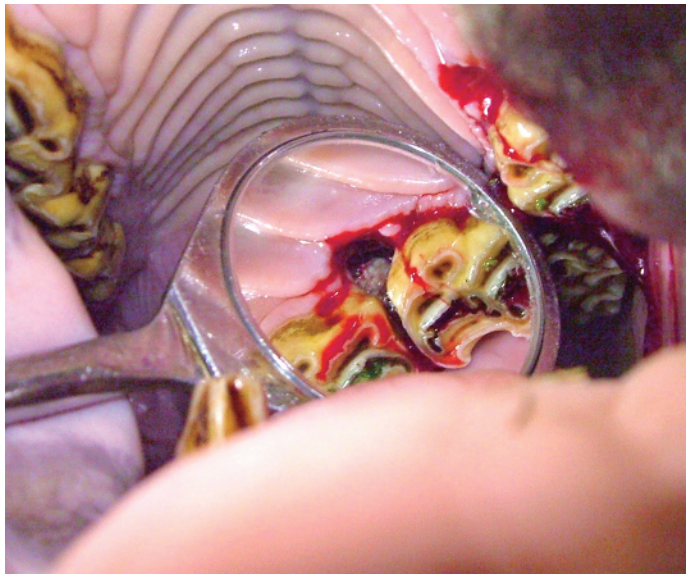


Figure 1.19c Same horse in fig. 1.19a; diastema and periodontal pocket between 506 and 507 after feed material was cleaned out. The grey tissue deep within the pocket is actually a free-floating "foreign body." Histological examination revealed that it was bone.

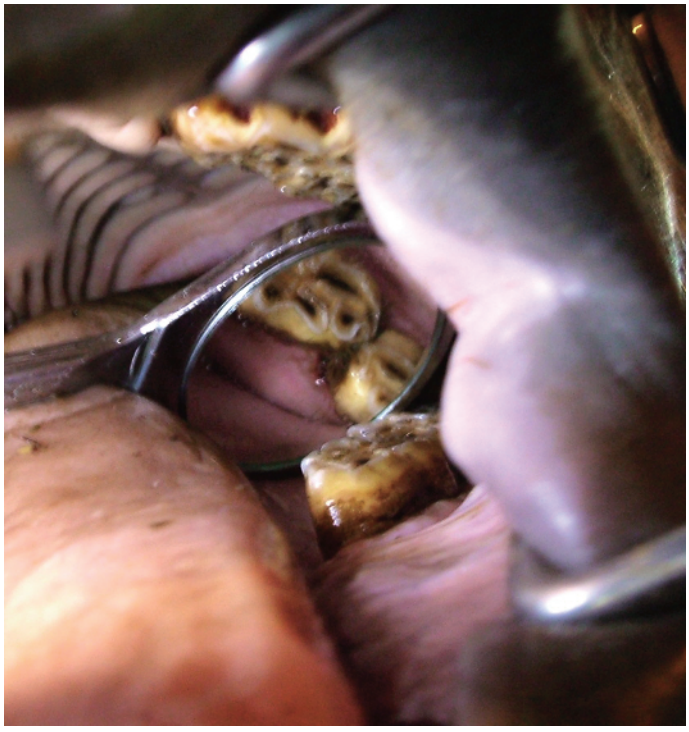


Figure 1.20 A 5-year-old paint mare with lingually displaced 308. The mirror is placed between 307 and 308. Feed is trapped at the lingual aspect of that interproximal space causing periodontal disease.

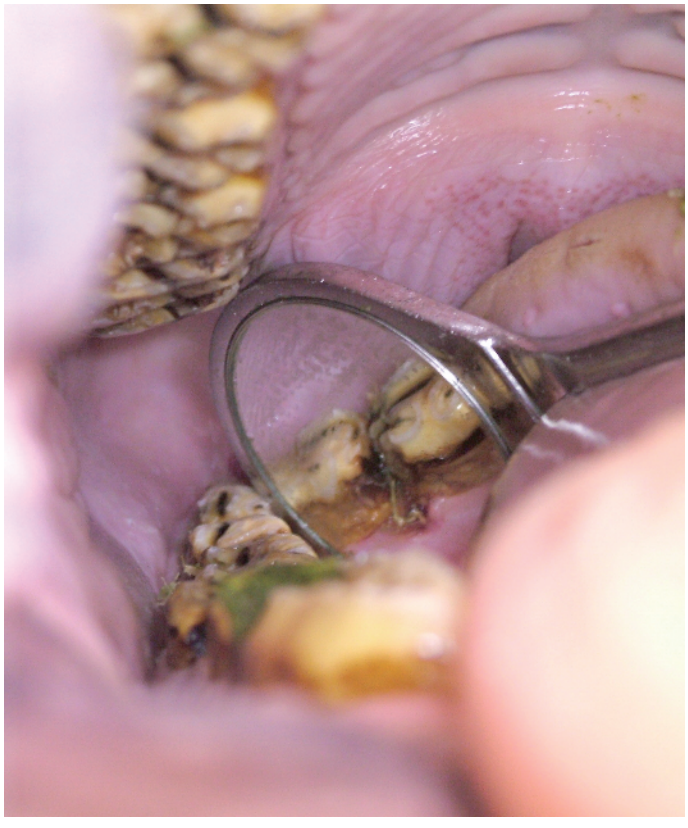


Figure 1.21 A 9-year-old Warmblood mare with lingual periodontal pocket at 410/11. Packed feed is visible in both views: the lingual (mirror) and the buccal aspect view. An overlong distal 411 has already been reduced. Excessive crown at the distal 311 or 411 may predispose to interproximal small diastema formation due to abnormal occlusal forces. Correction of malocclusions is sometimes curative. In other cases, primary treatment of the periodontal disease is also necessary.

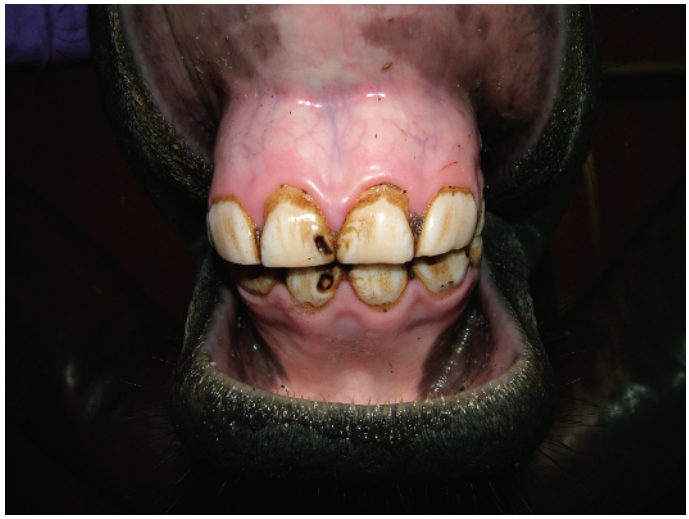


Figure 1.22a Focal areas of enamel decay at 501 and 801. The focal nature of the lesion involving deciduous teeth necessitates no treatment.

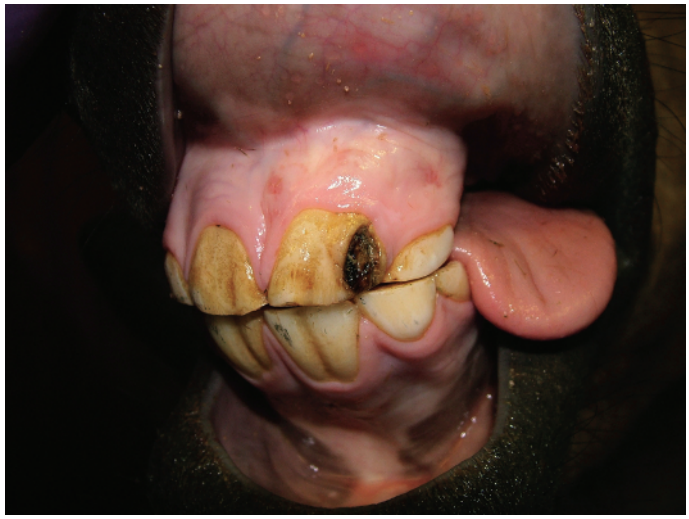


Figure 1.22b Large area of enamel decay involving a permanent incisor. This incisor quadrant is also oligodontic. Radiographic examination is warranted to fully explore the dental pathology. Debridement and/or endodontic or restorative procedures may be indicated pending deep structure evaluation via radiography.

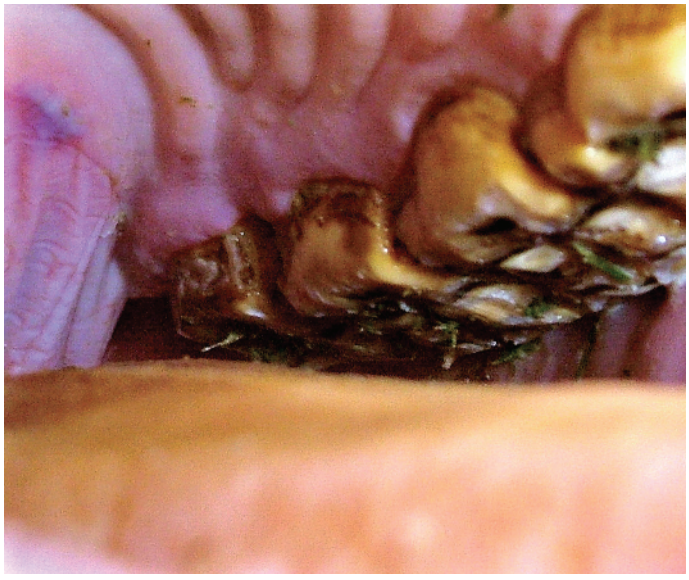


Figure 1.23 Peripheral cemental decay at the palatal aspect of 210 and 211. Note the normal yellow cementum on the palatal surfaces of 208 and 209. At 210 see darker staining roughened edge of a cemental "crescent" and a grey coating of "plaque" at the gingival margin marking early stage decay. At 211 see the underlying white enamel "skeleton," denuded of its cemental covering. Feed stasis is a common cause of this condition. Underlying causes for feed stasis should be identified and corrected.

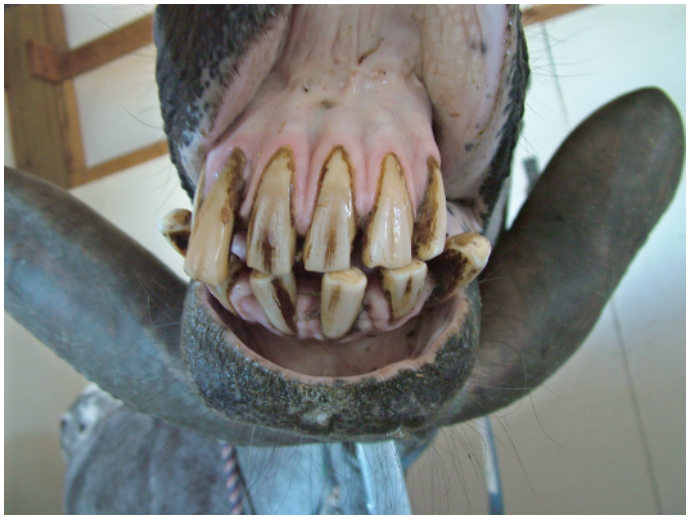


Figure 1.24a A 29-year-old Appaloosa gelding presented with dysphagia and acutely decreased water intake and loss of body condition. Diagnosis was chronic incisor periodontal disease with cemental hypoplasia. Radiographs showed predominantly cemental hypoplasia. The 102, 303, and 403 were grade 3 loose and were extracted. Water intake and feed consumption immediately returned to normal.

Figure 1.24b A 29-year-old Warmblood with chronic incisor periodontal disease. This disease is characterized by a concurrent cemental hyperplasia. This case displays predominantly cemental hyperplasia. None of the incisors are loose. Regular examination and periodic radiographs are recommended to monitor progress.

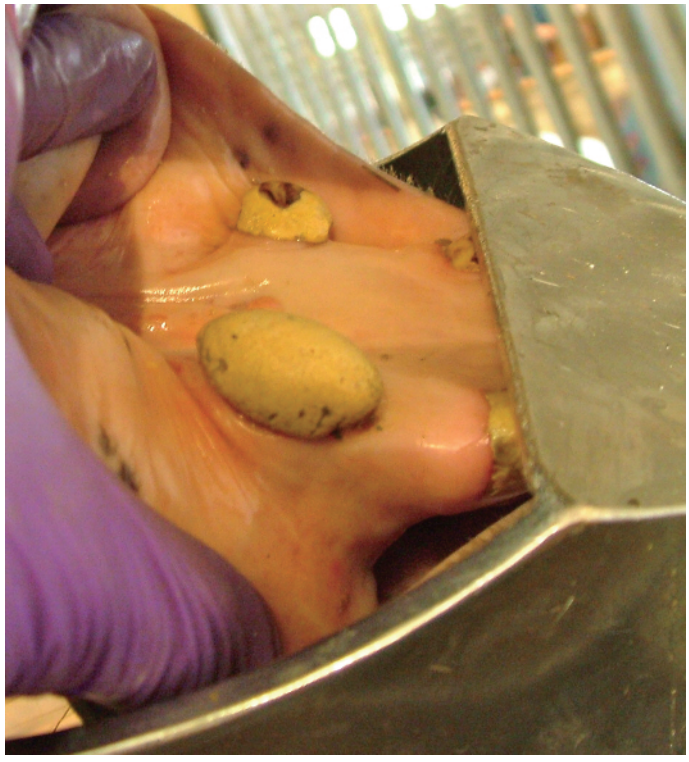


Figure 1.25 Severe calculus accumulation over and around 404. The 304 is also affected although to a lesser extent. Canine calculus can be a symptom of more severe periodontal disease. Careful examination of the affected tooth and its periodontal tissues is warranted in all cases of calculus formation.

Figure 1.26a Geriatric wear in an older horse, approximately 27 years old. Note the loss of transverse ridges on occlusal surfaces. Much of the enamel is worn away leaving smooth dentin and cementum. Commonly referred to as "cupped," these occlusal surfaces have significantly reduced grinding ability. Dietary management may be necessary to meet this older horse's nutritional needs.

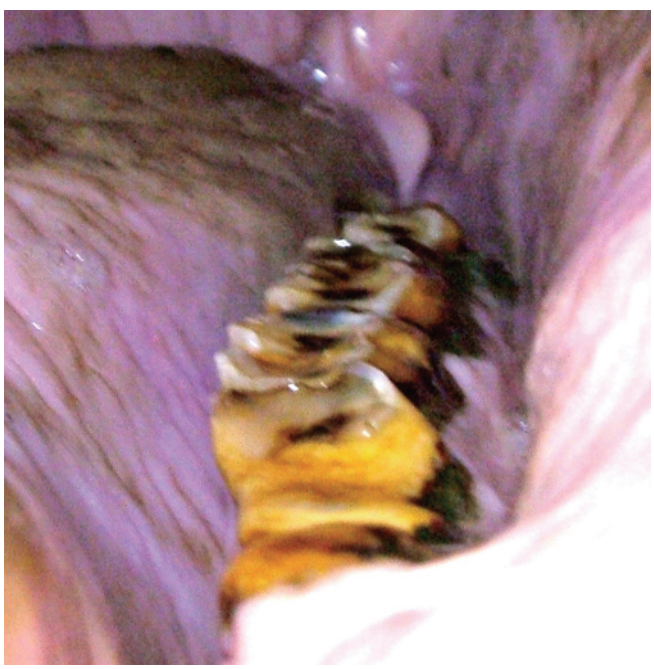


Figure 1.26b Geriatric wear involving mandibular cheek teeth in same horse in 1.26a. Note extreme wear at 306 and mesial 307. These teeth are often referred to as "smooth."

Teeth Eruption and Retained Deciduous Teeth "Cap"

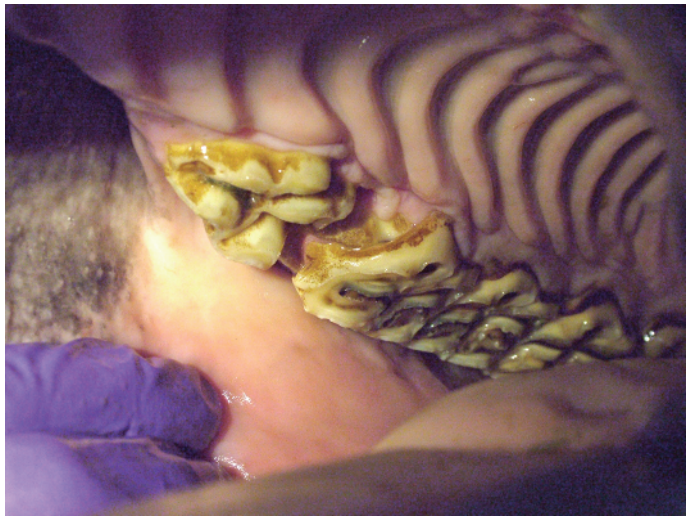


Figure 1.27 Typical appearance of erupting teeth in a 3-year-old patient. The 106 is erupted and very recently in wear. Tooth 107 is visible beneath its deciduous predecessor (507). The 507 in this stage is commonly referred to as a "cap." Deciduous teeth normally exfoliate spontaneously but if encountered during dental examination, it is safer to remove them if they are loose and the permanent tooth is visible beneath them. Premature removal of deciduous caps may result in damage to the permanent tooth.

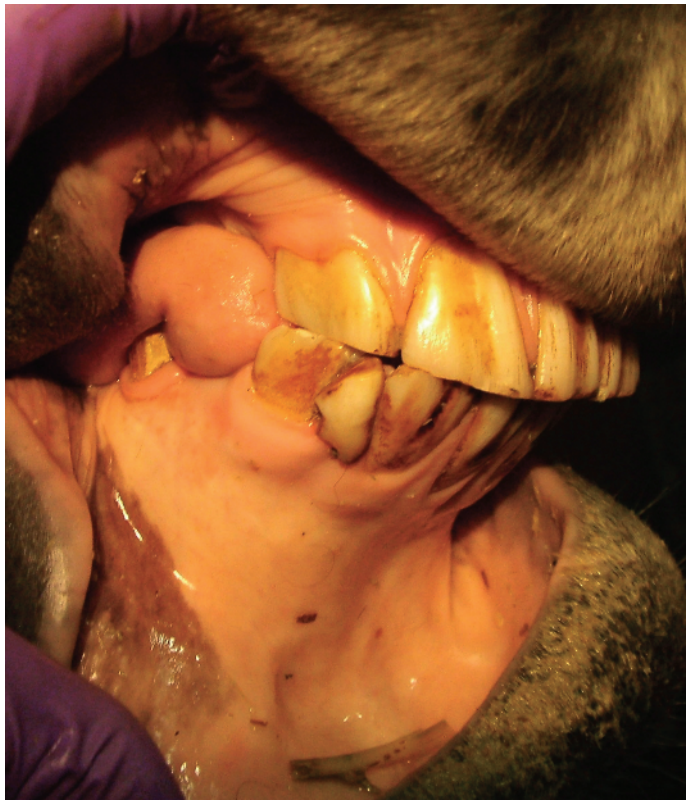


Figure 1.28 Retained 803 in a 5 1/2-year-old. Extraction is necessary to allow proper positioning of 403 and to avoid feed accumulation between teeth. Presence of even small root fragments from deciduous teeth can inhibit proper positioning of permanent teeth.

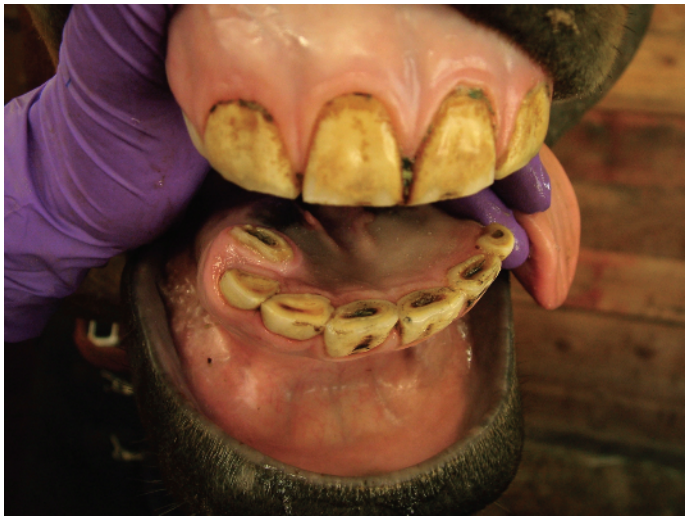


Figure 1.29a Retained tooth 802 in a 4-year-old Thoroughbred gelding. Note that tooth 302 is in normal position and in wear. Tooth 703 is still present. Tooth 803 is still present, and normally located. The right mandibular intermediate incisor is deciduous (802) and there is a permanent incisor erupted out of position distal to the rest of the arcade. Tooth 802 should be extracted.

Figure 1.29b Same horse in fig. 1.29a. Retained tooth 802 was extracted. Note no evidence of radicular resorption.

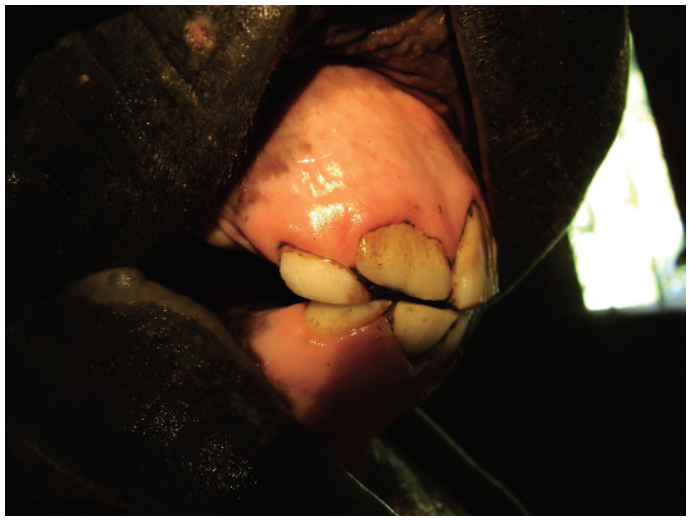


Figure 1.30 Crowding of 102 in a 3 year old. The 503 is preventing the 102 from full eruption into its normal position. Early detection and treatment may prevent permanent incisor malocclusions or periodontal disease.

Wolf Teeth

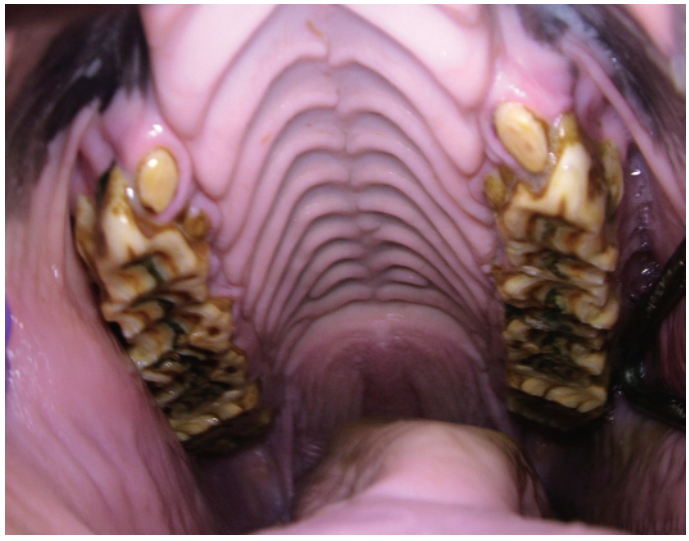


Figure 1.31 Very large wolf teeth in a 2-year-old Thoroughbred. Note that the mesial and buccal surfaces of the 506 and 606 have been previously rounded into a “bit seat” and wolf teeth have been reduced but not extracted. Large wolf tooth crown does not necessarily indicate a large root or a difficult extraction. Current recommendations are that wolf teeth be extracted before training to the bit. This is done to avoid “bit” discomfort. Excessive transverse ridges at the 109 and 209 are also present.

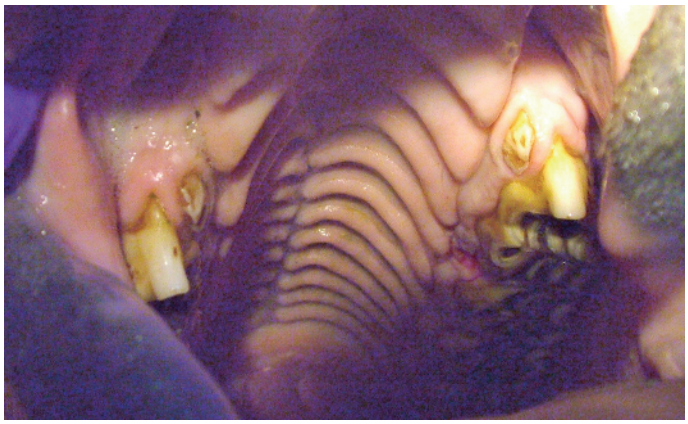


Figure 1.32 Atypical palatal location of wolf teeth in a yearling. Care should be taken when extracted to avoid the palatine artery.

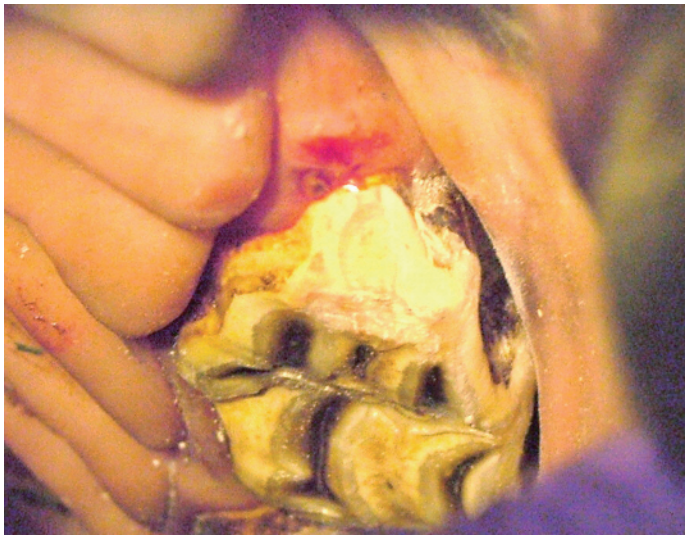


Figure 1.33 Iatrogenic soft tissue injury to the gingiva making a small wolf tooth fragment visible just palatal to 206. Wolf tooth fragments can cause biting discomfort and should be removed when identified. They can result from fracture at the time of initial extraction or may be rudimentary or polydontic and not visible at earlier examinations.

Deviation of the Maxilla

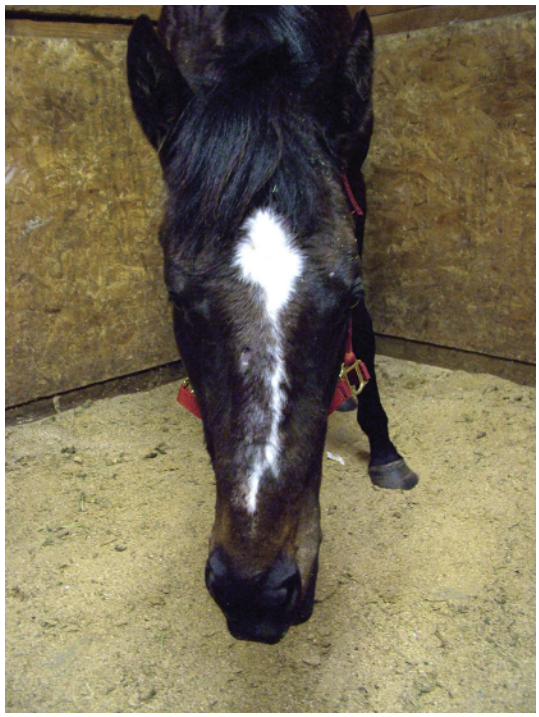


Figure 1.34 "Wry nose" in a 2-year-old Thoroughbred colt. His maxilla deviates to the right.

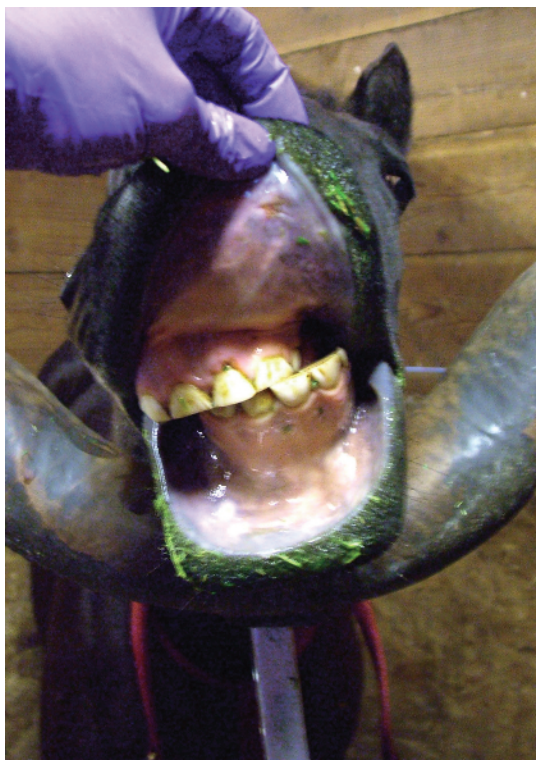


Figure 1.35 Same horse in fig. 1.34. Note the diagonal incisor malocclusion (DGL3) as a result of the deviation of the maxilla. This is a marked mandibular offset. Such malocclusions cannot be "corrected" but should be treated regularly to minimize overlong crown to maintain functional cheek teeth occlusion. If left unchecked as in this horse, it may progress to functional failure. This case exhibited other abnormalities including oligodontia, multiple diastemae, periodontal disease, and enophthalmos. See Diseases of the Respiratory System (Chapter 3) and Diseases of the Neonates (Chapter 12).

Asynchronous Teeth Eruption

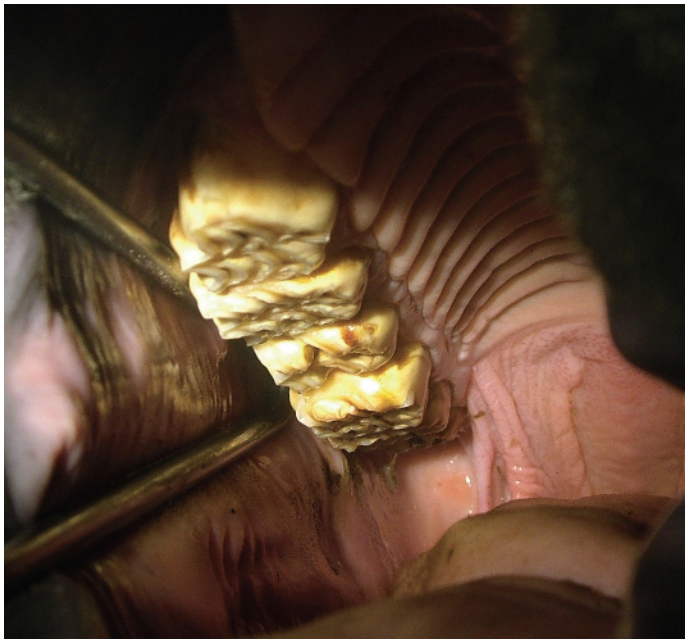


Figure 1.36 A 4-year-old horse with an overlong tooth 109. Note the difference in crown height when compared to tooth 110. Probable cause is asynchronous eruption. Tooth 109 likely erupted slightly before the 409. Failure to address this condition in the young horse can result in “wave” malocclusion.

Fractured Tooth

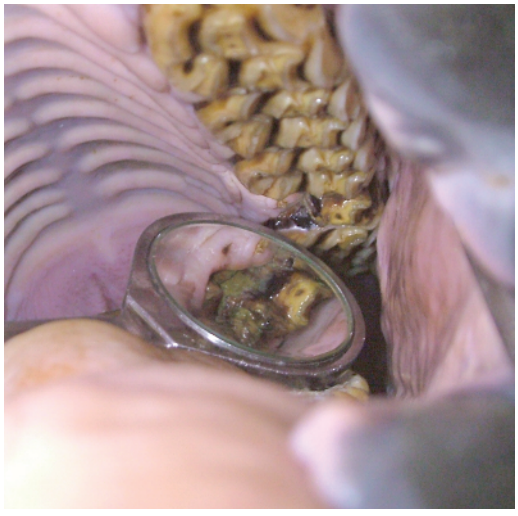


Figure 1.37a Cursory examination of the 200 teeth arcade reveals abnormality at the occlusal surface of tooth 209. When viewed with a dental mirror, a missing portion of the palatal crown was noted. Mirror also showed two small dental fragments embedded in the gingiva and mild superficial decay due to feed impaction.

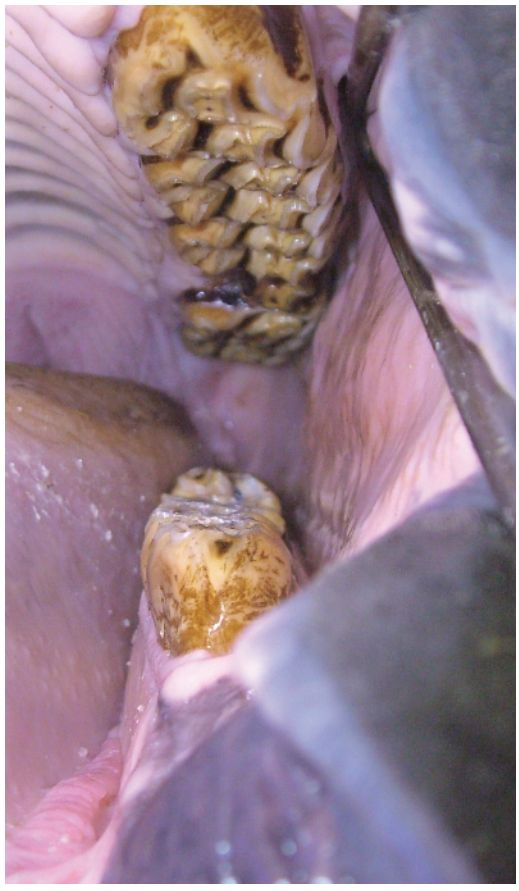


Figure 1.37b Same horse in fig. 1.37a. Overlong tooth 309 due to fractured tooth 209. Incomplete occlusion between the two teeth allows excessive crown overgrowth of the mandibular tooth. Commonly referred to as a "stepped tooth."

Figure 1.38a Sagittal fracture of tooth 206 in a middle-aged quarter horse mare. Fragments are displaced laterally and dorsally.

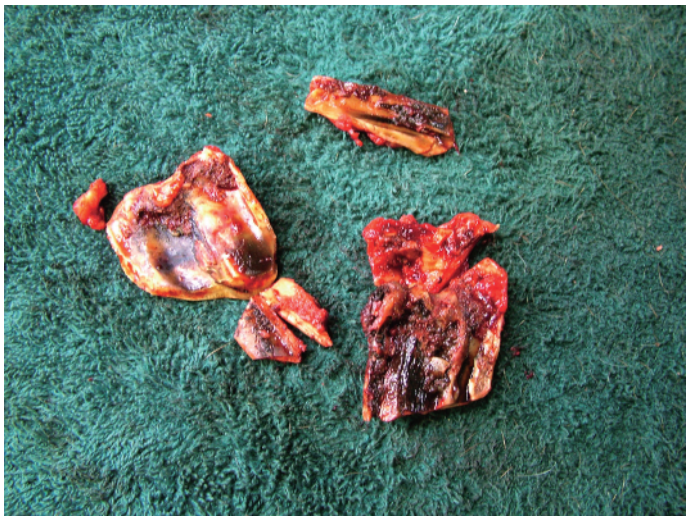


Figure 1.38b Extraction of the fractured tooth in fig. 1.38a. Multiple fragments were retrieved. Postprocedure radiographs are necessary to confirm that all fragments are removed.

Figure 1.39a An 8-year-old Warmblood gelding with sagittal fracture of tooth 308. Fracture line is through the 4th and 5th pulp chambers. Buccal fragment is loose but nondisplaced.

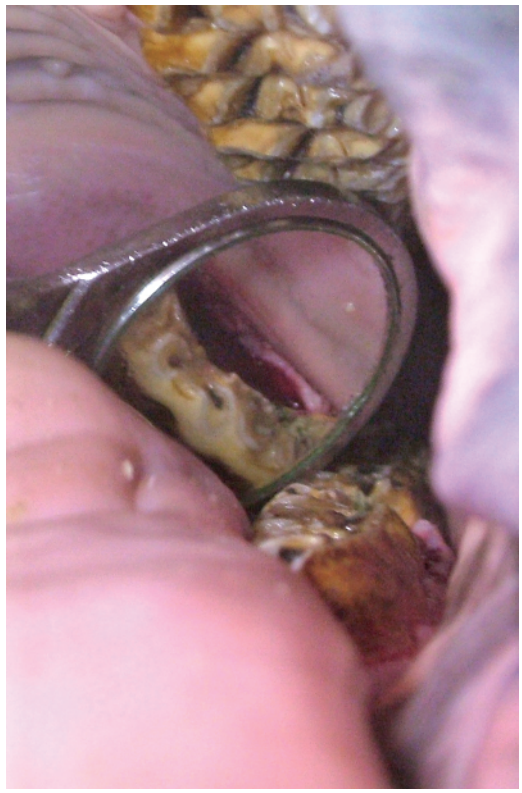


Figure 1.39b Same horse in fig. 1.39a. Appearance of tooth 308 following removal of buccal fragment. Radiographs showed no pulpar disease. Remaining tooth 308 was left in situ.

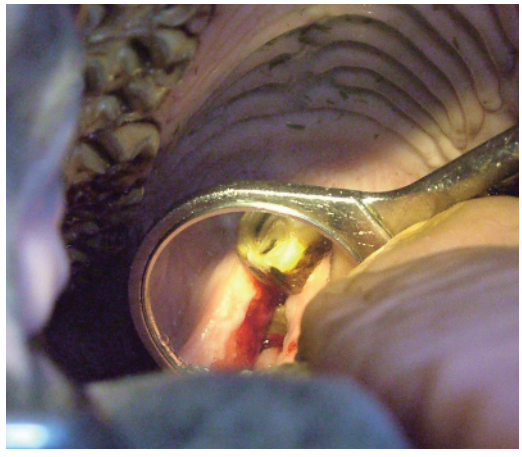


Figure 1.40a Tan object visible within the alveolus 8 weeks postextraction of fractured tooth 408. This is sequestrum of the alveolar wall. Pink mound distal to it is healthy granulation tissue. Subsequent extraction of sequestrae was curative.

Figure 1.40b Same horse in fig. 1.40a. Multiple sequestrae fragments were removed from tooth 408 alveolus.

Lingual and Buccal Laceration and Bit Pressure (Injury)

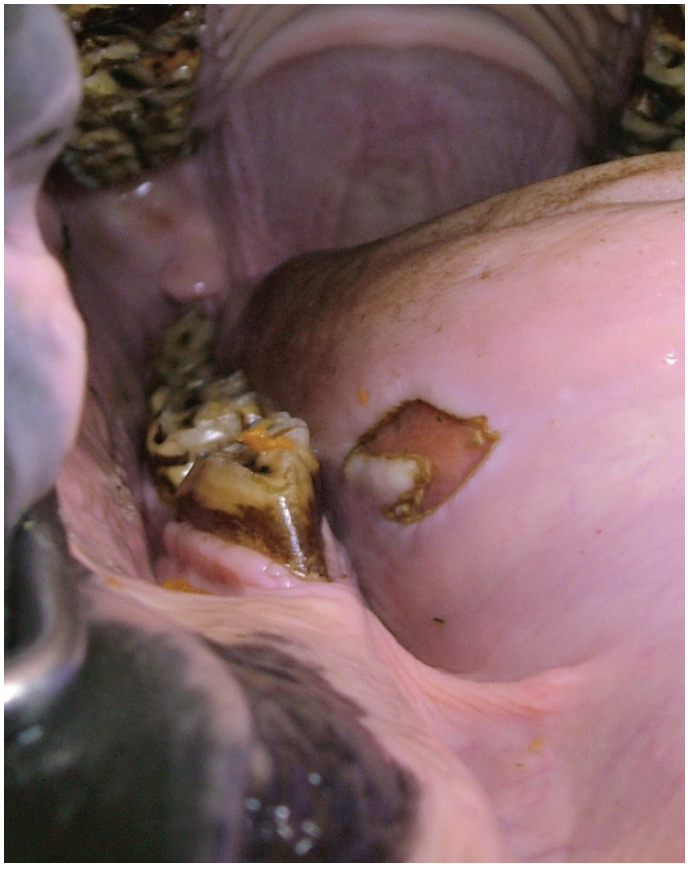


Figure 1.41 Lingual laceration in a 13-year-old Thoroughbred gelding presented with sudden reluctance to eat and increased salivation. Tooth 407 was fractured leaving a sharp shard of the tooth, which lacerated his tongue. Smoothing of the remaining portion of tooth 407 was curative. Fractured teeth can often be managed without extraction provided pulpar disease is not present.

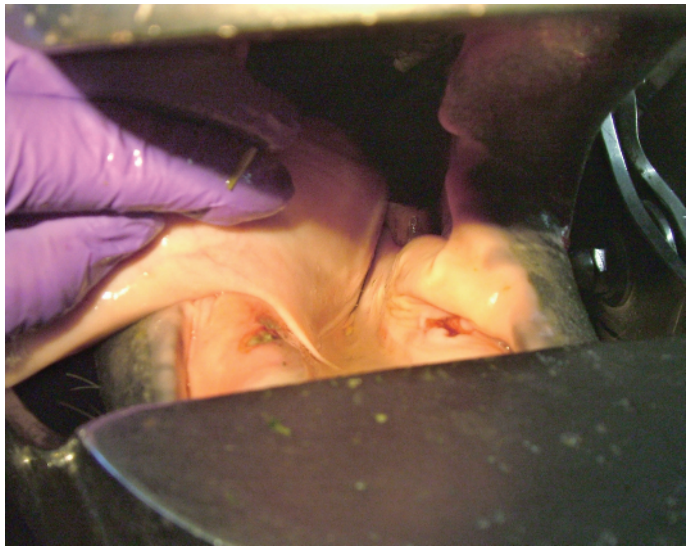


Figure 1.42a Bilateral soft tissue damage to the interdental spaces of a young quarter horse. Such injury is usually due to bit pressure, but autoinduced trauma using objects available in the horse's environment (i.e., edges and lips on feeders or water buckets) should be investigated. Radiographs may be indicated to rule out bony involvement.

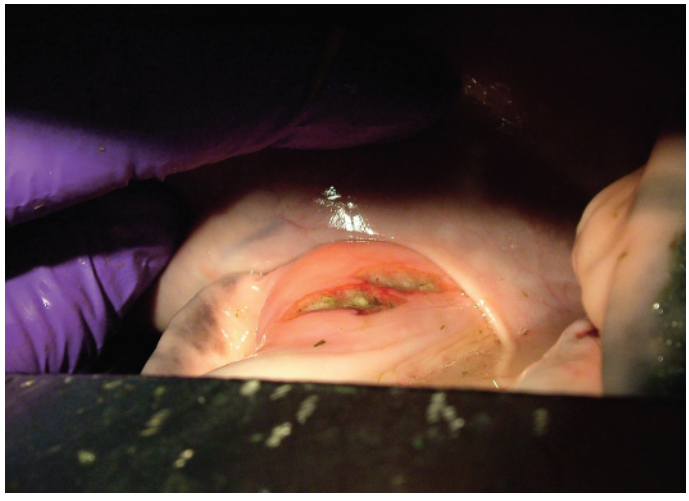


Figure 1.42b Close-up view of same horse in fig. 1.42a.

Figure 1.43 Chronic lingual laceration or ulceration due to sharp enamel points on the lingual aspects of the mandibular cheek teeth. Sharp points from fractured teeth or foreign bodies also cause similar soft tissue injuries.

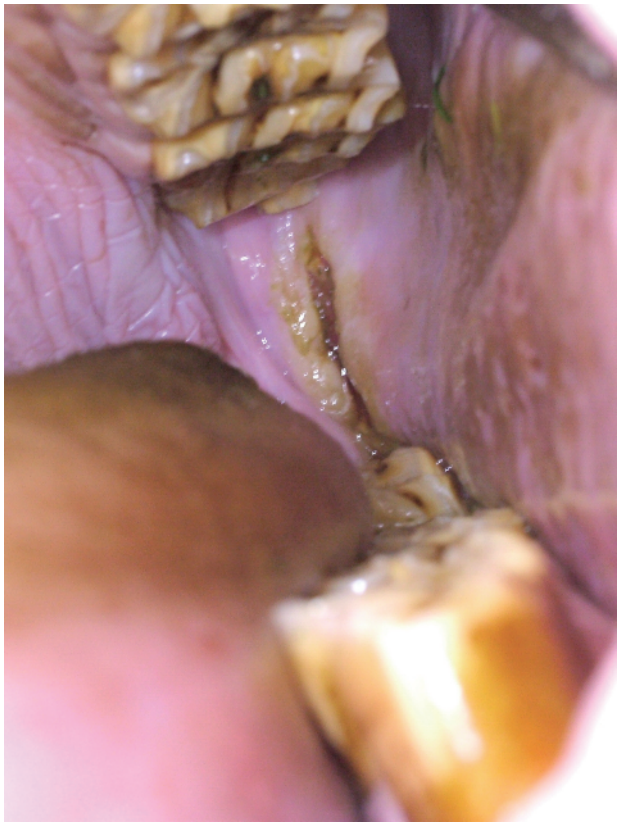


Figure 1.44 Severe laceration or ulceration of the oral mucosa caused by sharp enamel point on the distal aspect of 211 as it occludes with 311. Lacerations of this type illustrate the need for thorough oral examination with a full mouth speculum and powerful light source. This lesion heals spontaneously following removal of offending enamel point. Recurrence is likely if point is created due to persistent malocclusion and can be prevented by intervention at appropriate intervals.

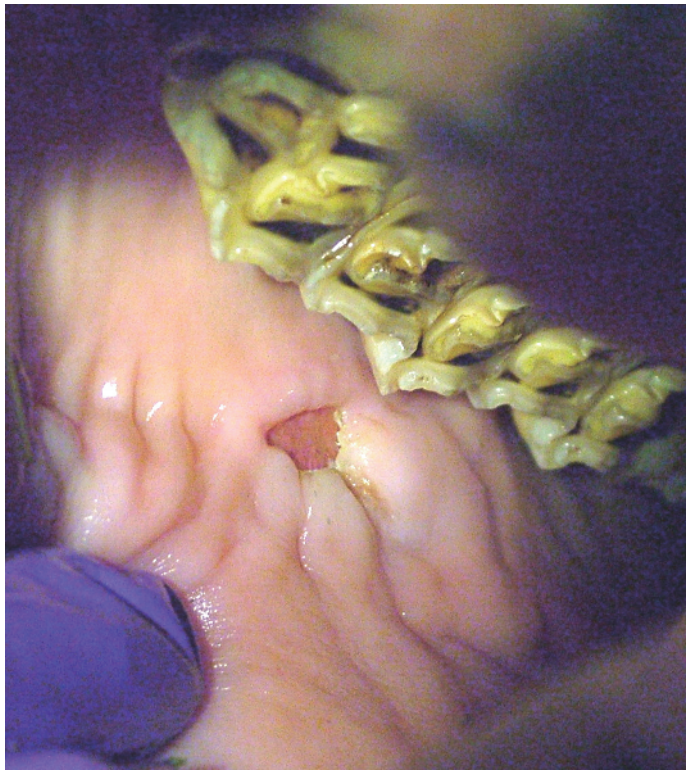


Figure 1.45 Buccal mucosal ulcer associated with sharp enamel point at the level of tooth 107. Such ulceration can cause performance impedance, abnormal mastication, or inappetence. Horses under the age of 8 years can develop sharp enamel points faster than mature horses. Some juveniles require routine care every 6 months to prevent such injury.

Gingival and Lingual Ulceration of Systemic Origin

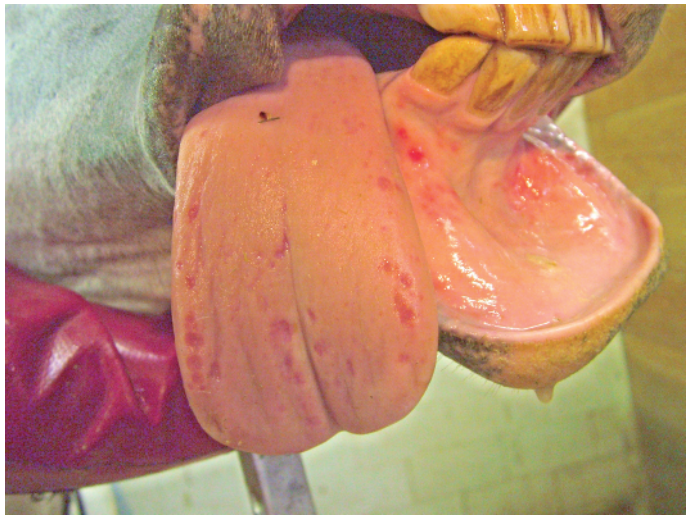


Figure 1.46 Multifocal gingival and lingual ulcerations that were found during routine oral examination of this middle-aged Thoroughbred mare. No other clinical signs were noted. No significant dental pathology was present. A thorough history, physical examination, and blood work are necessary to rule out significant viral pathogens. It is the veterinarian's responsibility to maintain adequate hygienic practices and disinfect instruments between patients and facilities to prevent disease spread.

Supernumerary Canine Tooth

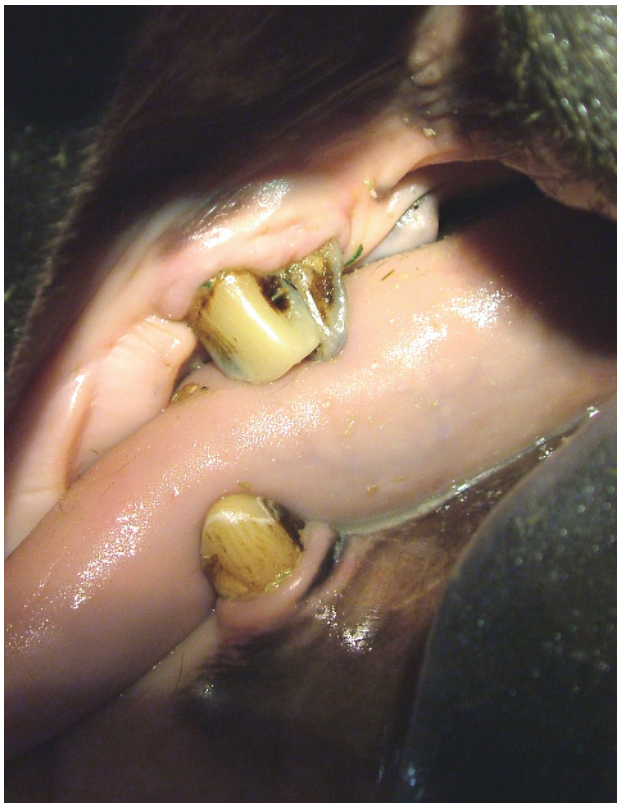


Figure 1.47 Aged horse with supernumerary maxillary canine tooth. No treatment was necessary since no periodontal disease was present. Conservative shaping of clinical crown is recommended to prevent soft tissue trauma.

Polydontia

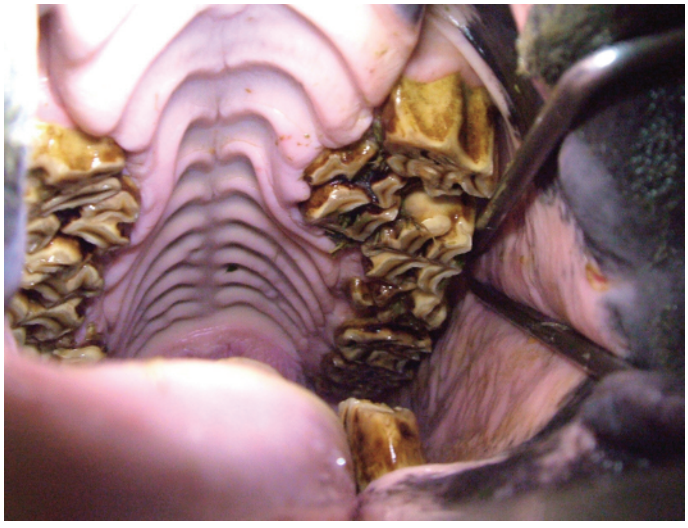


Figure 1.48 A 5-year-old draft mare with polydontia in maxillary teeth arcades. Supernumerary tooth is medial to apparent tooth 207. Feed was impacted between the supernumerary tooth and adjacent teeth resulting in periodontal disease. Some “extra teeth” may not be true supernumerary teeth and are retained deciduous teeth. Radiograph is warranted to find out if the supernumerary tooth is deciduous. Extraction of the displaced tooth is necessary to resolve periodontal disease.

Dysplastic Teeth

Figure 1.49 Dysplastic tooth 107 in an aged miniature horse with abnormal mastication. Note the abnormal architecture at the occlusal surface and the location of the gingival margin at the level of teeth 107/8 junction. This indicates bony remodeling due to chronic malocclusion. There is a small periodontal pocket at the level of teeth 106/7 and tooth 107 is loose. Radiographs showed evidence of chronic disease. The tooth was extracted and symptoms resolved.

Abnormal Tooth Wear

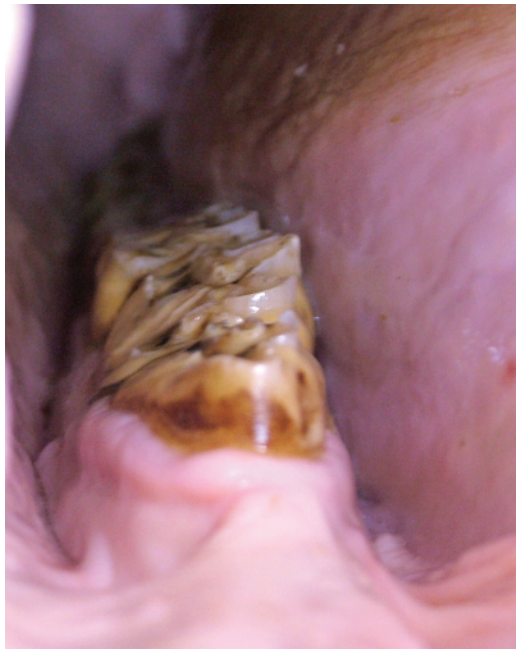


Figure 1.50 Abnormal wear at buccal aspect of tooth 406 and 407 in a 10-year-old paint gelding. Note the difference in appearance of the buccal aspect of 408. Suspected etiology is abnormal enamel formation of 406 and 407 allowing increased focal wear.

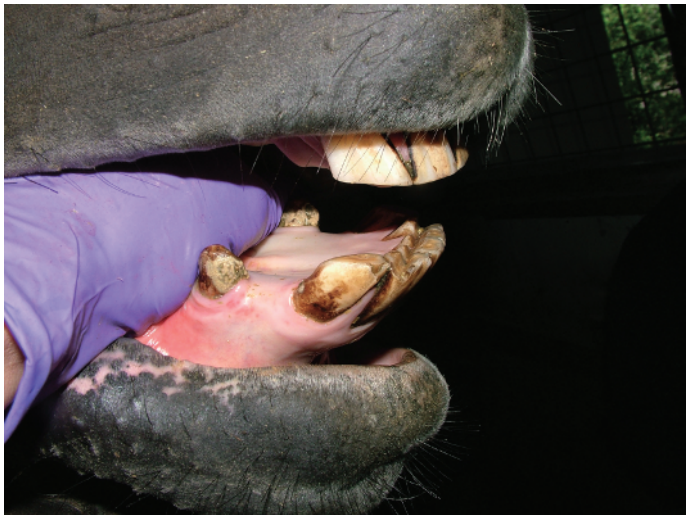


Figure 1.51 Severe atypical wear of mandibular incisors. This middle-aged horse is a known "cribber." His new stall door has a metal cap with an exposed edge that he was able to grasp causing extreme grooving on his mandibular incisors. Immediate removal of the metal cap arrested this wear. Pulpar exposure or fracture is possible if the situation were to continue unchecked.

Squamous Cell Carcinoma

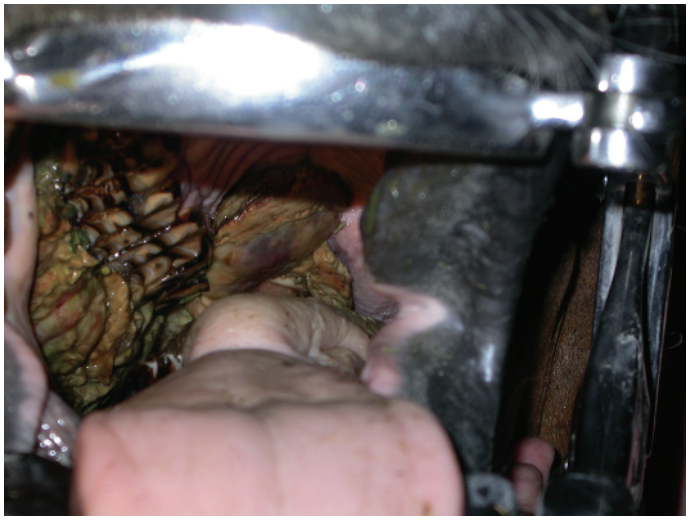


Figure 1.52 Oral squamous cell carcinoma (SCC) in a horse. SCC is the most common oral soft tissue tumor. It occurs in older horses and can involve any region of the mouth and is associated with fetid smell. (Image courtesy of Dr. Stephen Manning, WCVM, University of Saskatchewan)

Oral Foreign Body

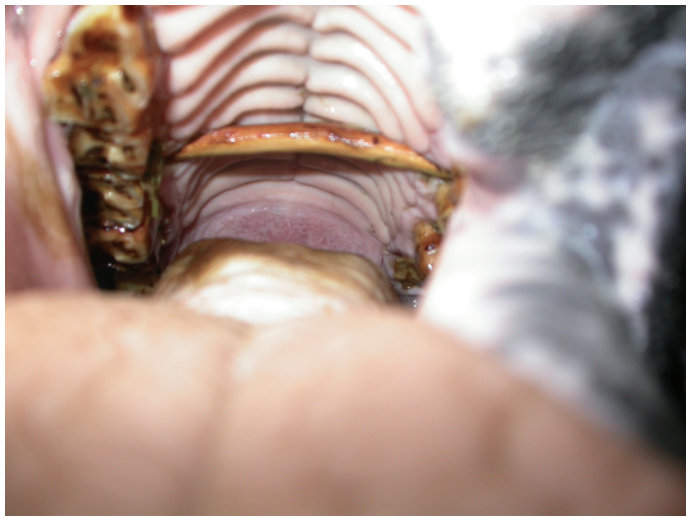


Figure 1.53 Oral foreign body in a horse. In this case there was a wooden stick lodged between the upper arcade of teeth. Affected horse may present with signs of dysphagia. Wooden sticks may also penetrate oral soft tissues and cause cellulitis. Wooden stick is the most common foreign body found in the oral cavity. (Image courtesy of Dr. Stephen Manning, WCVM, University of Saskatchewan)

Glossitis

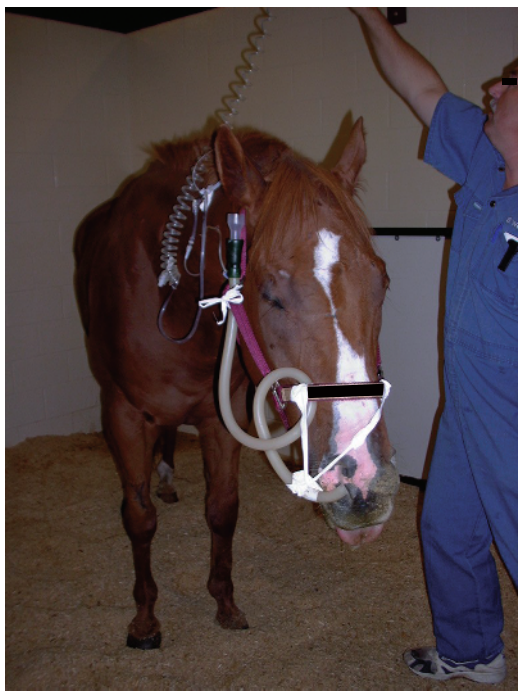


Figure 1.54 An adult horse affected with severe traumatic glossitis (trauma while oral dosing). The tongue was swollen and protruded from the mouth. The site of trauma can be seen in fig. 1.55. A nasogastric tube was used to feed the horse because the horse was severely dysphagic.

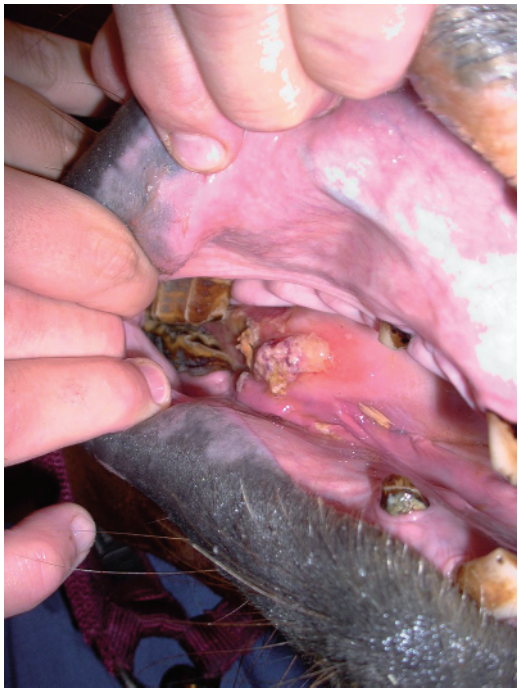


Figure 1.55 The site of trauma (penetrating) that has led to severe glossitis in fig. 1.54.

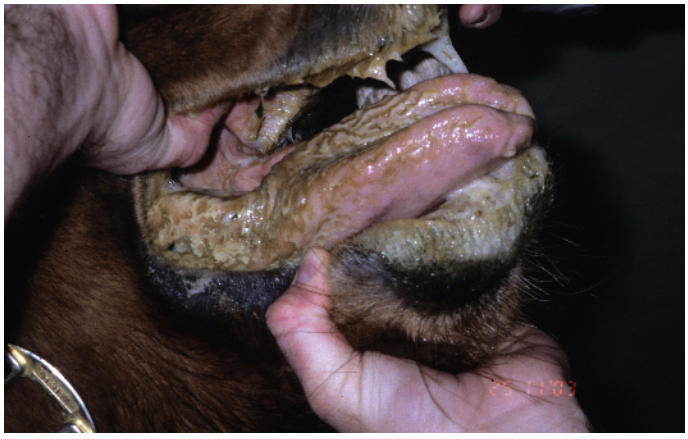


Figure 1.56 Glossitis in an adult horse that was suspected to be secondary to accidental ingestion of irritant chemical agent.

Esophageal Obstruction (Choke), Primary

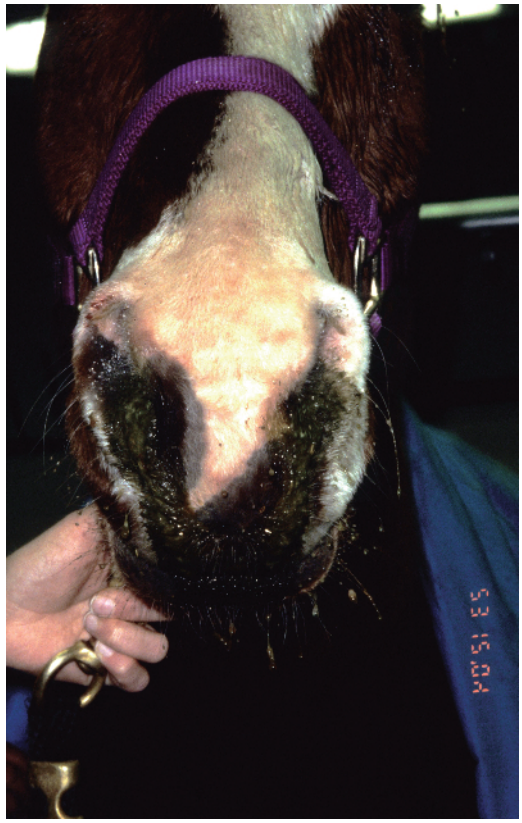


Figure 1.57 Choke in an adult horse; note the green nasal discharge. Choke or esophageal obstruction is the most common esophageal disorder seen in horses. Primary choke is usually caused by feed or foreign bodies (e.g., stones, bedding, medicinal boluses, carrot, potato, or wood fragments).

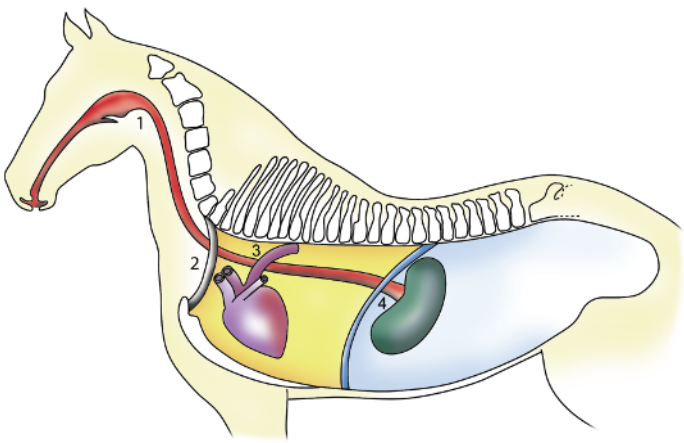


Figure 1.58 There are four common anatomical areas of natural narrowing where primary choke usually occurs; these are (1)the postpharyngeal area, (2)thoracic inlet, (3)base of the heart, and (4)cardia of the stomach (terminal esophagus). (Drawing by Dr. Juliane Deubner, WCVM, University of Saskatchewan)

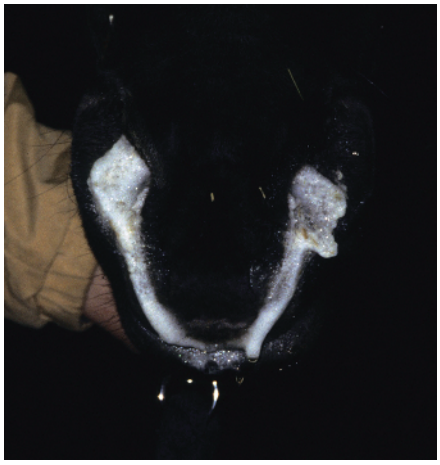
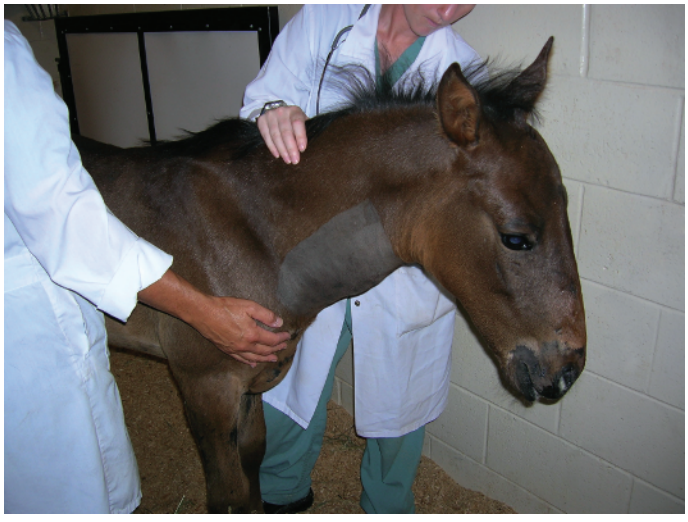
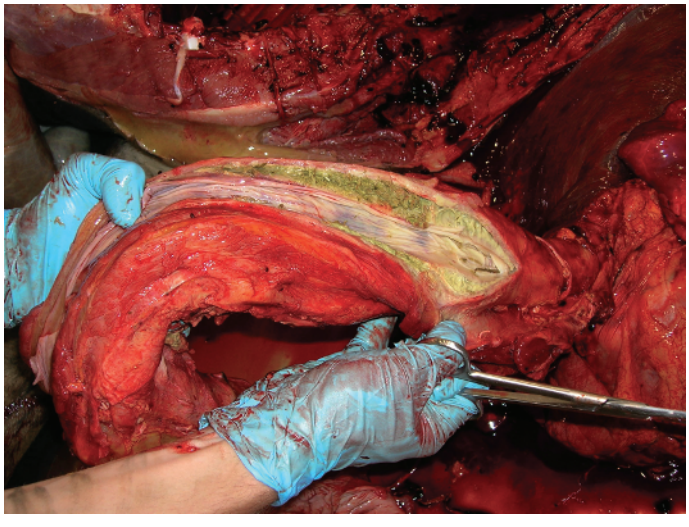


Figure 1.59 Clinical signs are usually acute and include anxiety, coughing, standing with the head and neck extended, gagging or retching, painful and repeated attempts at swallowing, bilateral white frothy nasal discharge, as in this photograph, or green and containing feed material. (Image from Abutarbush SM and Carmalt JL, Endoscopy and arthroscopy for the equine practitioner. Made Easy Series. Jackson, WY: Teton NewMedia, 2008)



a



b

Figures 1.60a,b Esophageal laceration and peri-esophageal cellulitis in a foal secondary to esophageal obstruction. Note the cervical swelling (fig. 1.60a) and the peri-esophageal inflammation and feed accumulation in the postmortem photograph (fig. 1.60b). The esophagus can rupture secondary to esophageal obstruction. This will lead to cellulitis or crepitus, and a palpable or visible mass on the left lateroventral aspect of the neck, if the obstruction is in the cervical area of the esophagus. (Image 1.59a from Abutarbush SM and Carmalt JL, Endoscopy and arthroscopy for the equine practitioner. Made Easy Series. Jackson, WY: Teton NewMedia, 2008)

Figure 1.61 An endoscopic image of esophageal obstruction. Diagnosis of primary choke is based on a history, physical examination findings, inability or difficulty in passing a nasogastric tube to the stomach, ultrasonography, endoscopy, or radiography. (Image from Abutarbush SM and Carmalt JL, Endoscopy and arthroscopy for the equine practitioner. Made Easy Series. Jackson, WY: Teton NewMedia, 2008)

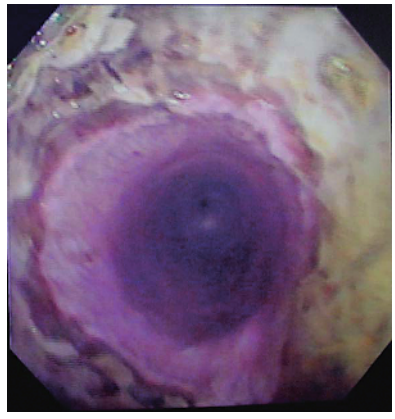


Figure 1.62 Endoscopic image of an esophagus after resolution of esophageal obstruction. Following resolution of choke, endoscopy can also be used to determine if ulceration, as in this photograph, perforation, masses, or strictures are present, which helps guide therapy and determine the prognosis. (Image from Abutarbush SM and Carmalt JL, Endoscopy and arthroscopy for the equine practitioner. Made Easy Series. Jackson, WY: Teton NewMedia, 2008)

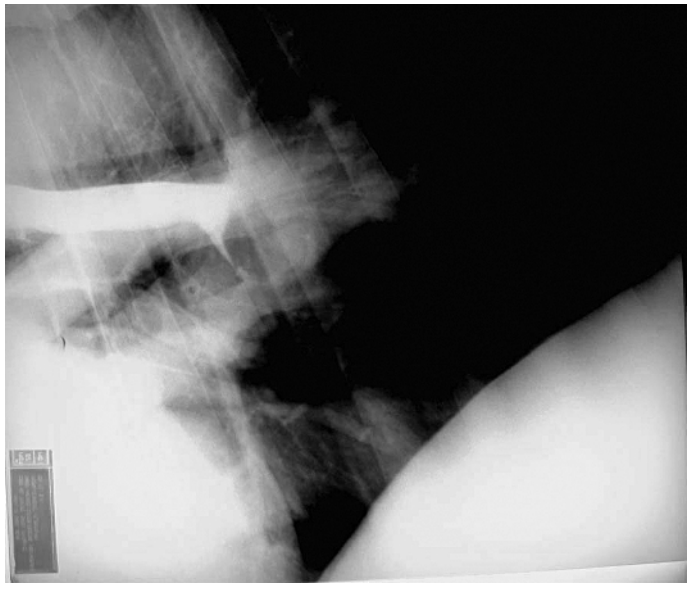


Figure 1.63 Radiological examination of the cervical and thoracic esophagus can be performed with portable equipment, but high-powered equipment and a grid are required to view the esophagus at the level of the shoulder and thoracic inlet. Plain films may be helpful but in most cases contrast radiography is more informative, as in this case.

Figure 1.64 To treat the esophageal obstruction, esophageal lavage can be performed in standing horses under profound sedation in order to keep the head low and prevent aspiration. Warm water can be pumped gently using a stomach pump through a cuffed or uncuffed tube into the esophagus cranial to the obstruction, while the tube is gently manipulated against the obstruction. The returning water and impacted material often comes out of the nose or the mouth of the horse and should be examined to determine the cause and nature of the impaction.

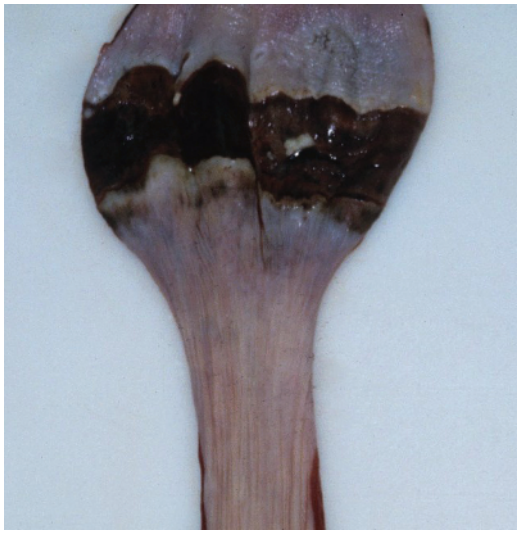


Figure 1.65 Postmortem image of circumferential esophageal ulceration in a horse secondary to long-standing esophageal obstruction. This type of ulceration is the one that is likely to result in esophageal stricture and narrowing. Other complications of esophageal obstruction that can be seen include stricture, perforation, megaesophagus (fig. 1.66), reobstruction, and aspiration pneumonia (fig. 1.67). (Image from Abutarbush SM and Carmalt JL, Endoscopy and arthroscopy for the equine practitioner. Made Easy Series. Jackson, WY: Teton NewMedia, 2008)

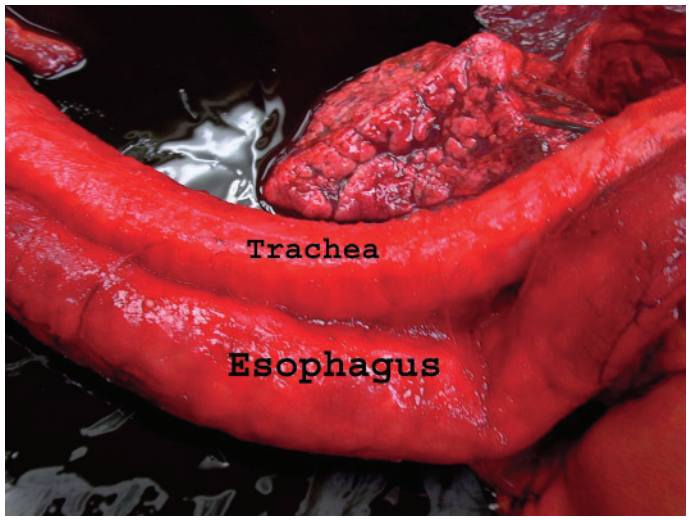


Figure 1.66 Postmortem photograph of megaesophagus in a foal secondary to esophageal obstruction.

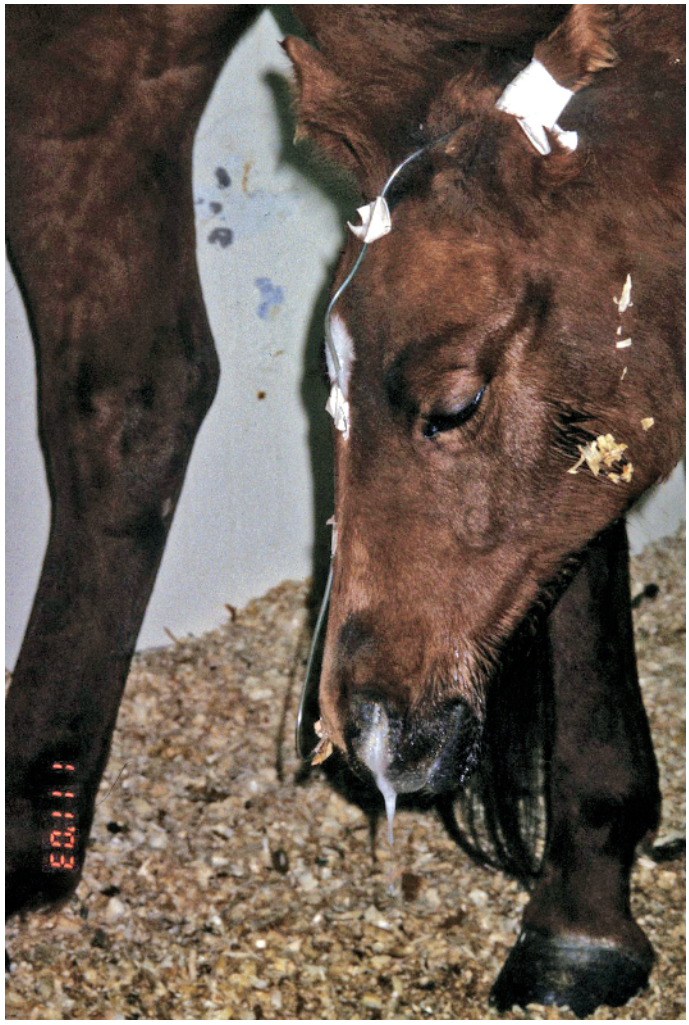
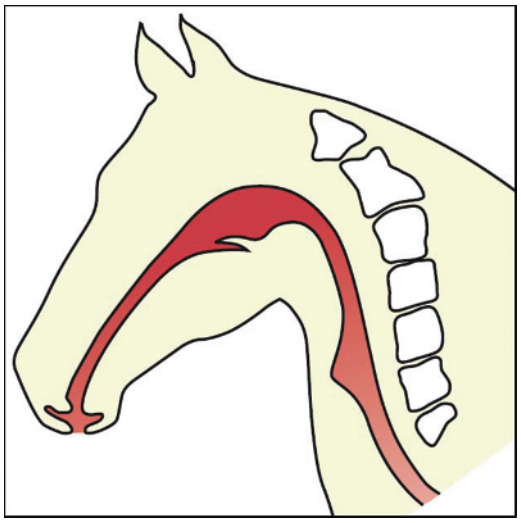
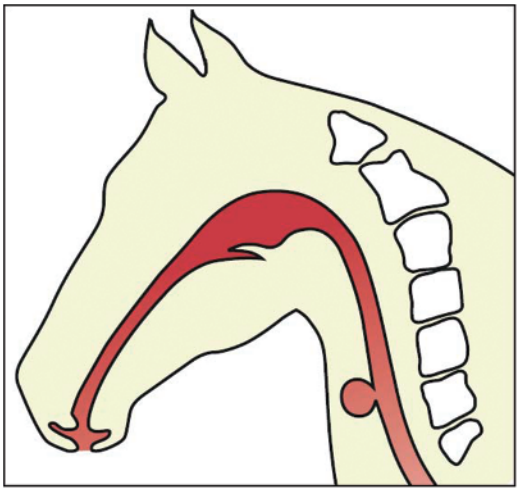


Figure 1.67 Aspiration pneumonia secondary to esophageal obstruction in a foal. Note the severe depression and nasal discharge.



a



b

Figures 1.68a,b Drawings of esophageal diverticulae. Secondary choke is caused by intralumenal or extralumenal abnormalities that mechanically impede feed passage. Intralumenal abnormalities include esophageal stricture, diverticula, cysts, and tumors. Horses usually have recurrent choke episodes. A diverticulum is a focal outpouching of the esophagus with an intact mucosa. There are two types of diverticulae: traction and pulsion diverticulum. In a traction diverticulum, the neck of the sac is much wider than the bottom (fig. 1.68a). In a pulsion diverticulum, the neck of the sac is narrower than the bottom (fig. 1.68b). Contrast radiography can be used to diagnose the presence of esophageal diverticula (fig. 1.69). Mediastinal and cervical masses (tumor or abscess), and vascular ring anomalies may cause extralumenal obstruction by impinging on the esophagus. (Images from Abutarbush SM and Carmalt JL, Endoscopy and arthroscopy for the equine practitioner. Made Easy Series. Jackson, WY, Teton NewMedia, 2008)

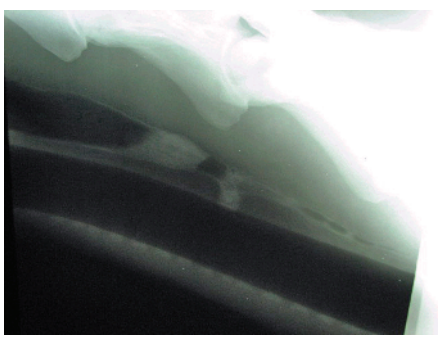


Figure 1.69 Radiographic image with contrast of a pulsion diverticulum in a horse.

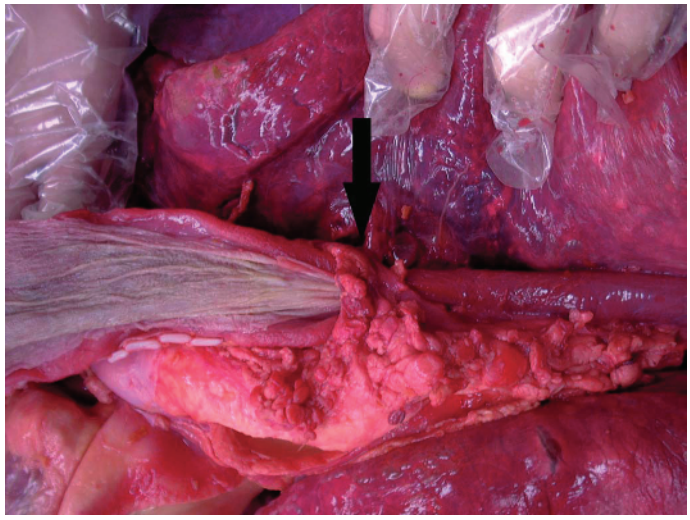


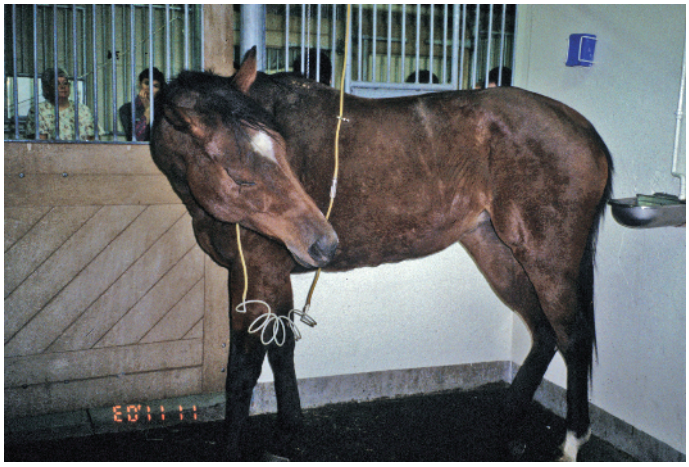
Figure 1.70 Postmortem photograph of vascular ring anomalies in a foal. It is a congenital anomaly of the aortic arch and its branches. These vessels may form a ring around the esophagus, which can lead to secondary esophageal obstruction. In foals, clinical signs start when the foal begins to eat solid feed. (Image from Abutarbush SM and Carmalt JL, Endoscopy and arthroscopy for the equine practitioner. Made Easy Series. Jackson, WY: Teton NewMedia, 2008)

Diseases of the Abdominal Region

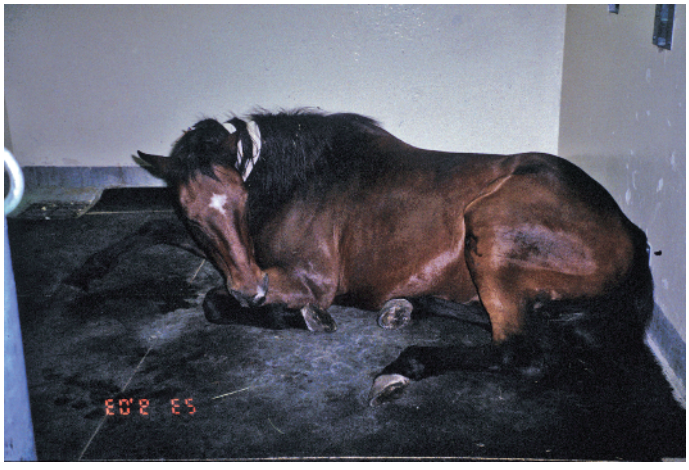
Abdominal Pain (Colic)

Colic is one of the most common problems in equine practice. Colic in horses can be divided into two major categories: gastrointestinal and nongastrointestinal. Nongastrointestinal colic cases are those showing signs of abdominal pain due to causes related to urinary, reproductive, nervous, respiratory, or musculoskeletal system disorders. Gastrointestinal colic is usually caused by gut distension, tension on the root of mesentery, ischemia, deep ulceration of the gastrointestinal tract, or peritoneal pain. Strangulating and nonstrangulating obstruction of the small and large intestines causes different degrees of abdominal pain.

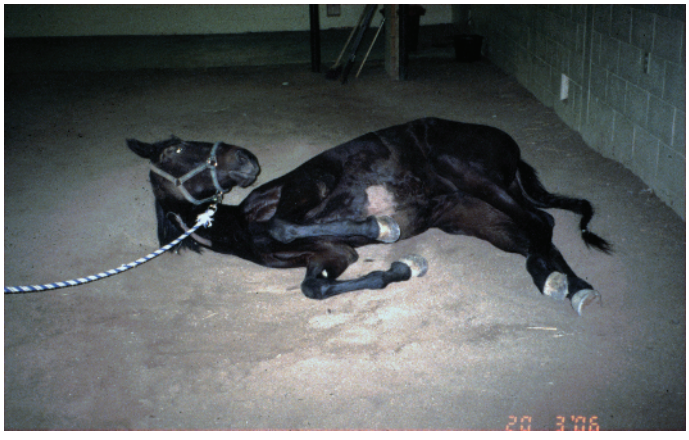
Clinical signs of colic include agitation, flank watching (figs. 1.71–1.74), pawing (fig. 1.75), stretching (figs. 1.76 and 1.77), kicking at the abdomen, frequent lying down (figs. 1.78–1.80), and rolling (figs. 1.81–1.84).



1.71



1.72



1.73

Figures 1.71–1.74 Clinical signs of colic include agitation and flank watching.

1.74

Figures 1.71–1.74 *Continued*

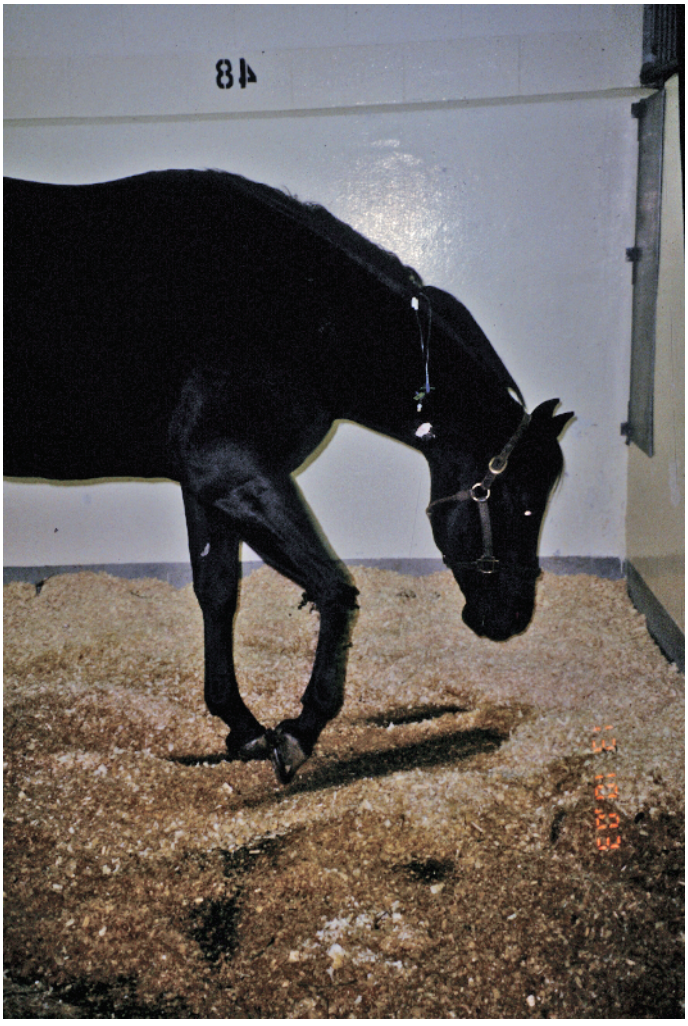
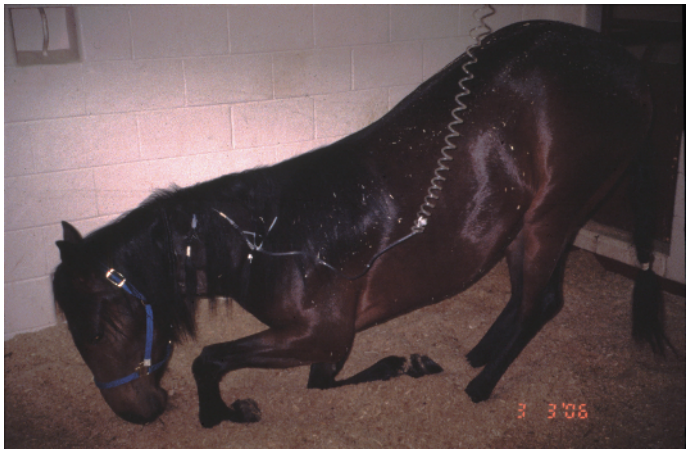
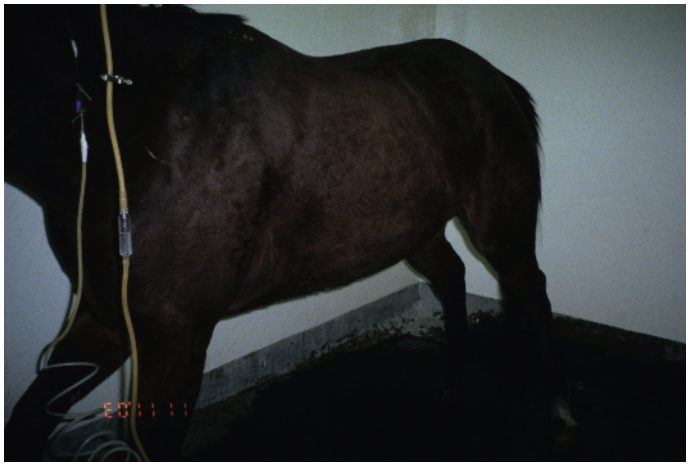


Figure 1.75 Pawing is another clinical sign of colic in horses.

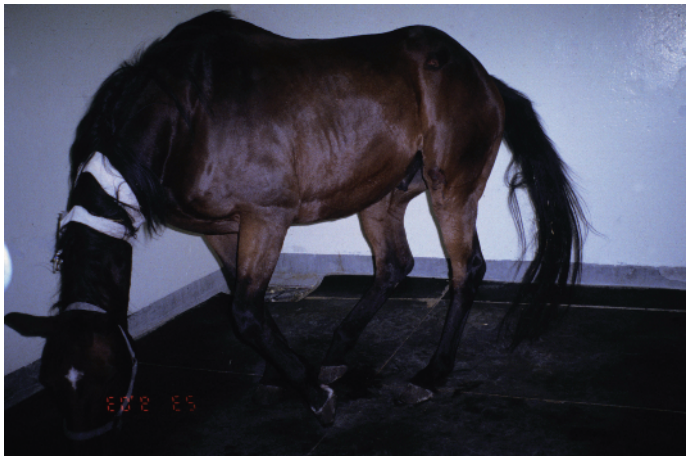


1.76



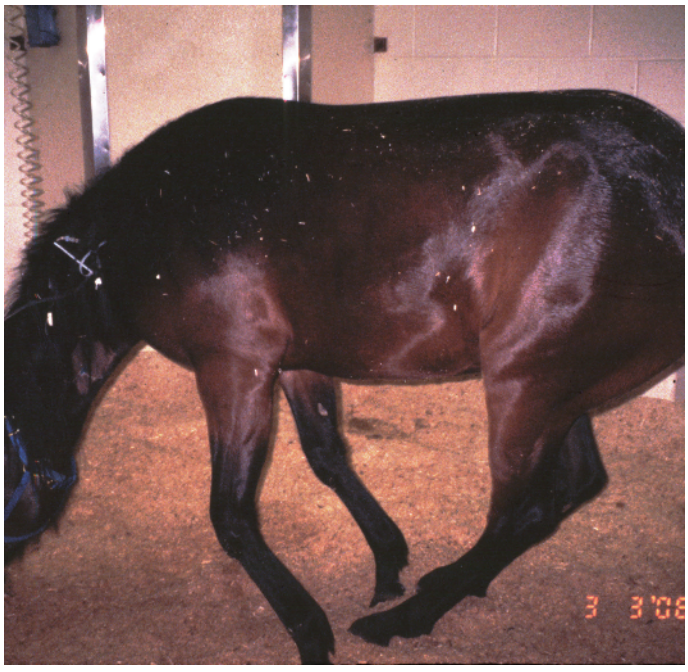
1.77

Figures 1.76–1.77 Colicky horses may stretch as in these figures.



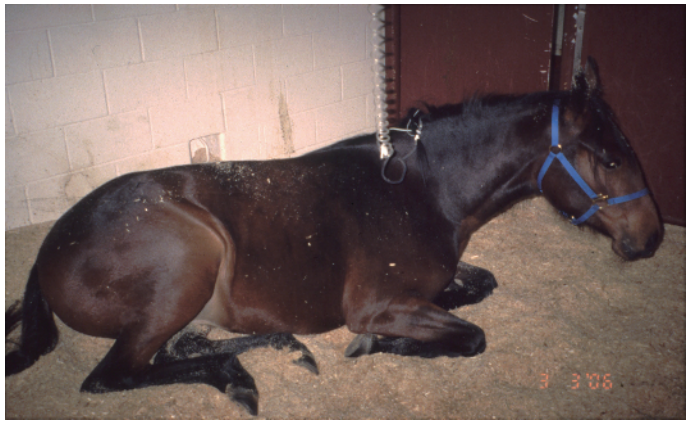
1.78

Figures 1.78–1.80 Colicky horses may kick at their abdomen and lie down frequently.



1.79

Figures 1.78–1.80 *Continued*



1.80

1.81

Figures 1.81–1.84 Rolling is a sign of severe colic in horses.

1.82

1.83

1.84

Figures 1.81–1.84 *Continued*

Diseases of the Stomach

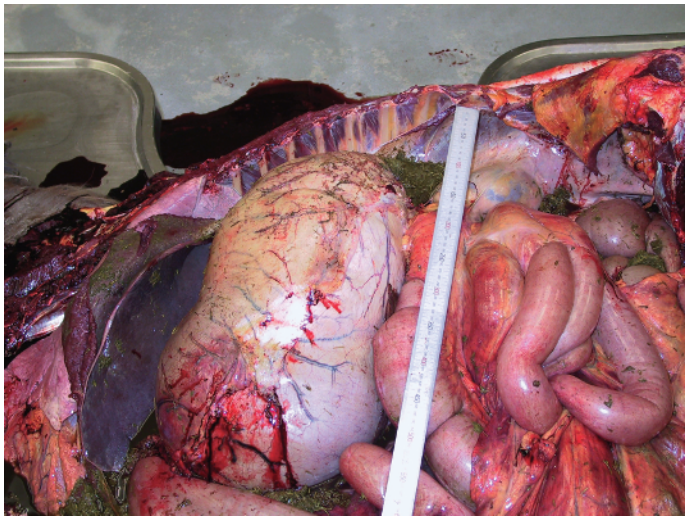
Gastric Dilatation

Figure 1.85 Gastric dilatation in a horse with small intestinal obstruction. Note the nasogastric reflux. Gastric dilatation is caused by gastric outflow obstruction (pyloric stenosis), intestinal contents reflux secondary to small intestinal obstruction, grain overload, gastric dilatation with air (aerophagea). So usually it is a secondary event to another disease, although it can be idiopathic. Clinical signs are not specific and are mainly abdominal pain in addition to other signs related to the associated condition. Some horses may regurgitate or vomit, which usually causes stomach rupture and is usually a terminal event. Passing a nasogastric tube is usually a lifesaving procedure and should be left in place to avoid rupture of the stomach (fig. 1.86). Stomach rupture and septic peritonitis can be the result of long-standing dilatation of the stomach (fig. 1.87).

Figure 1.86 The nasogastric tube is left in place to avoid rupture of the stomach in a horse with gastric dilatation and large volume of nasogastric reflux.

Figure 1.87 Postmortem photograph of a stomach rupture in a horse secondary to gastric dilatation.

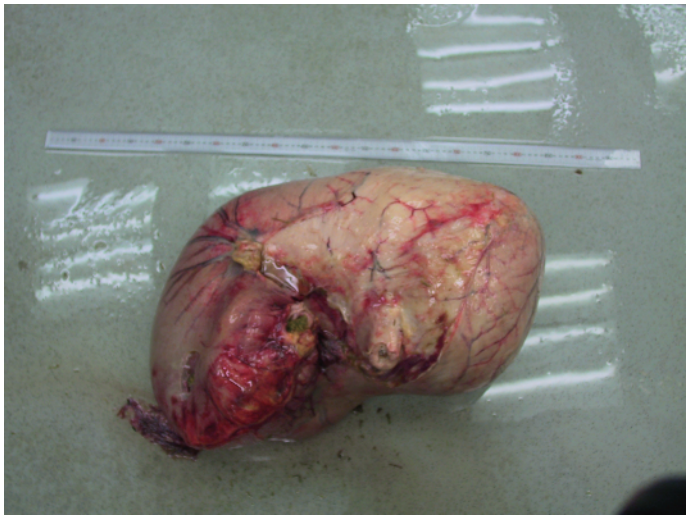
Gastric Impaction



1.88

Figures 1.88–1.89 Postmortem photographs of gastric impaction in a horse. Gastric impaction can be caused by insufficient access to water, poor teeth, and atony in old horses. Affected horses are presented with abdominal pain and regurgitation of ingesta and fluids through the nostrils may be seen. Stomach may rupture in some horses (fig. 1.90).

Figures 1.88–1.89 *Continued*



1.89

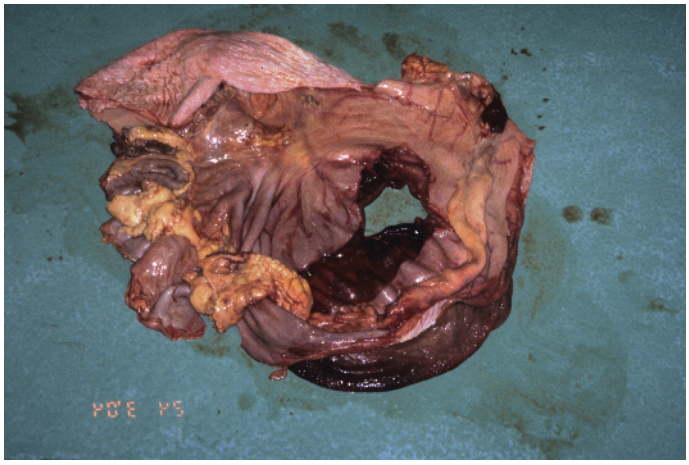


Figure 1.90 Postmortem photograph of gastric (stomach) rupture in a horse secondary to gastric impaction.

Gastric Ulcers

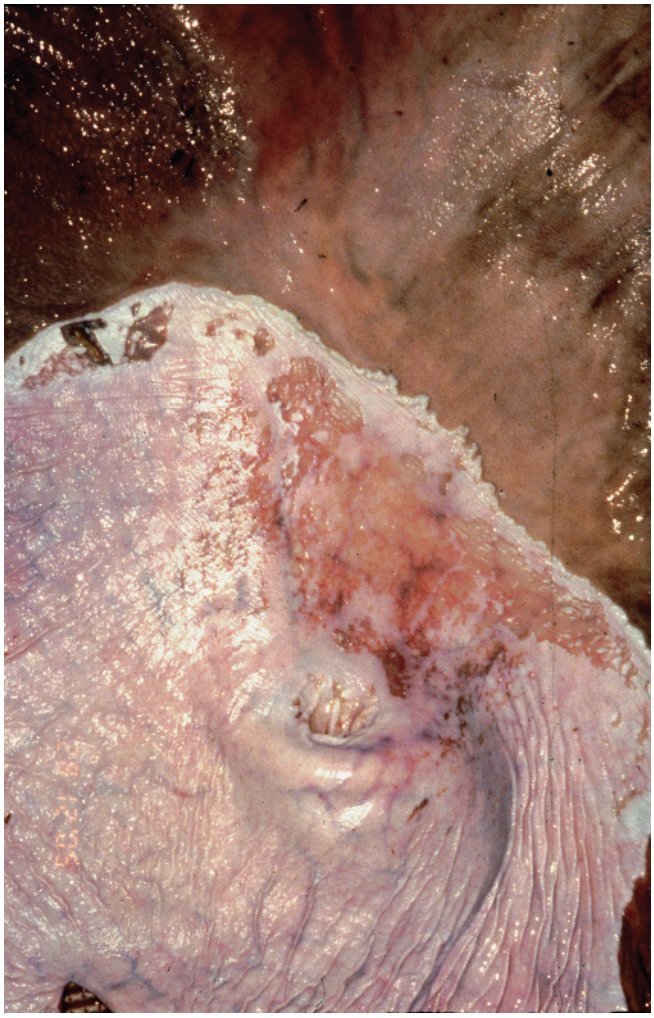


Figure 1.91 A postmortem photograph of gastric ulceration of the nonglandular part of the stomach. Gastric ulcers can be seen in foals and adult horses. Except for nonsteroidal anti-inflammatory drug toxicity, the exact cause is unknown. It can occur on the glandular (fig. 1.92) part of the stomach, nonglandular part of the stomach, or along the margo plicatus (fig. 1.93). Gastric ulcers are not clinical in most affected animals. Affected foals may show bruxism, ptyalism, froth at the mouth (fig. 1.94), colic signs (fig. 1.95), and dorsal recumbency (fig. 1.96). Please see Chapter 12, Diseases of the Neonates. Weanlings affected by chronic gastric ulceration are usually presented with intermittent colic and diarrhea and poor growth and hair coat. In adult horses, signs are mostly inapparent and vague. Affected horses may show mild intermittent colic, poor appetite and performance, and poor body condition. Endoscopic examination of the stomach provides a definitive diagnosis (fig. 1.97).

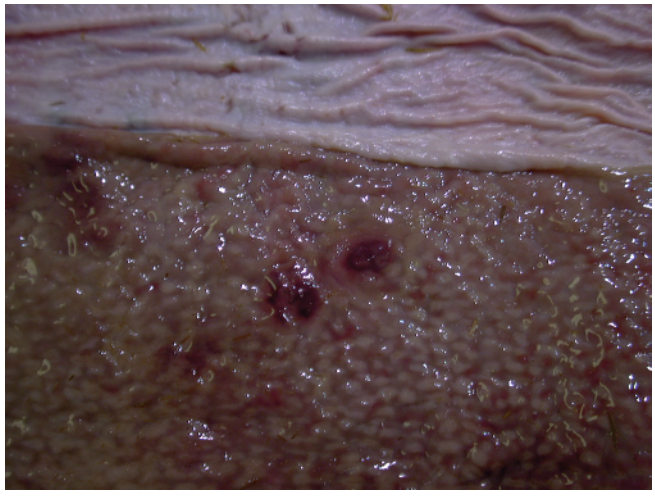


Figure 1.92 A postmortem photograph of gastric ulceration of the glandular part of the stomach.

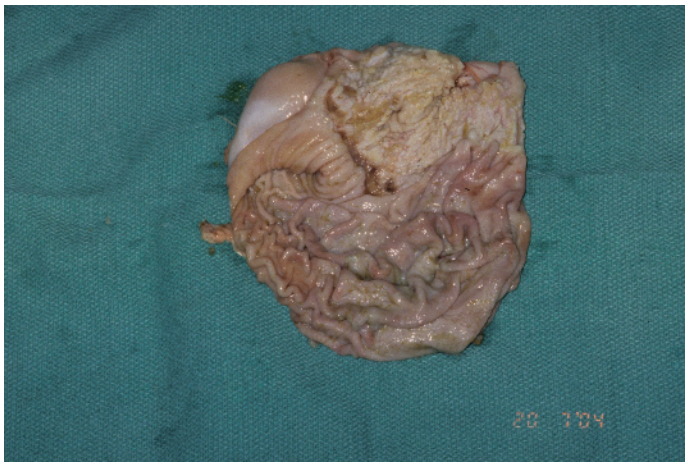


Figure 1.93 A postmortem photograph of gastric ulceration along the margo plicatus in a foal.

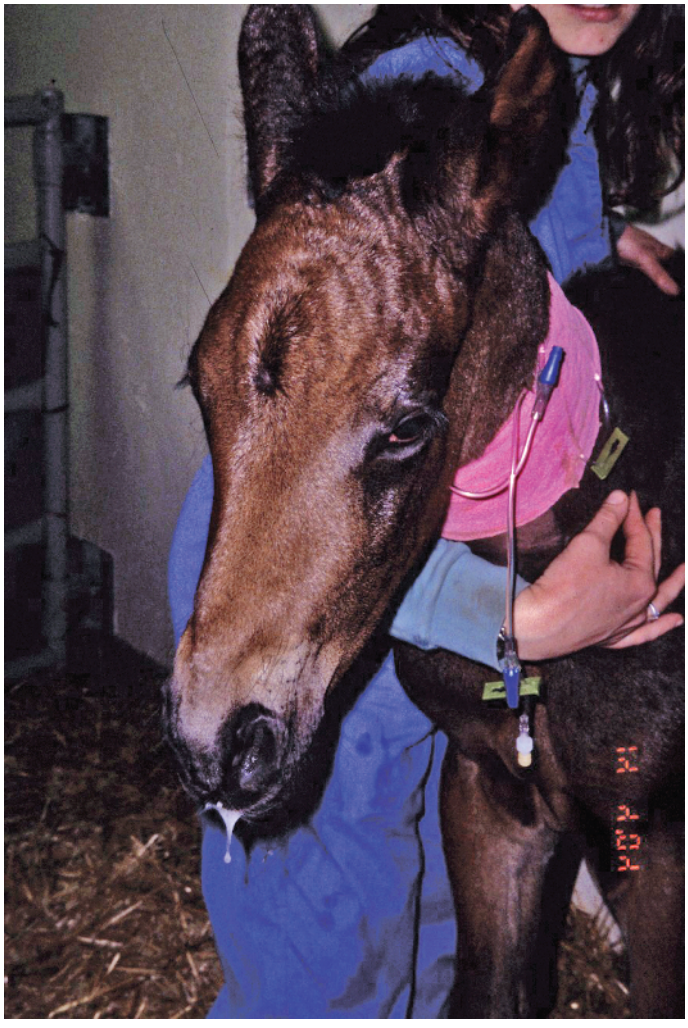


Figure 1.94 Foal affected with gastric ulcers. Note the presence of a froth at the mouth.

Figure 1.95 Foal affected with gastric ulcers showing signs of colic (rolling).

Figure 1.96 Foal affected with gastric ulcers. Note the dorsal recumbency.

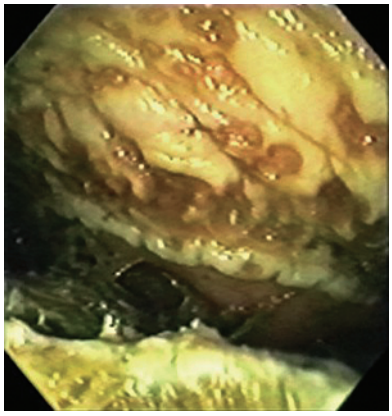


Figure 1.97 Endoscopic image of gastric ulcers of the nonglandular part of the stomach.

Diseases of the Small Intestine

Simple Obstruction of the Small Intestine

It is manifested by obstruction of the intestinal lumen only. Simple obstruction of the small intestine is usually associated with various degrees of abdominal pain. The presence of a nasogastric reflux and abnormal peritoneal fluid will depend on the stage of the disease and its location, the latter especially important or absence of nasogastric reflux.

Ileal Impaction

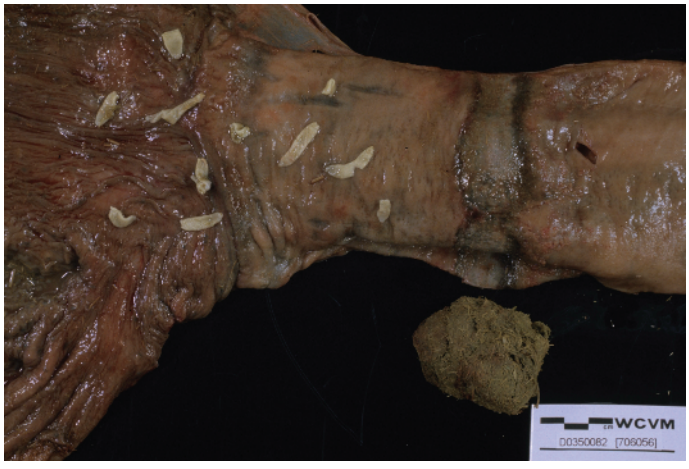


Figure 1.98 Postmortem photograph of ileal impaction in a horse. Note the circumferential mucosal ulceration of the ileum due to the impaction. This disease is usually seen in adult horses in the southwestern United States. There are a few tapeworms in the cecum.

Ileal Hypertrophy

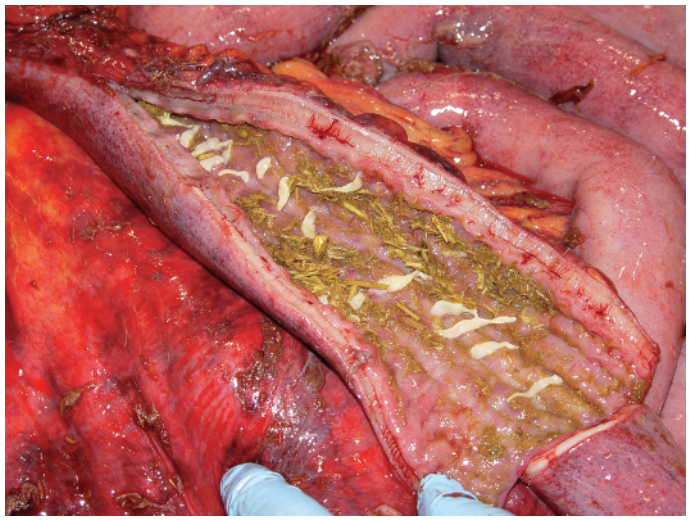


Figure 1.99 Postmortem photograph of ileal hypertrophy in an adult horse. There are also a few tapeworms present in the ileum. Hypertrophy of the muscular layer of the ileum is of unknown etiology. Initially affected horses have a history of recurrent colic.

Ascarid Impaction

Figure 1.100 *Parascaris equorum*. Impaction with this parasite is usually seen in weanlings and yearlings and is caused by complete lumenal obstruction by *Parascaris equorum*. Anthelmintics that cause sudden paralysis of the ascarid worms are implicated. Affected horses usually show signs of colic within 5 days of anthelmintic administration.

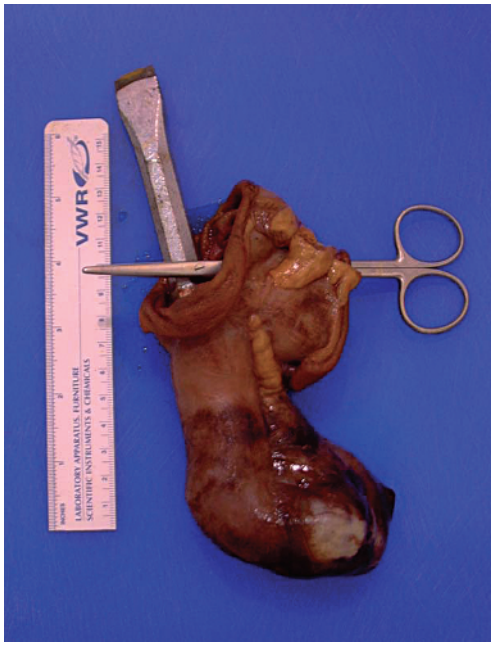


Figure 1.101 Postmortem photograph of Meckel's diverticulum. It is an embryonic remnant that can be found in the mid-jejunum area. It can become impacted and cause recurrent colic or serve as a point where intestines could twist.

Strangulating Obstruction

It is manifested by obstruction of both the intestinal lumen and blood supply. Strangulating obstruction is usually associated with severe abdominal pain, nasogastric reflux, and abnormal peritoneal fluid (serosanguinous).

Mesodiverticular Band

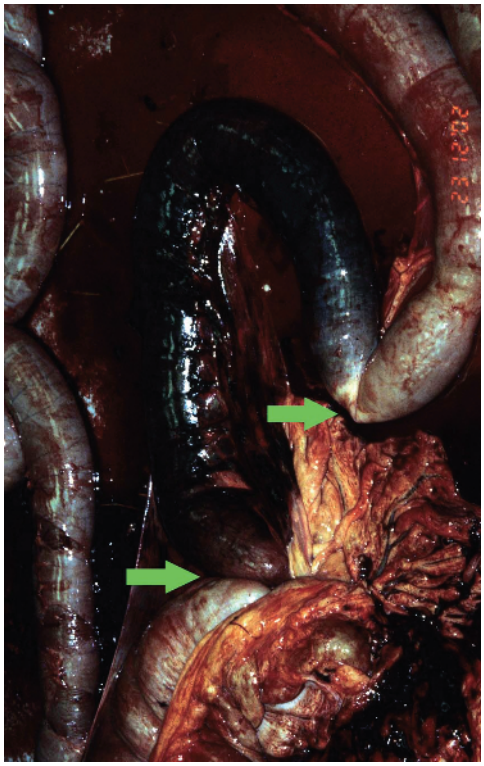


Figure 1.102 Postmortem photograph of a small loop of the small intestine that was strangulated by a mesodiverticular band. The arrows point at the area where the band was located. Mesodiverticular band is an embryonic remnant that extends from *Meckel's* diverticulum to the umbilical remnant or from the embryonic ventral mesentery to the antimesenteric surface of the small intestine. The small intestine can be strangulated by the mesodiverticular band and this is usually associated with severe colic signs.

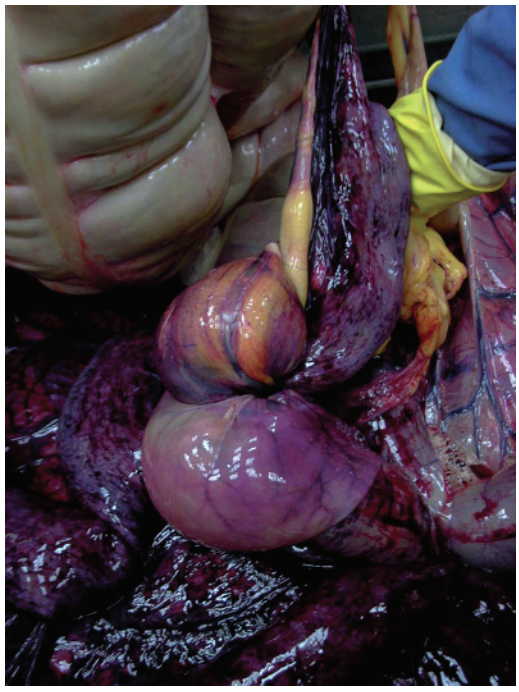
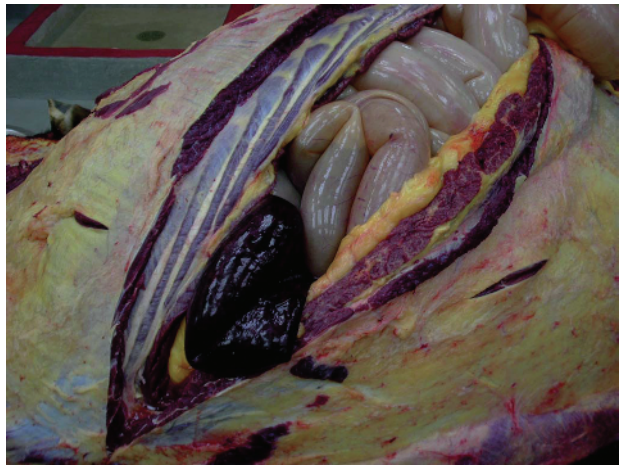
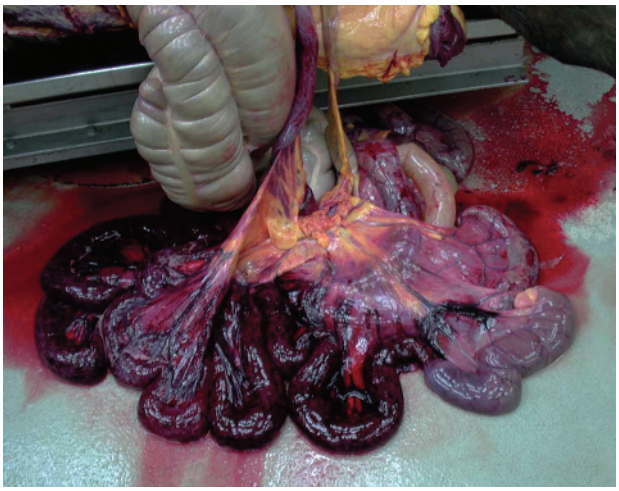


Figure 1.103 Postmortem photograph of mesenteric torsion in an adult horse. It can be partial or complete (involving all the small intestine). It appears to be more common in foals than adult horses. Mesenteric torsion causes severe colic signs and is one of the most serious causes of colic in horses. Affected horses are usually unresponsive to sedatives.

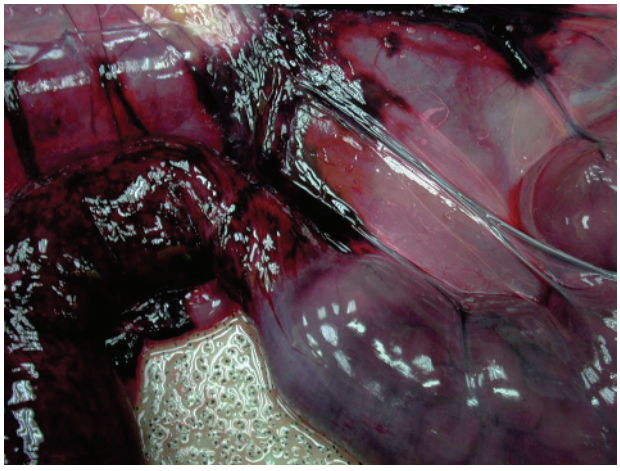


a

Figures 1.104a–c Postmortem photographs of partial mesenteric torsion in an adult horse. Note that not all the small intestines are involved. Also note the sharp demarcation between normal and abnormal small intestines.



b

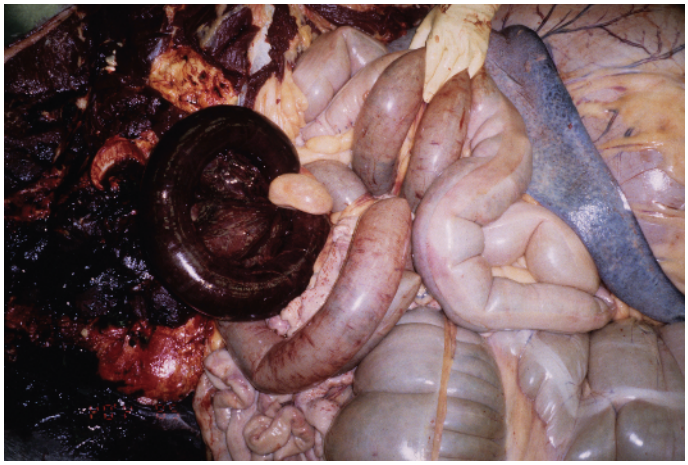


c

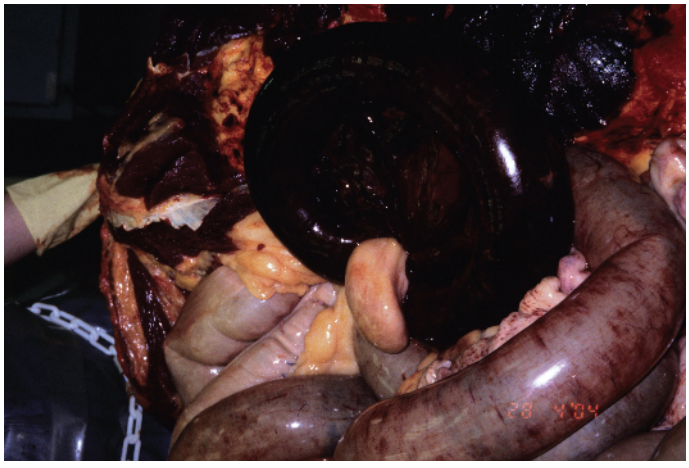
Figures 1.104a–c *Continued*

Figure 1.105 Peritoneal fluid from a horse affected with mesenteric torsion. Note the red color and the foam on the top of the fluid. The red color is mainly due to the presence of red blood cells while the foam is due to high protein content.

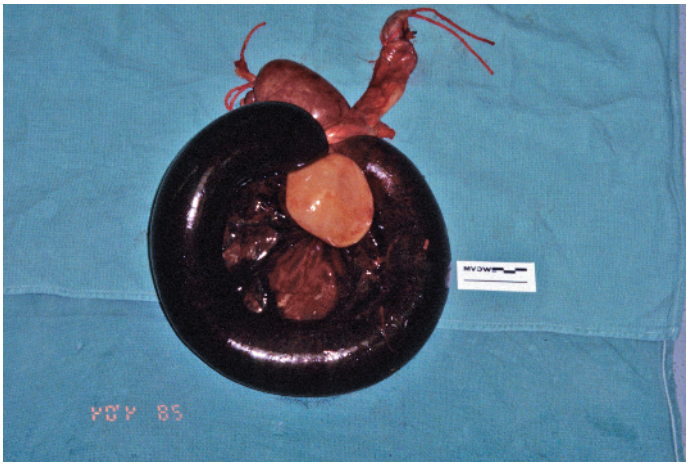
Small Intestinal Strangulation Caused by a Pedunculated Lipoma



a

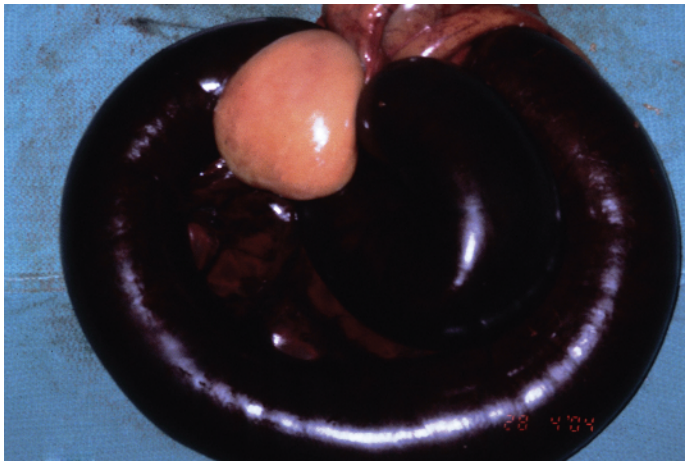


b



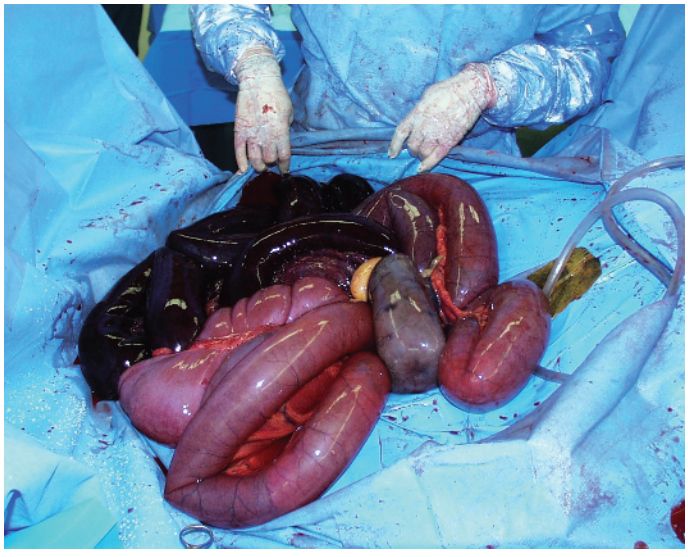
c

Figures 1.106a–e Figures a to d are postmortem photographs and e is an intraoperative photograph of small intestinal strangulation caused by pedunculated lipoma. Lipoma is a benign tumor in the horse and is usually spherical in shape (fig. 1.107). Pony horses and geldings are more predisposed to the disease. Also it is a disease of the older horse. It is usually located on the mesentery and it may or may not have a long stalk (fig. 1.108). Lipoma with a long stalk can strangle loop(s) of the small intestines (fig. 1.106 and 1.109) or serve as a point where intestines may rotate (fig. 1.110).



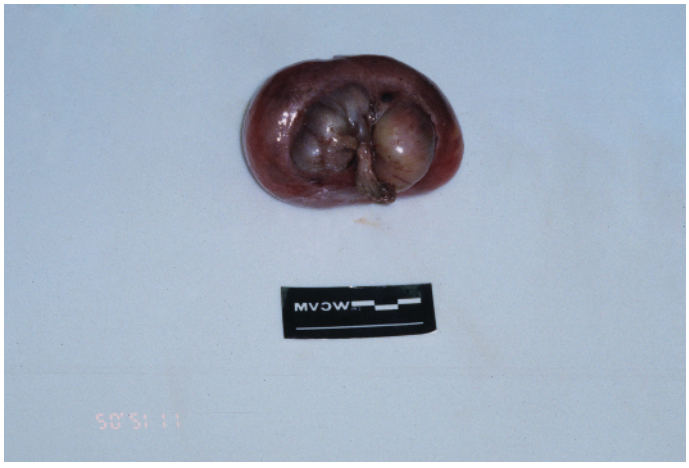
d

Figures 1.106a–e *Continued*



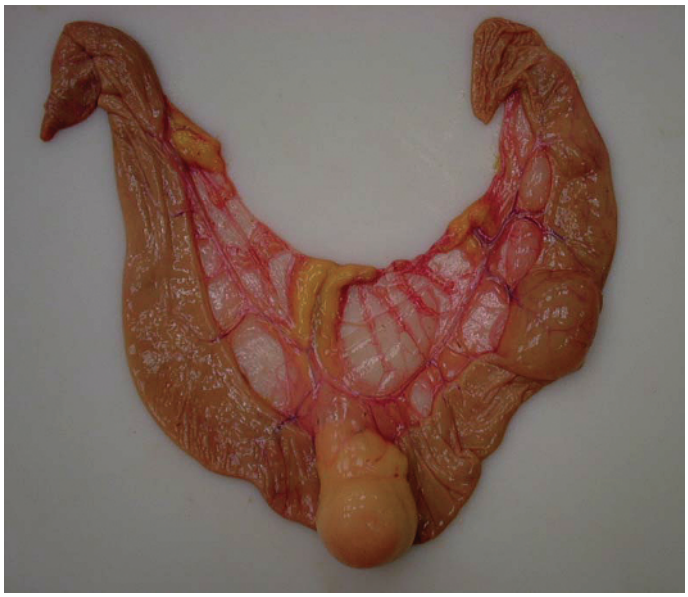
e

a

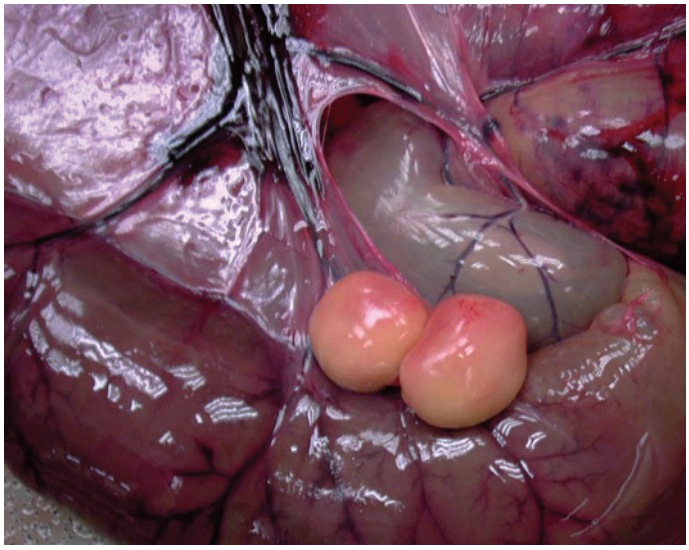


b

Figures 1.107a,b Postmortem photograph of a lipoma. It is a spherical benign tumor that is fatty in nature (cross section in fig. 1.107a).



a



b

Figures 1.108a,b Short-stalk lipoma. Lipoma is usually located on the mesentery and may be found as an incidental finding.

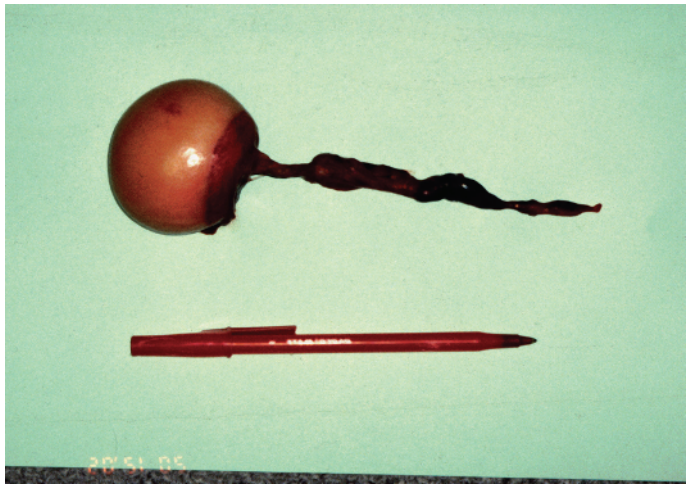


Figure 1.109 Long-stalk lipoma.

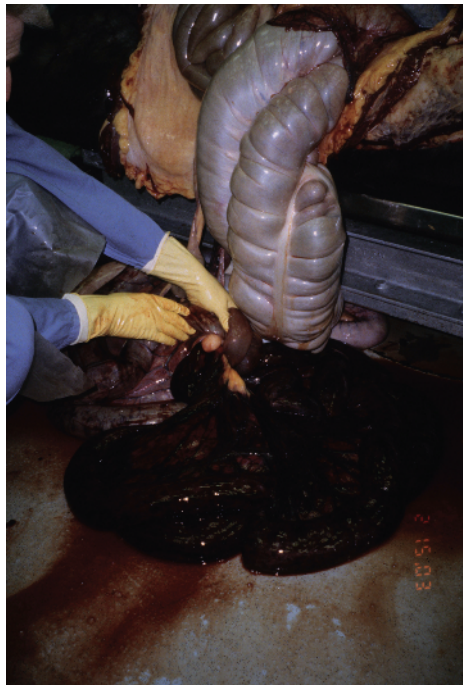


Figure 1.110 Postmortem photograph of a lipoma that served as a point where intestines rotated.

a

b

Figures 1.111a,b Postmortem photograph of epiploic foramen entrapment of the small intestines. Note the devitalized long loop of small intestines (fig. 1.111b). Strangulated small intestines are usually present in the cranial part of the abdomen (fig. 1.111a). It used to be considered a disease of the old horse only, but this assumption is no longer valid. Small intestines are the part of the bowel that is usually entrapped, but the large bowel can get entrapped too.

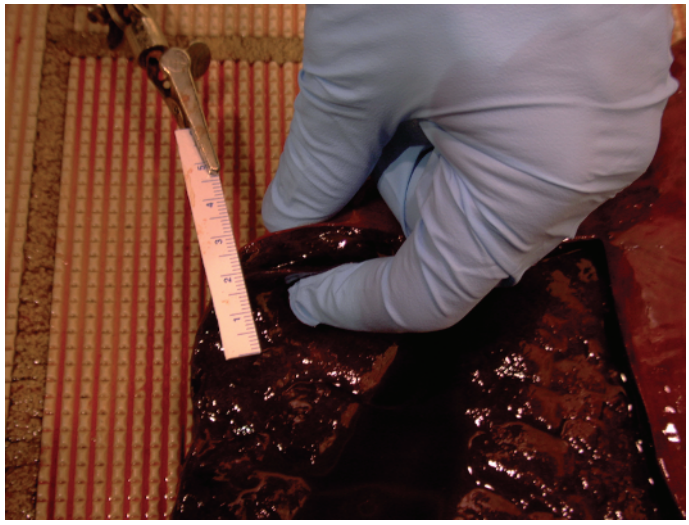
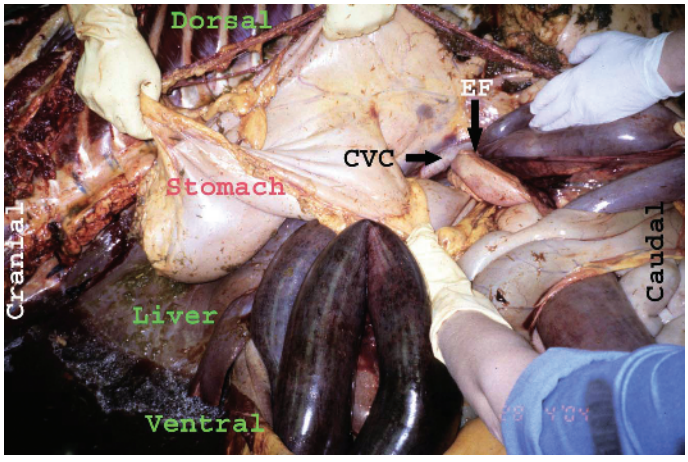
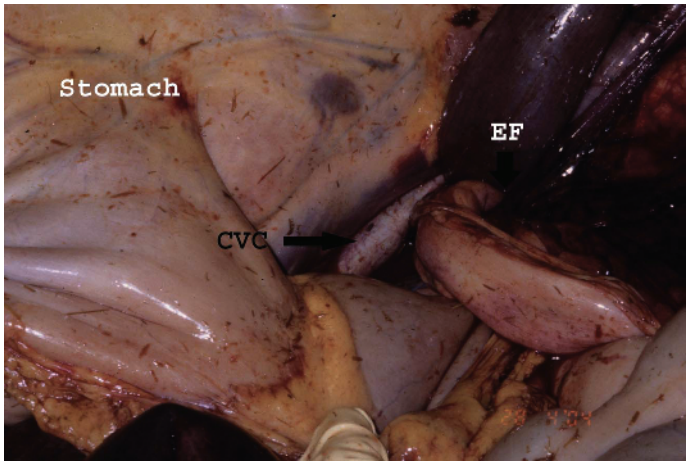


Figure 1.112 Postmortem photograph of epiploic foramen entrapment of the small intestines. Note the thickened small intestines.

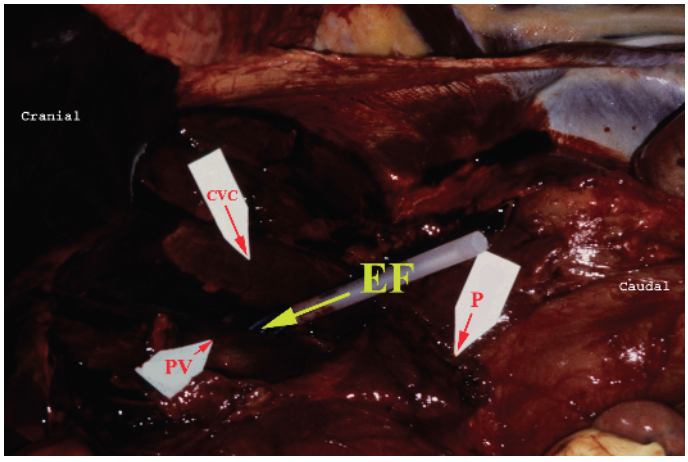


a



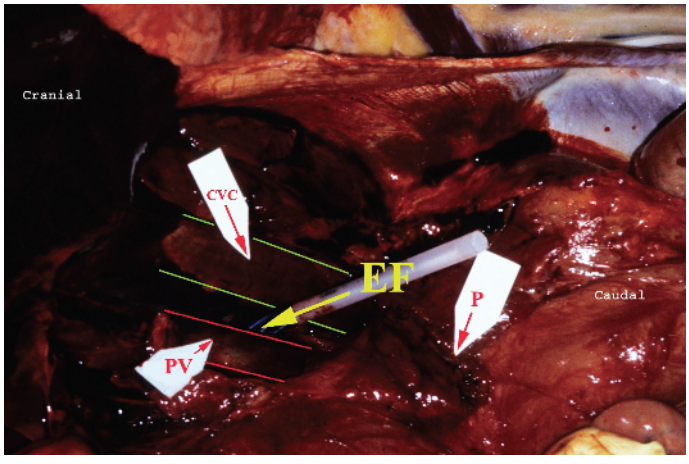
b

Figures 1.113a,b Postmortem photograph of epiploic foramen entrapment (EF) of the small intestines. Epiploic foramen is a natural opening in the abdomen to the omental bursa. It is bounded dorsally by the caudal vena cava (CVC) and caudate process of the liver, and ventrally by the portal vein and pancreas (figs. 1.114a,b). Fig. 1.113b is a close-up view of fig. 1.113a.



a

Figures 1.114a,b Postmortem photograph of a normal abdomen in a horse showing the normal boundaries of epiploic foramen (EF). Caudal vena cava (CVC), portal vein (PV), and pancreas (P). Fig. 1.114a and b are the same, but in fig. 1.114b, CVC and PV are outlined.



b

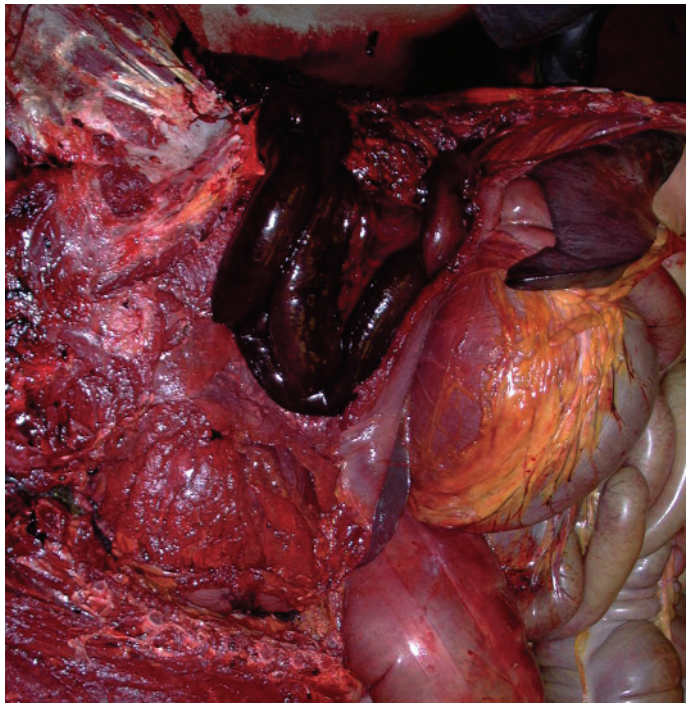


Figure 1.115 Postmortem photograph of diaphragmatic hernia and small intestinal strangulation. Note the presence of small intestines cranial to the diaphragm and in the thoracic cavity. It can be congenital or acquired and can be seen in all ages. Congenital diaphragmatic hernia is caused by incomplete fusion of the embryonic component of the diaphragm. Also, foals can develop diaphragmatic hernia because of the abdominal compression during birth. Acquired diaphragmatic hernia is assumed to be caused by trauma.

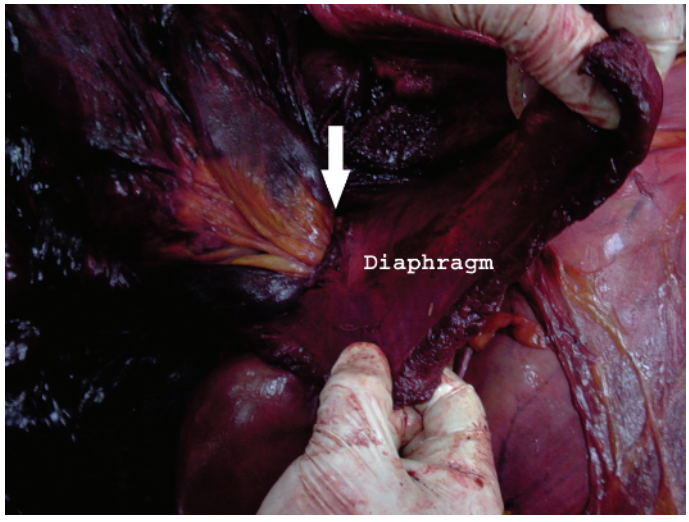
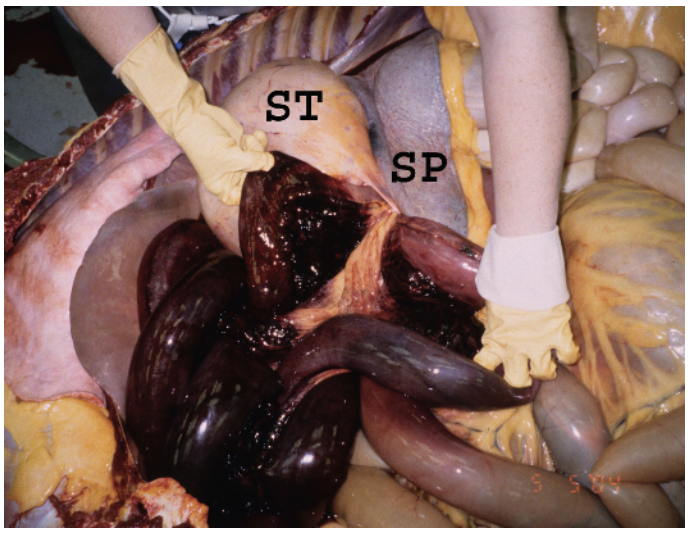


Figure 1.116 Point of herniation (arrow) in the diaphragm of the horse in fig. 1.115.



a

b

Figures 1.117a,b Postmortem photograph of incarceration of the small intestine through the gastrosplenic ligament. There is a congenital or traumatic rent in the gastrosplenic ligament through which the small intestine gets strangulated (fig. 1.118). Gastrosplenic ligament is located between the greater curvature of the stomach (ST) and spleen (SP). Fig. 1.117b is a close-up view of fig. 1.117a.

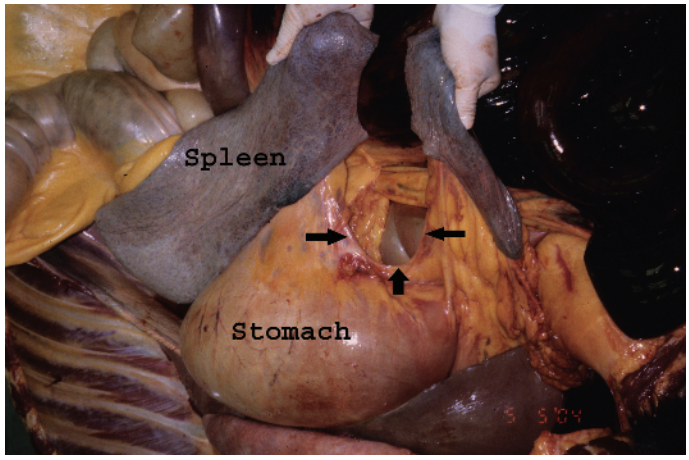


Figure 1.118 Postmortem photograph of a rent in the gastrosplenic ligament (arrows) through which the small intestines have gotten strangulated (fig. 1.117).

Intussusception

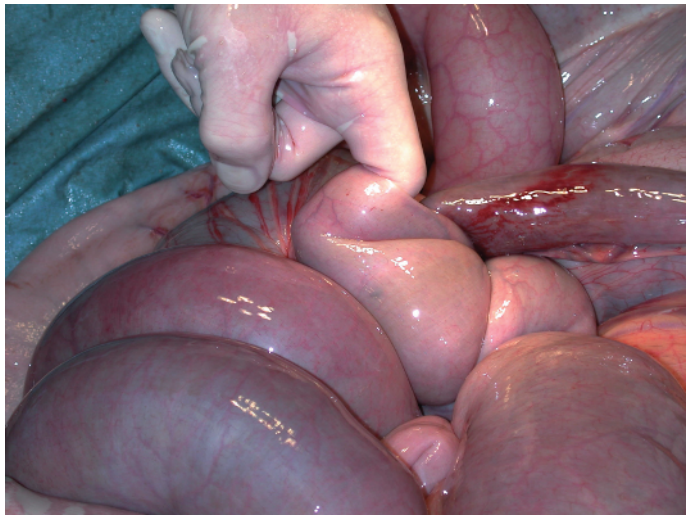


Figure 1.119 A photograph of small intestinal intussusception that was taken during exploratory laparotomy in an adult horse. Intussusception is seen more commonly in young horses. It is an invagination of a segment of the intestine into the adjacent segment. Tapeworms have been implicated to predispose the disease. Ileocecal intussusception is the most common.

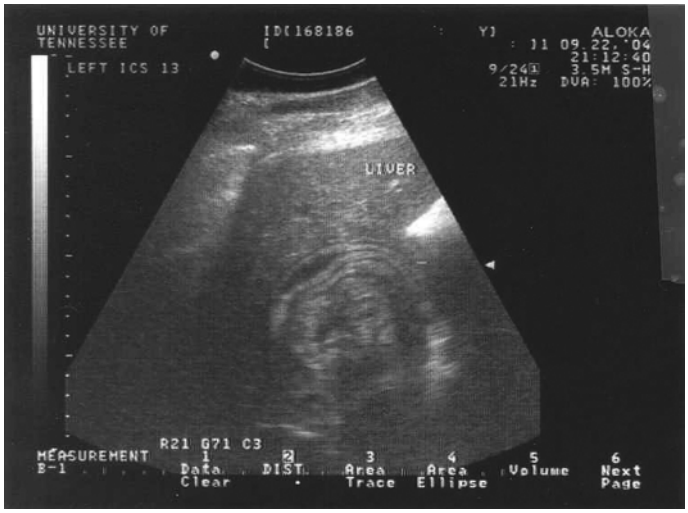


Figure 1.120 Ultrasonographic image of the right 13 intercostal space showing a classic target lesion of the duodenum in a horse presenting for chronic colic and weight loss. Target lesion (bull's-eye) seen on ultrasonography is diagnostic for the presence of intussusception.

Functional Obstruction of the Small Intestine

It is manifested by various degrees of abdominal pain and nasogastric reflux.

Duodenitis-Proximal Jejunitis (DPJ) (Anterior or Proximal Enteritis)

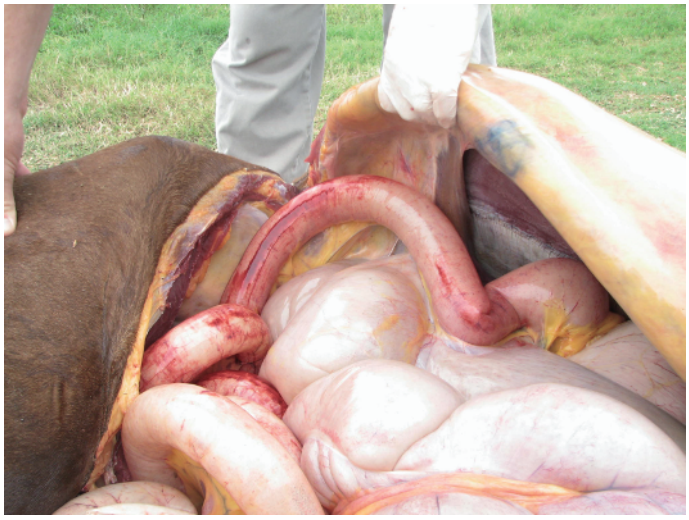


Figure 1.121a Postmortem photograph of duodenitis-proximal jejunitis (DPJ). Note the inflamed duodenum and distended jejunum. DPJ is a relatively new syndrome of unknown etiology. It is characterized by fever, abdominal discomfort, large volumes of nasogastric reflux (brownish orange with fetid odor), depression, and dehydration. Transrectal examination reveals distended loops of the small intestine. These signs are caused by a severe inflammation and edema of the duodenum and part of the jejunum. It is extremely important to differentiate DPJ from cases of small intestinal strangulating obstruction. The major difference is that in DPJ cases, colic signs are replaced by severe depression after the gastric decompression; this is not the case usually in strangulating lesions of the small intestines.

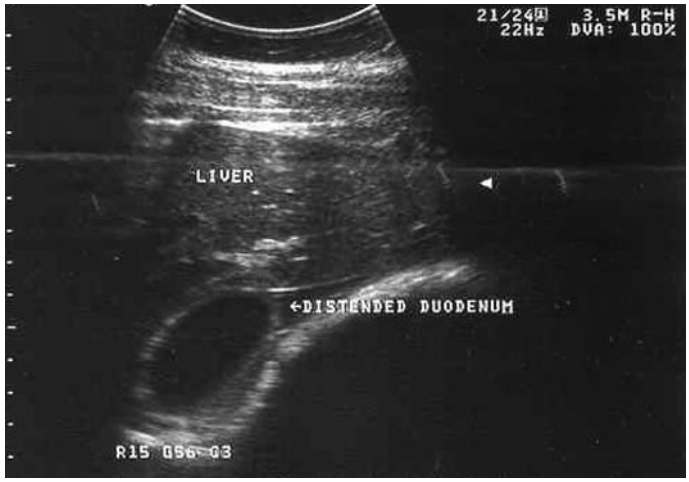


Figure 1.121b Ultrasonographic image of the right caudal abdomen showing distension and thickening of the duodenum (dorsal is to the left) in a case of duodenitis-proximal jejunitis.

Proliferative Enteropathy (*Lawsonia intracellularis*)

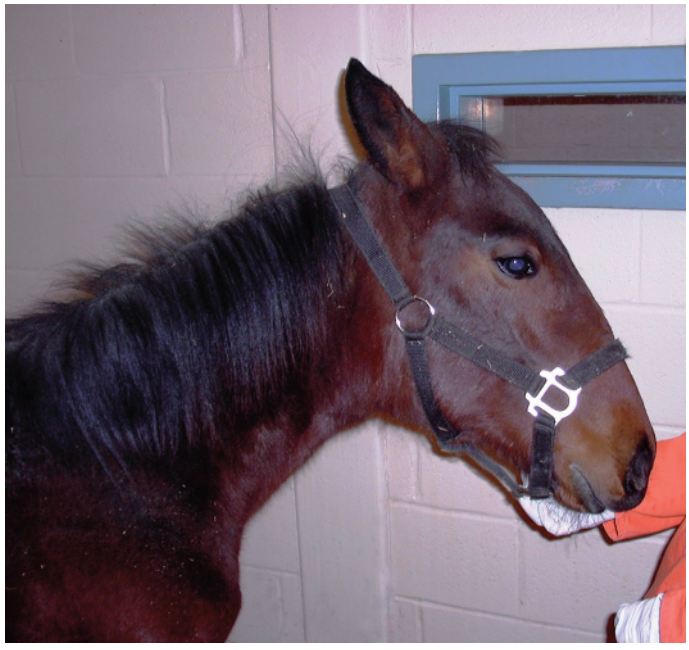


Figure 1.122a A foal affected with proliferative enteropathy. Note the intermandibular edema. Proliferative enteropathy is associated with *Lawsonia intracellularis*. Affected foals are 3 to 13 months old. Clinical signs include depression, weight loss, colic, diarrhea, and sometimes death in 2 to 3 days (fig. 1.122b). Affected horses are hypoproteinemic and usually develop ventral and intermandibular edema (fig. 1.122c). Thickening and irregular corrugation of the small intestines is usually seen on postmortem examination (fig. 1.122d).

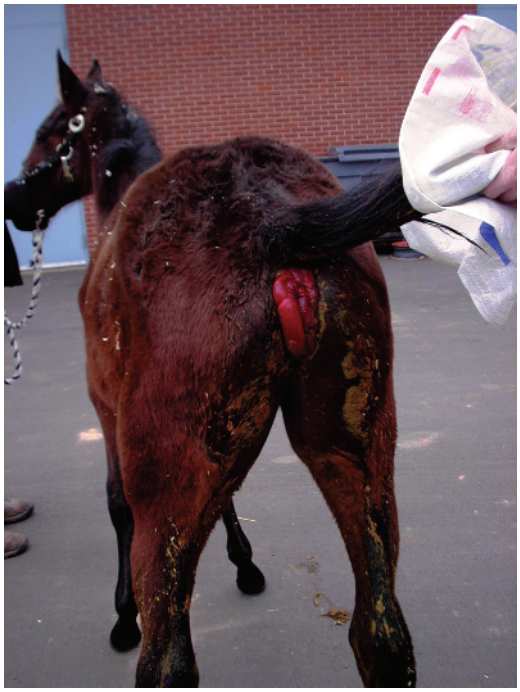


Figure 1.122b A foal affected with proliferative enteropathy. Note the diarrhea. In addition, the foal had a rectal prolapse.

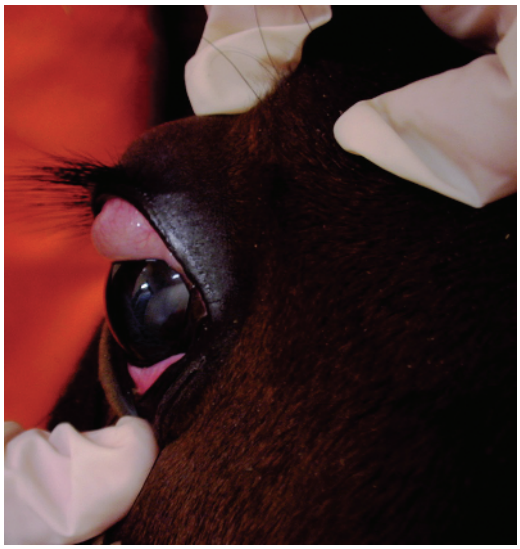


Figure 1.122c A foal affected with proliferative enteropathy. Note the chemosis that had developed secondary to severe hypoproteinemia.

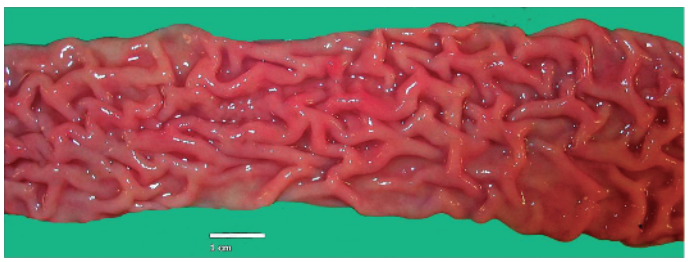


Figure 1.122d A postmortem photograph of the intestine of a foal affected with proliferative enteropathy. Note the thickened and irregular corrugation of the small intestines.

Diseases of the Large Intestine

Large Colon Volvulus (LCV)

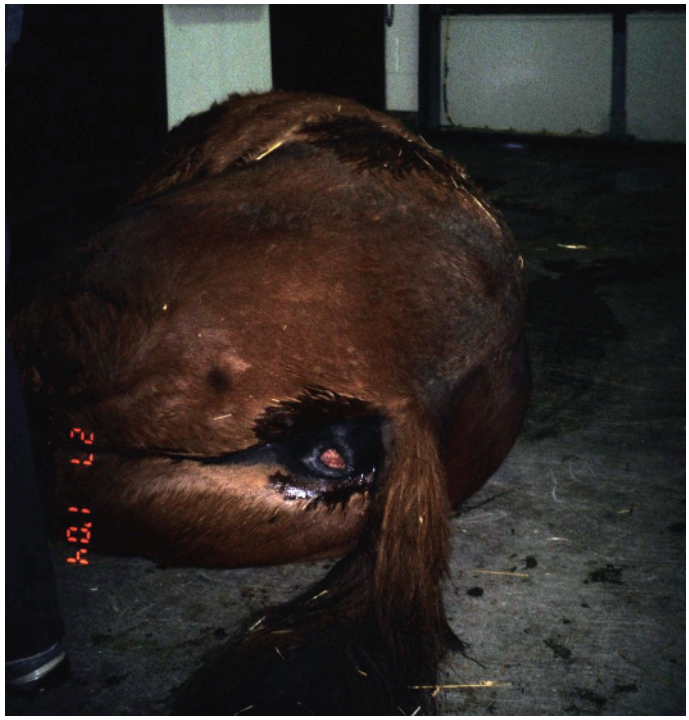


Figure 1.123 A horse with large colon volvulus. Note the abdominal distention that progresses very fast. Large colon volvulus is serious and often fatal. Horses with LCV usually have an acute onset of severe, unrelenting, abdominal pain, high heart rate, abdominal distention and are in a state of shock. Postpartum mares may be overrepresented. Diagnosis is usually based on clinical signs, severely distended large colon on transrectal examination and, recently, by using ultrasonography.

Figure 1.124 Postmortem photograph of large colon volvulus in a horse. Note the devitalized large colon and bloody intestinal content.

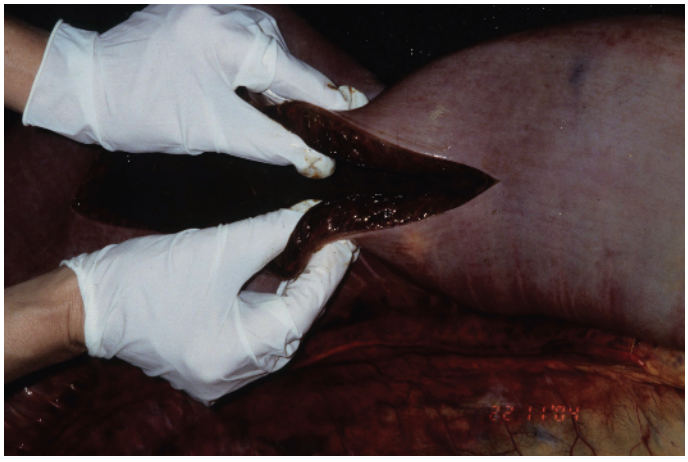


Figure 1.125 Postmortem photograph of large colon volvulus in a horse. Note the severely thickened large colon.

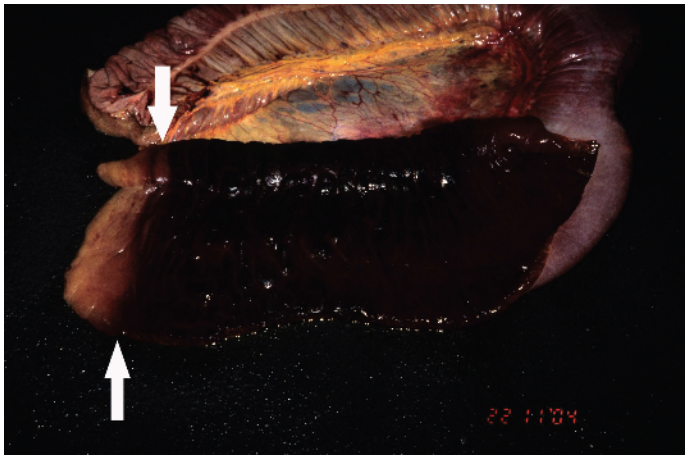


Figure 1.126 Postmortem photograph of large colon volvulus in a horse. Note the sharp demarcation at the site of rotation between the strangulated and nonstrangulated parts of the large colon (arrows).

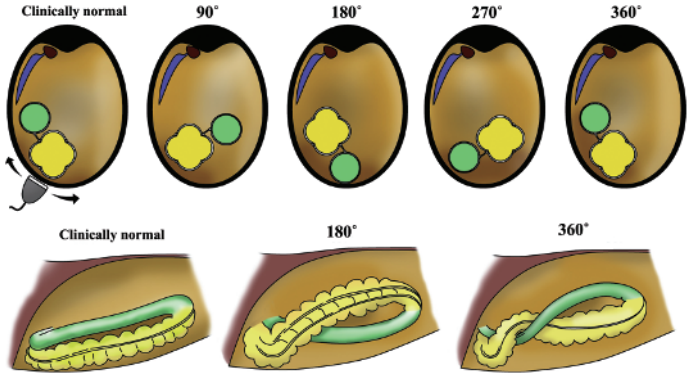


Figure 1.127 Cross and sagittal section diagram of the location of the left large colon in normal horses and horses with different degrees of large colon volvulus. There are different degrees of rotation of the large colon (80°–720°), the higher the degree the more severe the cases. There are two levels at which the LCV usually occurs – at the base of the cecum or at the level of the diaphragmatic and sternal flexures. (Used with permission from Abutarbush SM, Use of ultrasonography to diagnose large colon volvulus in horses, Journal of the American Veterinary Medical Association, 2006; 228[3] 409–13)

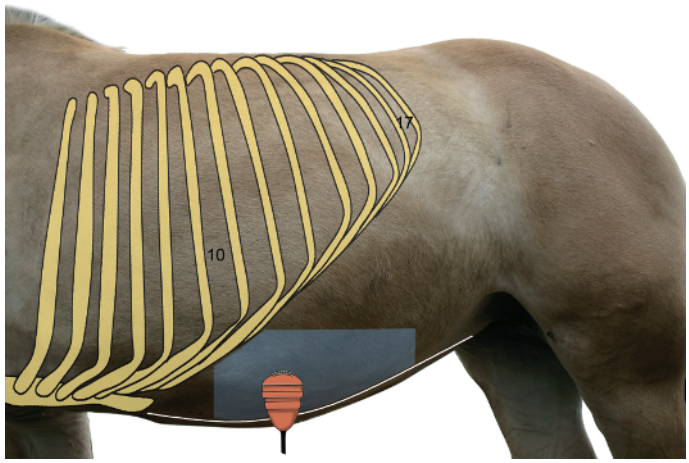
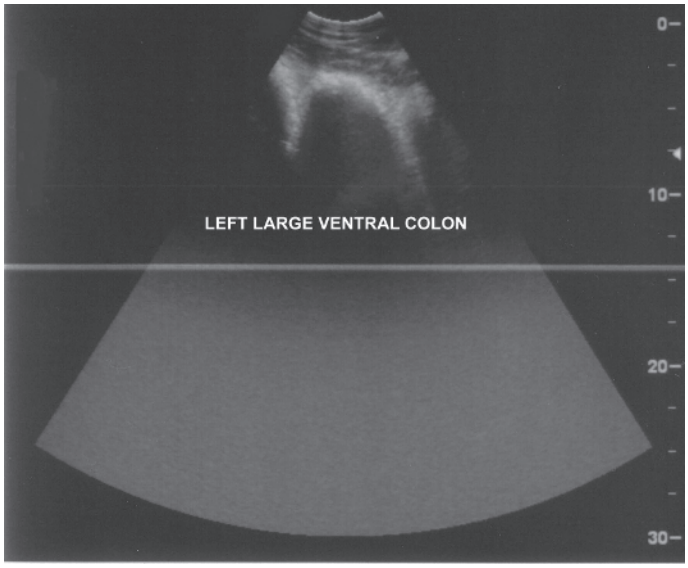
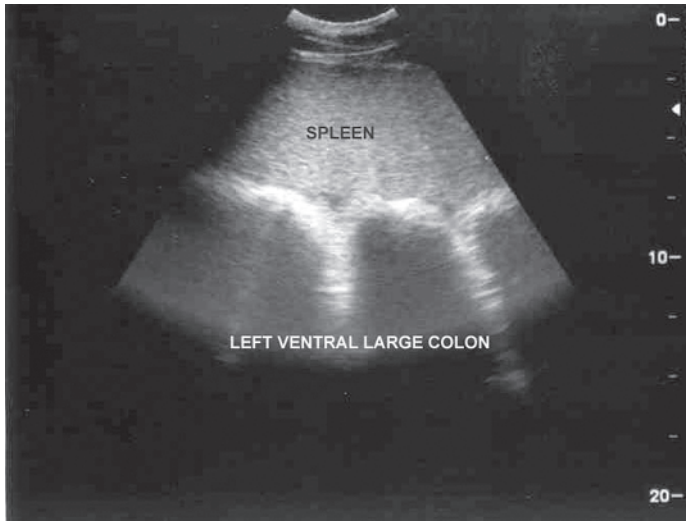


Figure 1.128 Ultrasonography can be used to diagnose LCV. This figure illustrates the landmarks that are used to ultrasound the left ventral colon (shaded area), which is used to diagnose large colon volvulus in horses based on the anatomical features of the left ventral and dorsal colon. The boundaries of the shaded area are midline, two vertical lines at the level of the 10th and 17th intercostal spaces, and a horizontal line between the two vertical lines at the level of the costal arch of the 10th intercostal area. (Used with permission from Abutarbush SM, Use of ultrasonography to diagnose large colon volvulus in horses, Journal of the American Veterinary Medical Association, 2006; 228[3] 409–13)

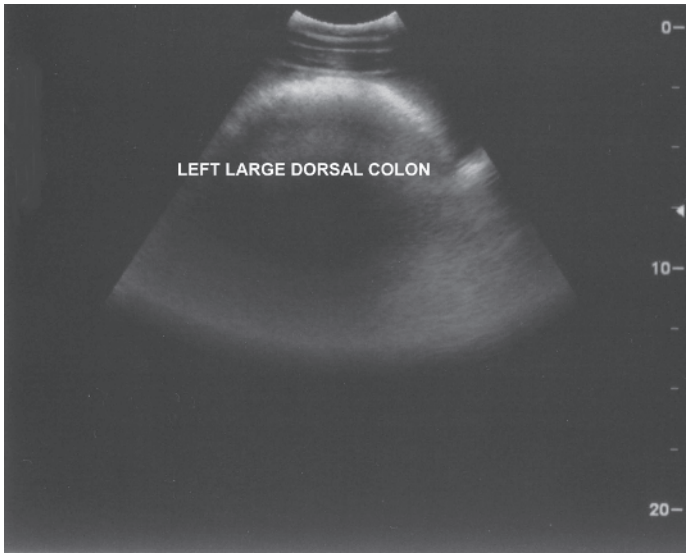


a

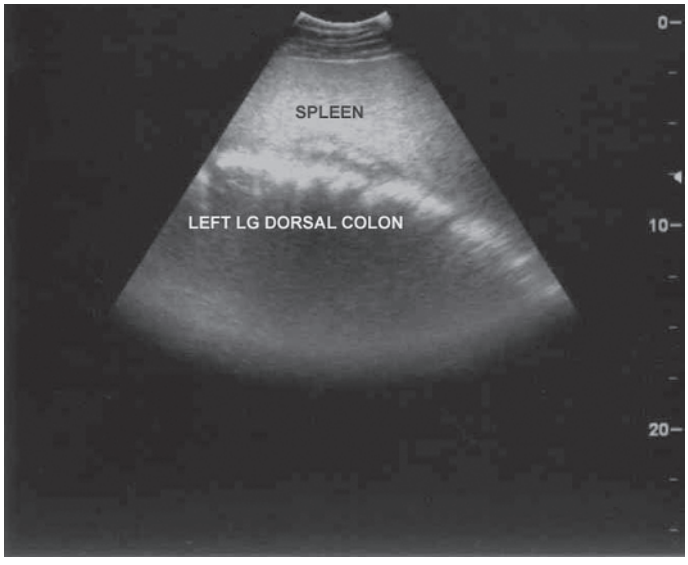


b

Figures 1.129a,b Ultrasonographic images of the abdomen in a normal horse as outlined in fig. 1.128. The large ventral colon is sacculated, while the dorsal is not. In normal horses, ultrasonography of the left ventral abdomen reveals the presence of the sacculated left large ventral colon next to the body wall (a) and sometimes close to the spleen (b). (Images used with permission from Abutarbush SM, Use of ultrasonography to diagnose large colon volvulus in horses, Journal of the American Veterinary Medical Association, 2006; 228[3] 409–13)



a



b

Figures 1.130a,b Ultrasonographic images of the left ventral abdomen in a horse with a 540° large colon volvulus as outlined in fig. 1.127. Note the abnormal ventral location of the left dorsal large colon (no sacculation) next to the body wall (a) or the spleen and body wall (b). The principle of using ultrasonography to diagnose LCV is to identify the left dorsal colon in a ventral position. The presence of the nonsacculated large colon, when ultrasounding the left ventral abdomen, is indicative of a LCV, which means that the dorsal left colon is in a ventral position. LCV with a rotation of 360° or 720° will not be diagnosed using this method because the ventral colon will be in a ventral location (fig. 1.127). (Images used with permission from Abutarbush SM, Use of ultrasonography to diagnose large colon volvulus in horses, Journal of the American Veterinary Medical Association, 2006; 228[3] 409–13)

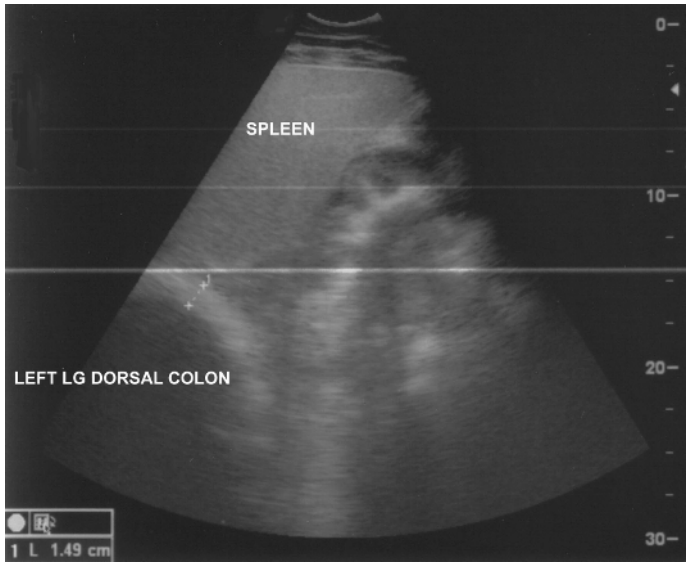
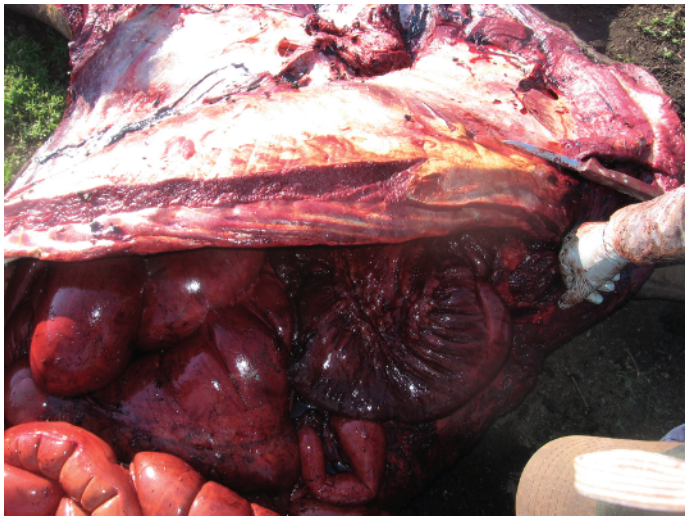


Figure 1.131 Ultrasonographic image of the left ventral abdomen in a horse with large colon volvulus. Note the thickened large colon that can be seen in cases of LCV.

Large Colon Displacement (LCD)

Not an uncommon cause of abdominal pain in horses. There are two main classifications for LCD: left LCD and right LCD.

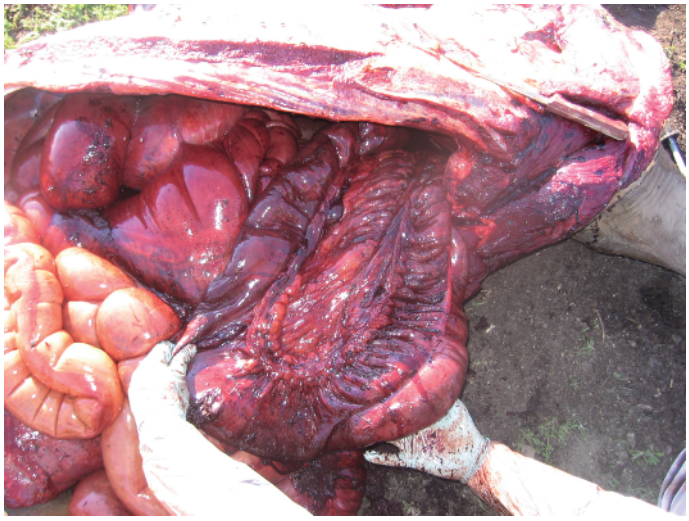
Right Displacement of the (Left) Large Colon (RDLC)



1.132

Figures 1.132–1.133 Postmortem photograph of a horse with right displacement of the large colon. The horse is on lateral position with the left side down and the right side up. Note the presence of the pelvic flexure (the connection between the left dorsal and ventral large colon) in the right side of the abdomen lateral to the cecum fig. 1.133. With RDLC, the colon displaces to the right of the cecum (lateral).

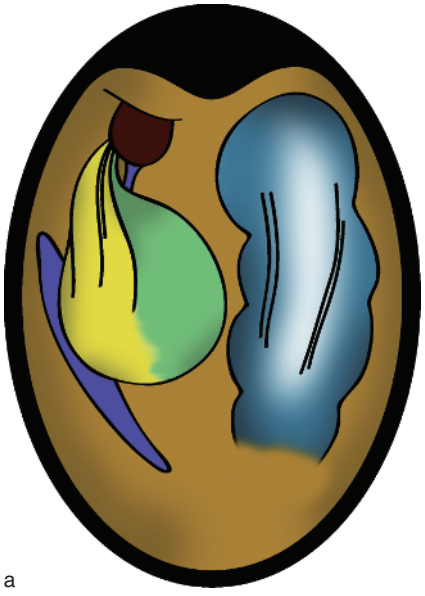
Figures 1.132–1.133 *Continued*



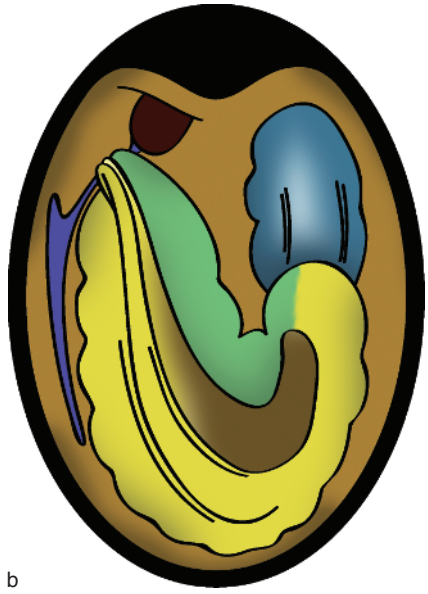
1.133

Left Dorsal Displacement of the Large Colon (LDLC)

Figures 1.134a,b Cross-section diagrams of the abdomen of a horse with early (a) and advanced (b) left dorsal displacement of the large colon (nephrosplenic entrapment). The left large colon can be entrapped between the left kidney and spleen (nephrosplenic entrapment) or between the spleen and the left body wall; both are referred to as left dorsal displacement of the large colon. Nephrosplenic entrapment occurs over a wide age range, 8 months to 16 years, and is more frequently observed in middle-aged horses. Geldings are more frequently affected, but displacement can occur in any gender. Large-framed or large horses are at higher risk of developing NSELC. The cause is unknown. Diagnosis of LCD can be made by transrectal palpation, percutaneous ultrasonography of the upper left flank (nephrosplenic entrapment), or exploratory laparotomy. (Drawings by Dr. Juliane Deubner, WCVM, University of Saskatchewan)

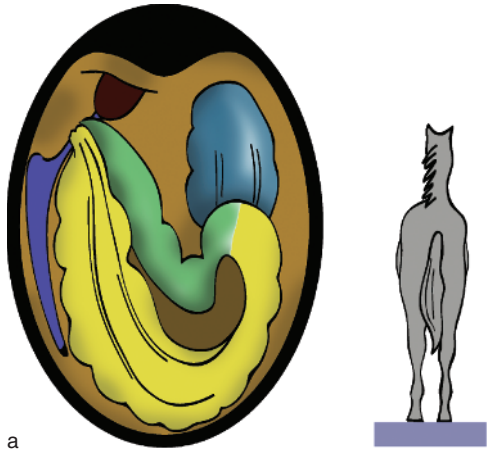


a

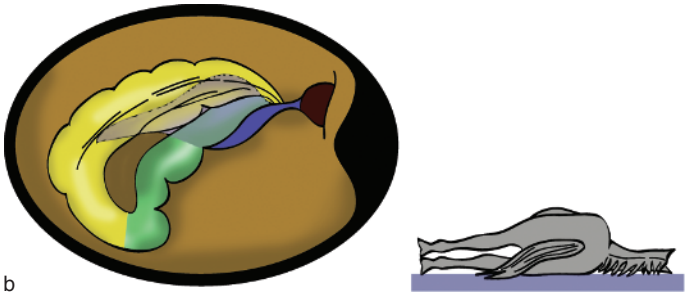


b

Figures 1.134a,b *Continued*

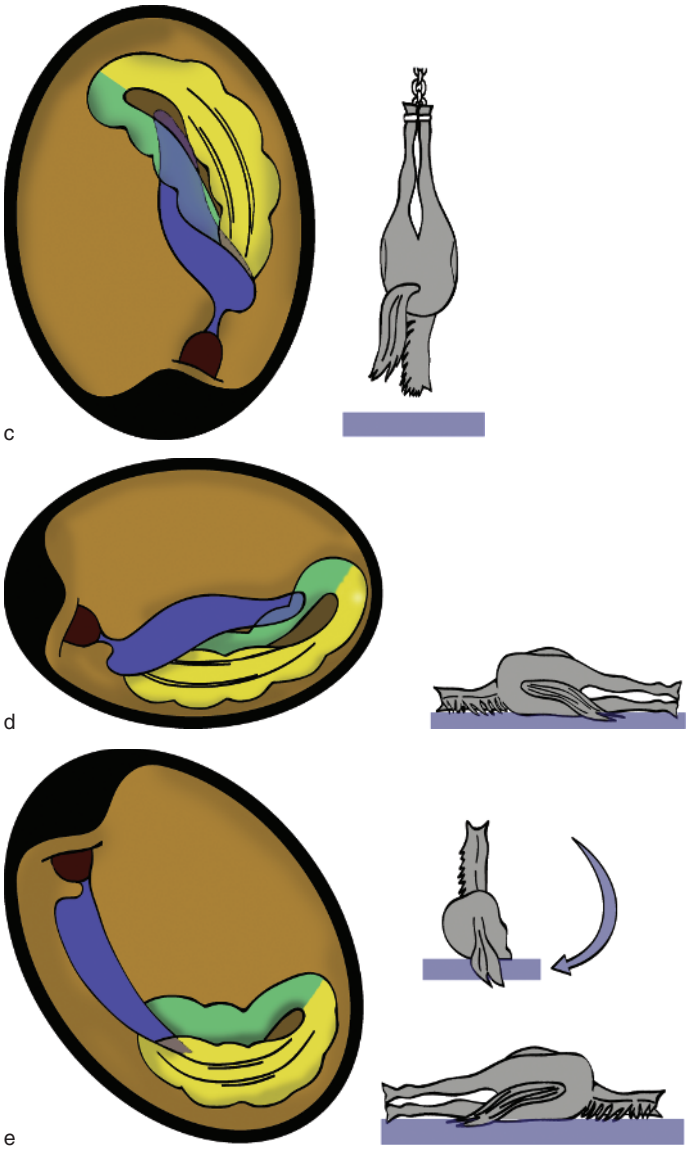


a



b

Figures 1.135a–e Cross-section diagrams of the abdomen of a horse with left dorsal displacement of the large colon (nephrosplenic entrapment) and its treatment by rolling. Treatment by rolling should be performed under general anesthesia. The horse is anesthetized and placed in right lateral recumbency (b), then rotated up to dorsal recumbency. The abdomen is rocked back and forth for a few minutes. Then the hind limbs are hooked to a chain hoist and the hind quarters are elevated off the ground (c). The horse is then rolled to the left lateral side and evaluated (d) or rolling is continued to the right lateral side and then evaluated (e). Correction is evaluated by both rectal examination and per cutaneous ultrasound of the left flank. (Drawings by Dr. Juliane Deubner, WCVM, University of Saskatchewan)



Figures 1.135a–e *Continued*

Large Colon Impaction (LCI)

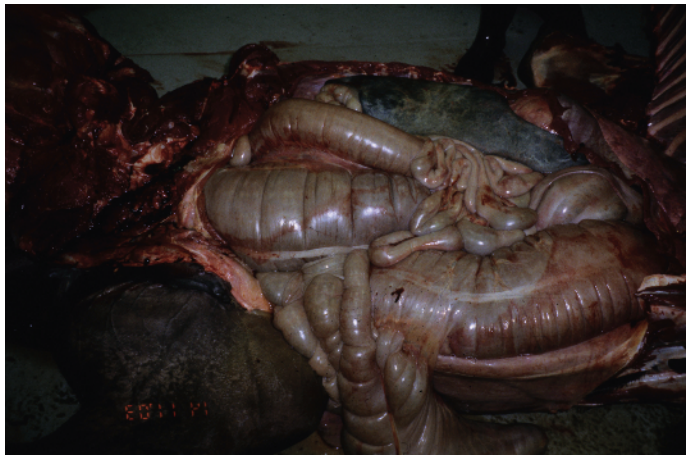


Figure 1.136a Postmortem photograph of a horse with large colon impaction (a). LCI could occur at any location in the large colon, but occurs commonly at areas of natural narrowing of the diameter of the lumen of the large colon, pelvic flexure, transverse colon. Risk factors include exercise restriction, large concentrate meals, and restricted access to water in cold climates (frozen water sources). Clinically it is manifested by an onset of mild pain and sometimes diarrhea. The production of watery fecal fluids followed by boluses of fibrous ingesta can be seen. If not treated, severe cases may result in colitis or rupture of the colon (figs. 1.136b,c). Doughy, ingesta-filled viscus may be felt rectally, however, it depends on the location of the impaction, whether it is reachable by hand or not. Large colon impaction should be treated with IV fluid therapy, analgesics, and oral laxatives.

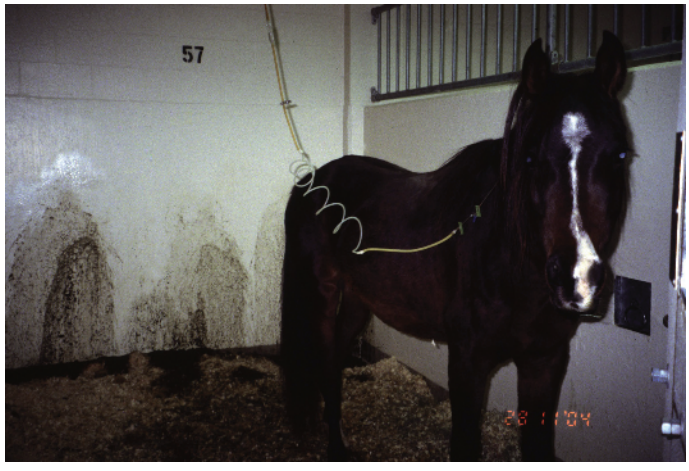


Figure 1.136b An adult horse with long-standing large colon impaction that has developed colitis and diarrhea.

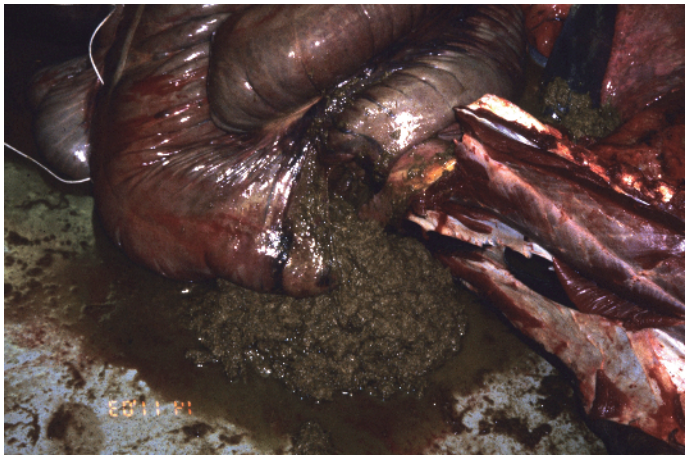


Figure 1.136c Postmortem photograph of a horse with long-standing large colon impaction. Note the ruptured colon.

Large Intestinal Intussusception

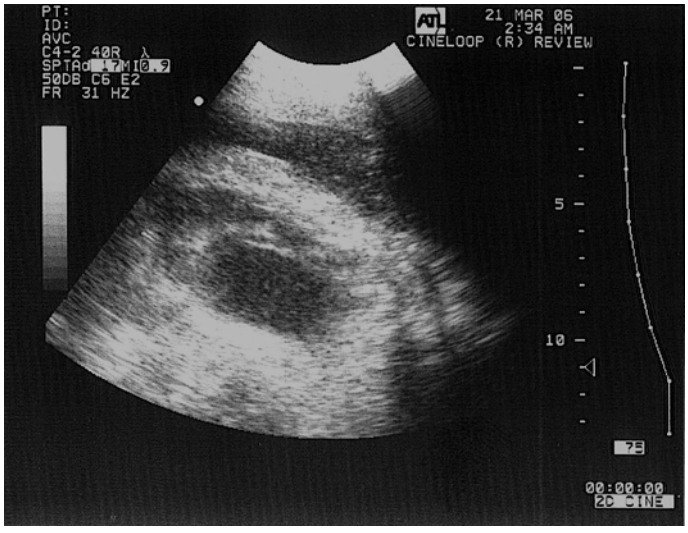
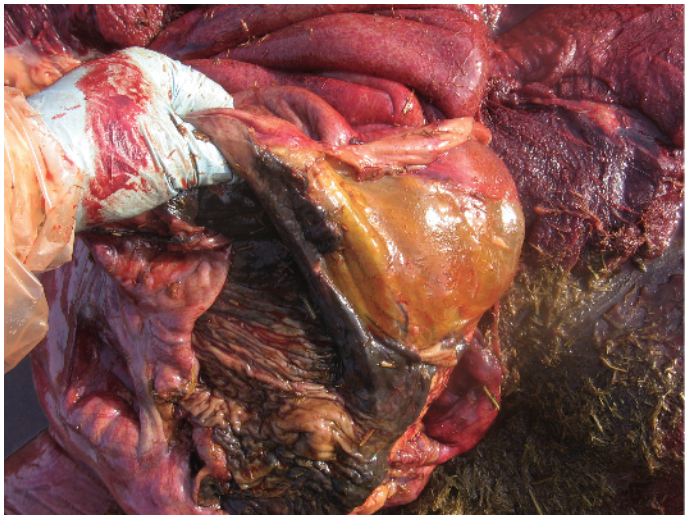


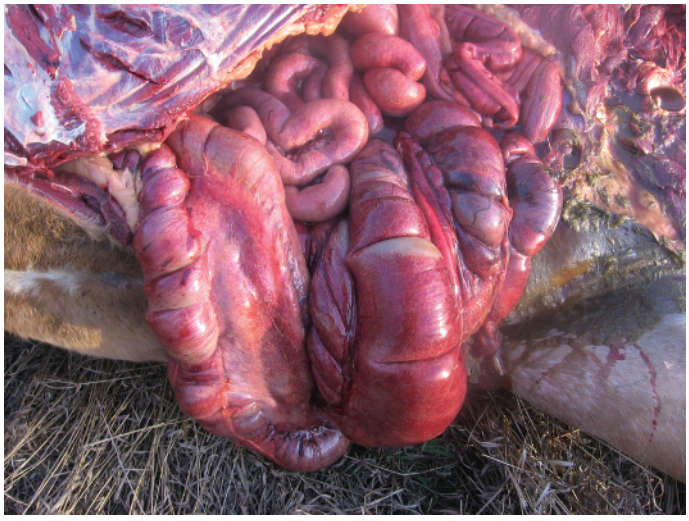
Figure 1.137 Ultrasonographic image of the right paralumbar fossa showing a classic target lesion (bull's-eye) of intussusception of the large intestine. It is an uncommon cause of colic that is usually seen in young horses (2–3 years old). It can occur at different locations in the large intestine and the most common one is the cecocolic intussusception. Clinical signs are usually associated with acute onset of abdominal pain that differs in the severity according to the severity of the intussusception. Intussuscept may be felt transrectally.

Salmonellosis

Figure 1.138 Horse affected with salmonellosis. Note the watery diarrhea. Salmonellosis is a serious disease and can be fatal. It is zoonotic and can cause serious sickness in humans. *S. typhimurium* and *agona* are common isolates, but other serotypes can cause the disease. *Salmonella* infection can be latent and horses may become carriers.



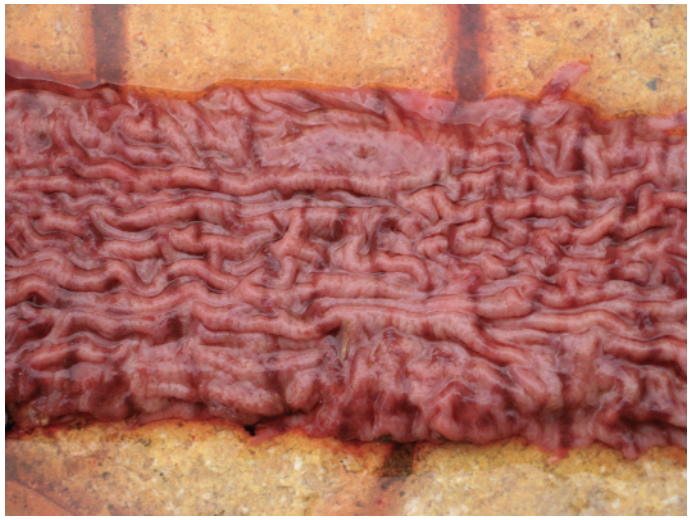
a



b

Figures 1.139a–d Postmortem photograph of a horse affected with acute salmonellosis. Note the severe enterocolitis. The large colon is edematous (a), thickened, and very inflamed (b, c). Small intestines can be affected also (d).

c



d

Figures 1.139a–d *Continued*

Figure 1.140 Horse affected with salmonellosis. Note the severe dehydration and skin tenting.

Figure 1.141 Horse affected with salmonellosis. Note the dark red and tacky gum. Affected horse can show signs of endotoxemia, as in this case.

Strongylosis

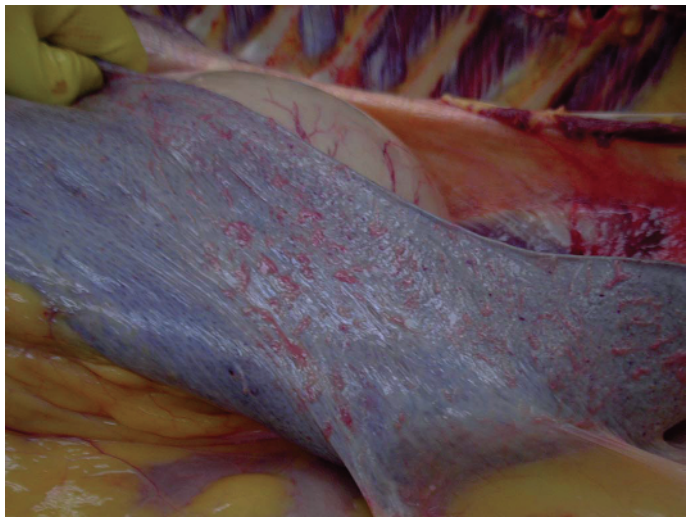


Figure 1.142 Postmortem photograph of a horse. Note the presence of fibrous tags on the spleen, which is indicative of parasitic migration, mainly due to *Strongylus* spp. Strongylosis is caused by *Strongylus vulgaris, Strongylus edentatus, Strongylus equinuus. S. vulgaris* by far is the most important. Diarrhea is caused by larval migration through the intestinal wall causing inflammation and abnormal intestinal motility and function. Fibrous tags, as in this figure, on the abdominal organs and heamomalasma ilii (fig. 1.143) are found in necropsy as evidence of larval migration. It is more commonly seen in young and naive horses. Clinical signs include fever, depression, poor weight gain, intermittent mild colic and diarrhea. Diagnosis is based on clinical signs, elevated alpha- and beta-globulin, and IgG(T). Fecal analysis might be unrewarding.

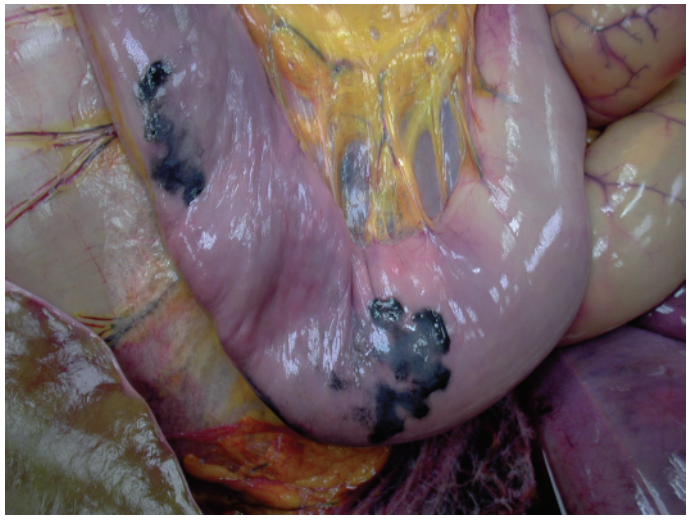


Figure 1.143 Postmortem photograph of the small intestine of a horse. Note the heamomalasma ilii. It can be found in necropsy and is an evidence of larval migration.

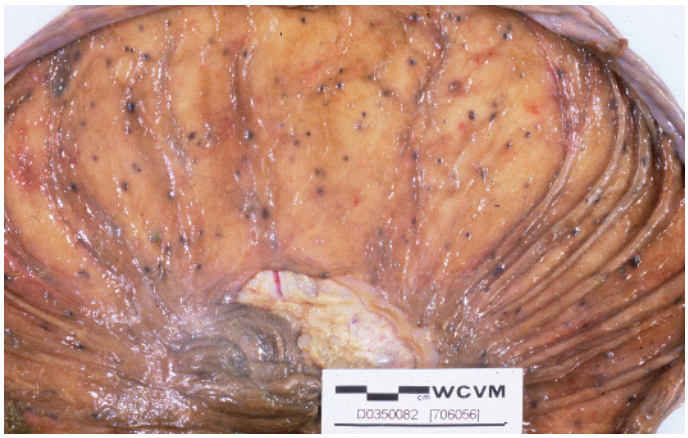
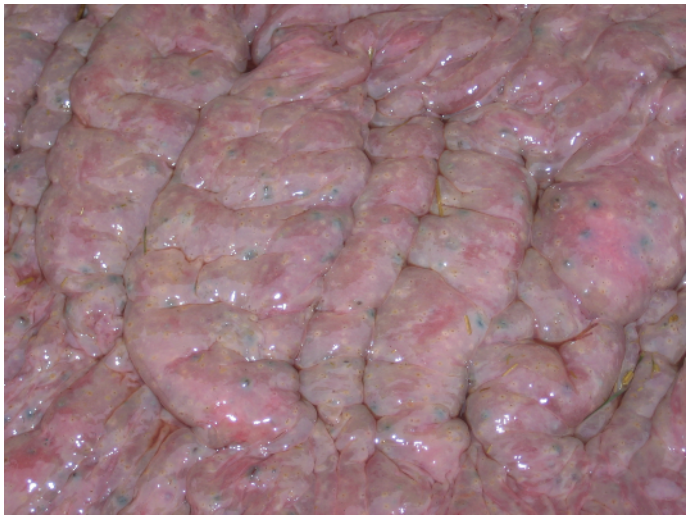
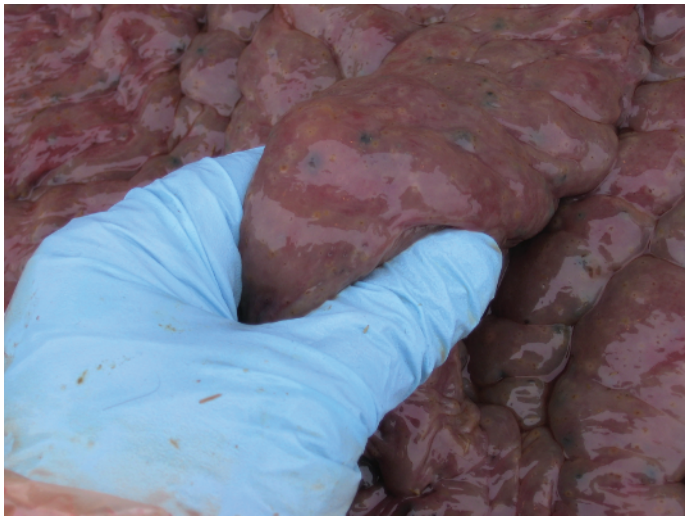


Figure 1.144 Postmortem photograph of the cecum of a horse. Note the presence of black dots on the mucosa, which are encysted small strongyles larvae (cyathostomes). This can be found incidentally on postmortem examination, as in this case. Cyathostomiasis is caused by small strongyles (cyathostomes) and typhlocolitis is usually precipitated by intramural larval stages, and sudden emergence of the encysted larvae triggers severe mucosal inflammation (figs. 1.145 and 1.146). In the northern temperate zones, it is usually seen in the late winter or early spring; in the southern temperate zones, it occurs in the fall or winter. Cyathostomiasis is usually associated with chronic diarrhea, but can cause severe acute diarrhea that becomes chronic. In addition to diarrhea, affected horses exhibit ill thrift and have a fever, weight loss, ventral edema, and intermittent mild episodes of abdominal pain. Appetite is usually normal. Diagnosis is based on clinical signs, the presence of hypoalbuminemia, and histopathological examination of cecal and ascending colon biopsies. Fecal analysis might be unrewarding.



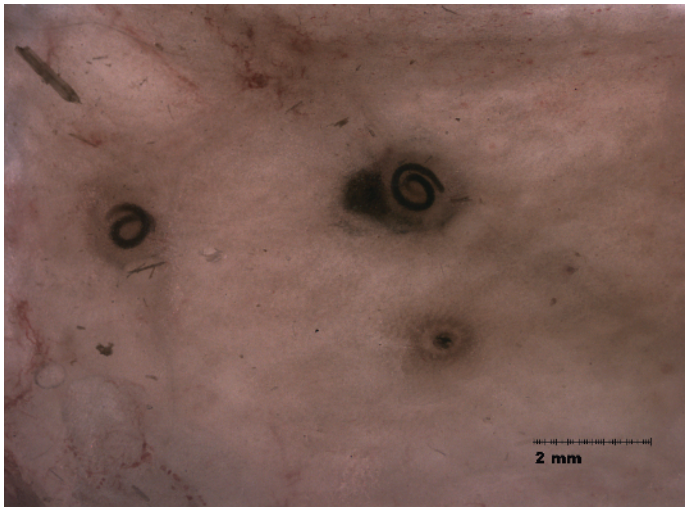
1.145

Figures 1.145–1.146 Postmortem photograph of the large colon of a horse affected with typhlocolitis caused by cyathostomes. Note the thickened, edematous colon with the encysted larvae (black dots) embedded in the intestinal wall.



1.146

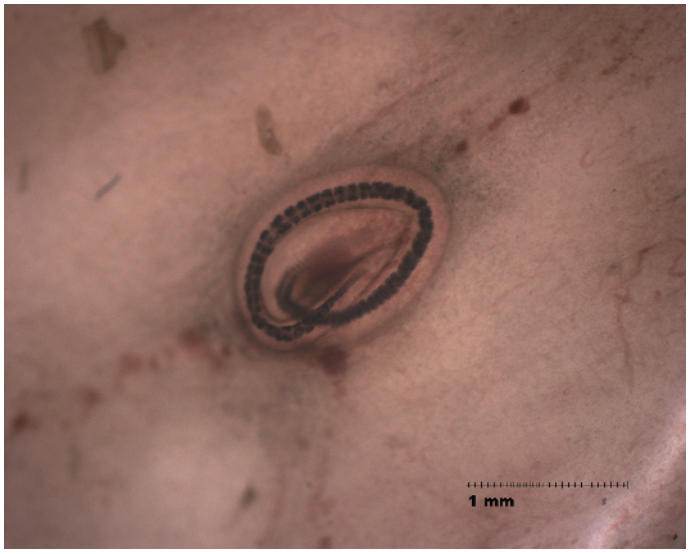
Figures 1.145–1.146 *Continued*



1.147

Figures 1.147–1.148 Microscopic examination of the encysted larvae (black mucosal dots) in the intestinal mucosa seen in fig. 1.144.

Figures 1.147–1.148 *Continued*



1.148

Nonsteroidal Anti-inflammatory Drugs (NSAIDs) Toxicity

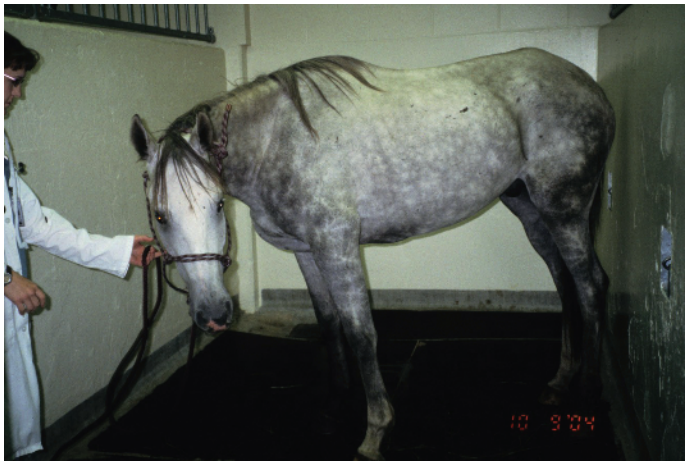


Figure 1.149 An adult horse affected with NSAIDs toxicity. Note the weight loss and ventral edema. Toxicity with NSAIDs can cause GI and renal disease. All NSAIDs can invariably cause toxicity, but phenylbutazone is the drug that is commonly reported to cause toxicity in horses. Overdose or dosage error are the common scenario, however, toxicity has been reported in horses that have been administered the correct dose. Clinical signs include generalized ulceration of the GI tract, starting from the mouth (fig. 1.150), colitis (right dorsal colitis) (fig. 1.151), anorexia, colic, depression, fever, diarrhea, weight loss (protein losing enteropathy), endotoxemia, hypoproteinemia, and ventral and peripheral edema (figs. 1.152 and 1.153). Horses might be presented with chronic clinical signs of mild recurrent colic and protein losing enteropathy. Diagnosis is based on history, clinical signs, ultrasonographic finding of thickened right dorsal colon (>0.5 cm) (fig. 1.154).

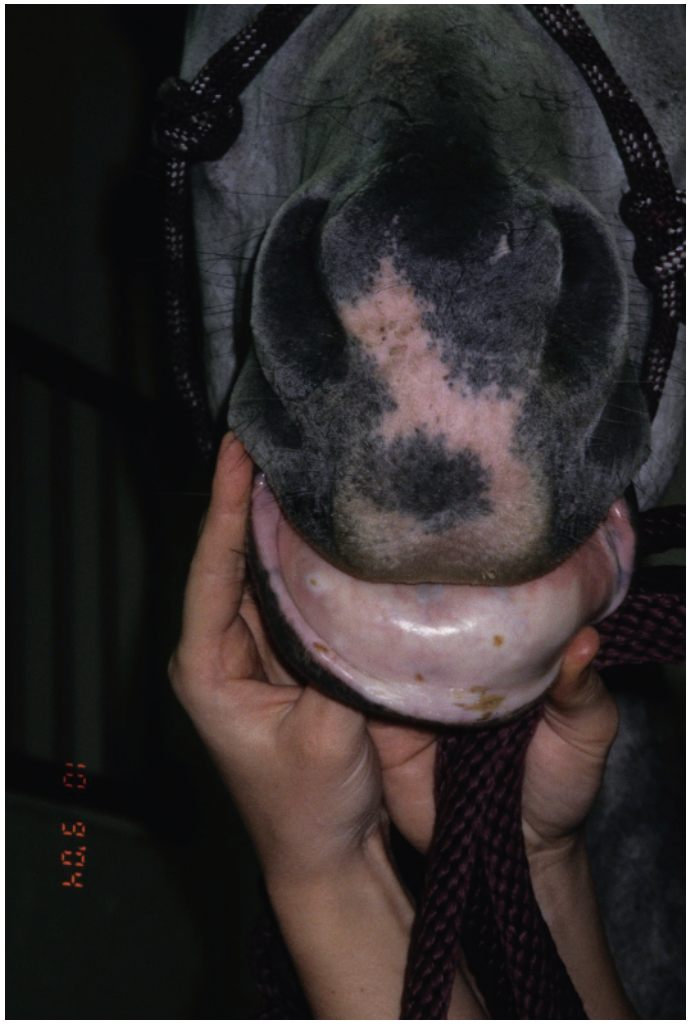


Figure 1.150 An adult horse affected with NSAIDs toxicity. Note the oral ulcers.

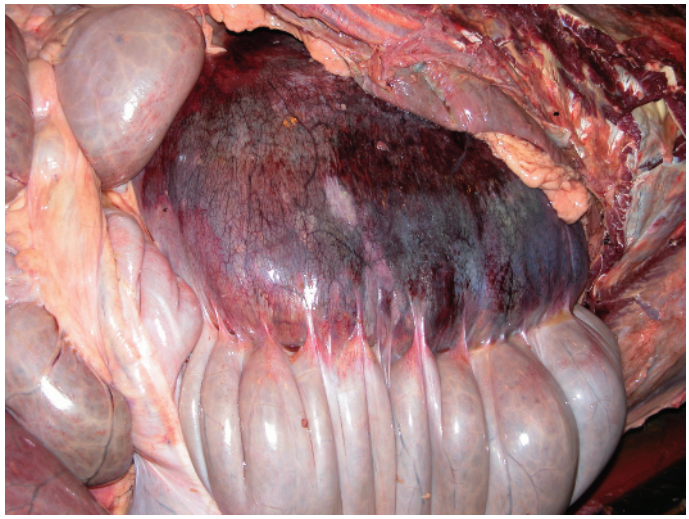


Figure 1.151 Postmortem examination of an adult horse affected with NSAIDs toxicity. Note the severe necrotic right dorsal colon.

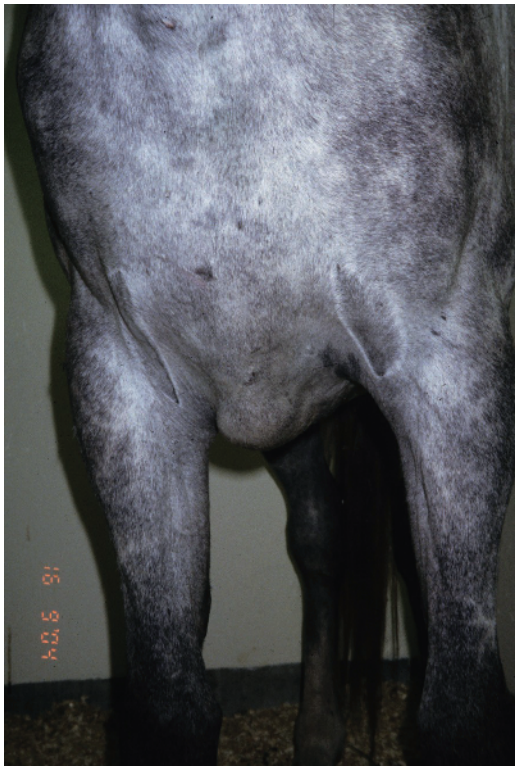


Figure 1.152 Brisket edema in an adult horse affected with NSAIDs toxicity.

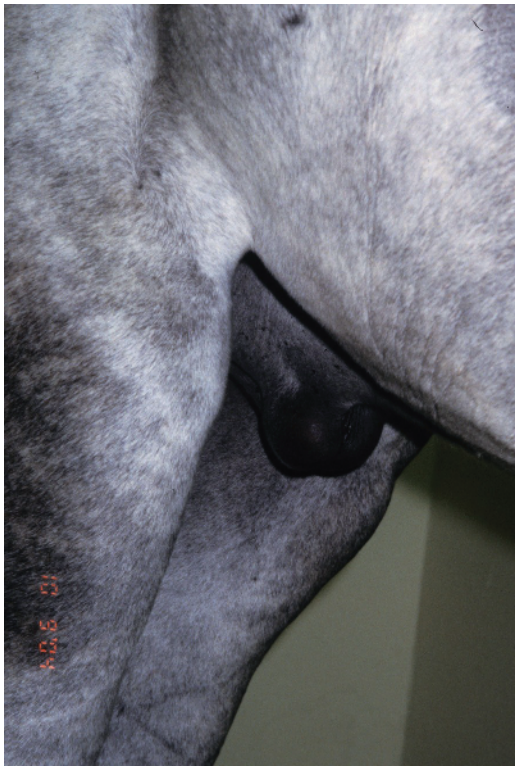


Figure 1.153 Preputial edema in an adult horse affected with NSAIDs toxicity.

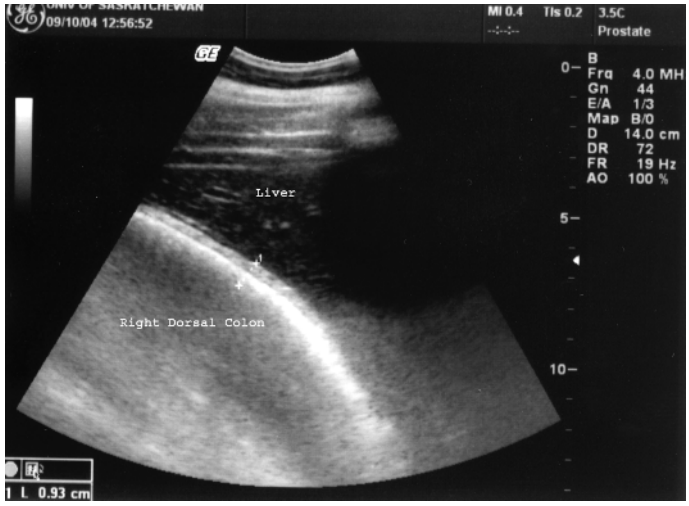


Figure 1.154 An ultrasonographic image of the right dorsal colon in an adult horse affected with NSAIDs toxicity. Note the thickened wall of the right dorsal colon. This figure was captured during ultrasonographic imaging of the right 11th to 14th intercostal spaces.

Grain (Carbohydrate) Overload

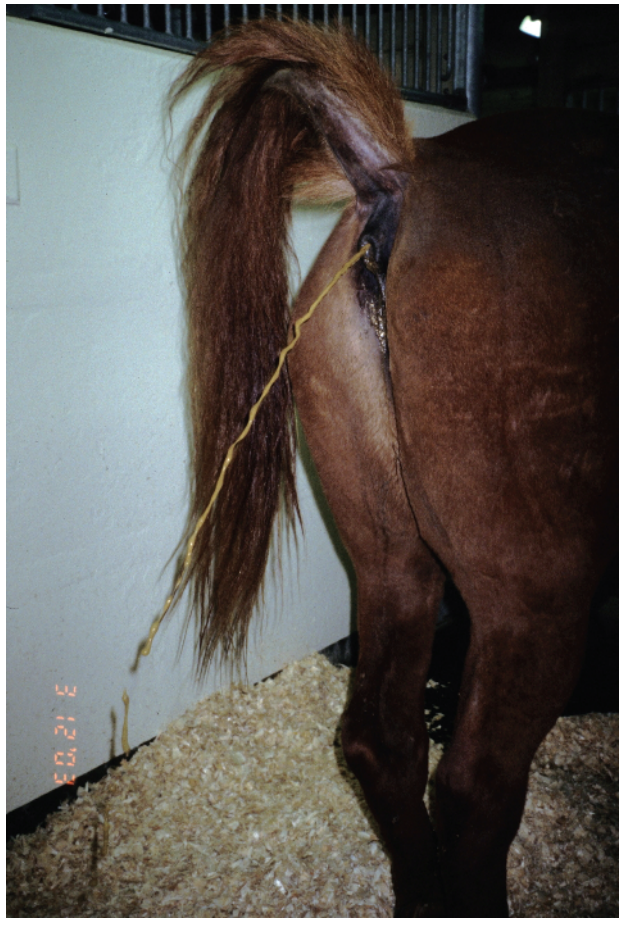
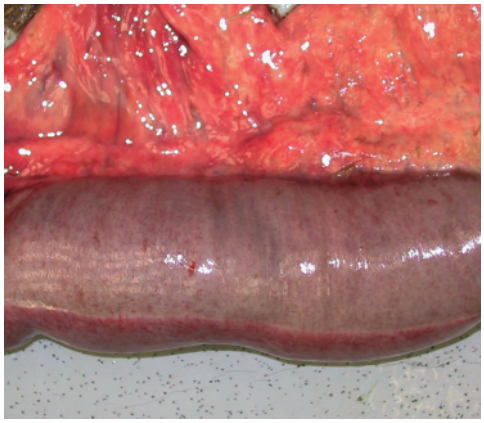


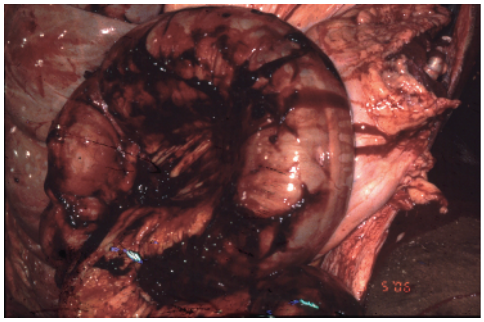
Figure 1.155 An adult horse with severe diarrhea due to grain overload. Grain overload results from feeding horses unusual amounts of grain (corn, barley, wheat). It can cause osmotic diarrhea and colitis. Clinical signs depend on the amount of grain ingested. Clinical signs are identical to those of enterocolitis and range from mild diarrhea to fatal enterocolitis and death. Other clinical signs include the presence of large amounts of undigested grain in the feces, colic, abdominal distention, depression, dehydration, and signs of endotoxemia and laminitis (fig. 1.156). Diagnosis is based on history, clinical signs, the presence of hypocalcemia and metabolic acidosis.

Figure 1.156 The same horse seen in fig. 1.155 after he developed laminitis. The horse had severe and painful laminitis due to which he spent a lot of time laying down.

Small Colon Impaction



a



b

Figures 1.157a,b Postmortem photographs of a horse with small colon impaction. It is the most common abnormal condition of the small colon. The small colon gets impacted with firm ingesta. It occurs usually in the fall and winter. Salmonella is implicated as the cause of small colon impaction. Medical treatment includes aggressive IV fluid therapy and judicious use of analgesics and oral laxatives. Severe surgical intervention is indicated if no improvement is noticed, or if the affected horse becomes progressively painful and develops severe abdominal distension.

Intraluminal Obstruction of the Small Colon with Enteroliths, Fecaliths, or Foreign Bodies

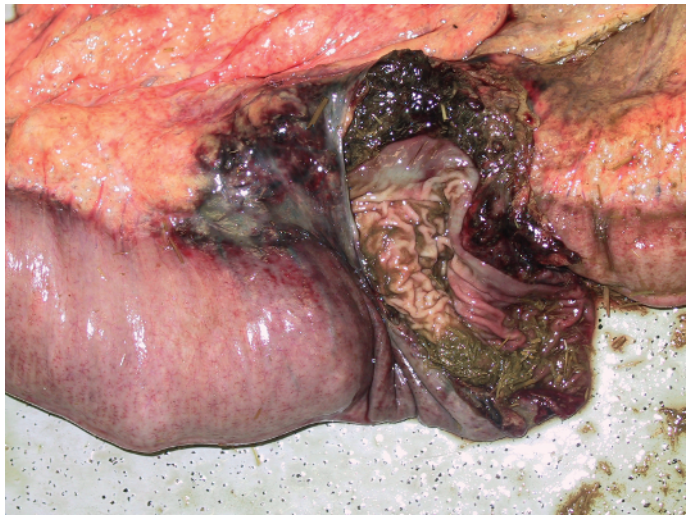


Figure 1.158 Postmortem photographs of a horse with small colon rupture as a result of a foreign body; this is a known complication. Fecaliths (inspissated feces), enteroliths (fig. 1.159), and foreign bodies can lodge in the small colon and cause secondary backup of ingesta and small colon impaction. Fecaliths causing small colon impaction are more common in miniature horses and are thought to be caused by feeding coarse forage.

Figure 1.159 A photograph of an enterolith. Enteroliths are more common in the Arabian breed. It is usually seen in the southwestern United States and California. Abdominal radiography may aid in the diagnosis of obstructive enterolith.

Idiopathic Inflammatory Bowel Disease (Granulomatous Enteritis, Basophilic Enterocolitis, Lymphocytic-Plasmocytic Enterocolitis, Multisystemic Eosinophilic Epitheliotropic Disease, and Idiopathic Eosinophilic Enterocolitis)

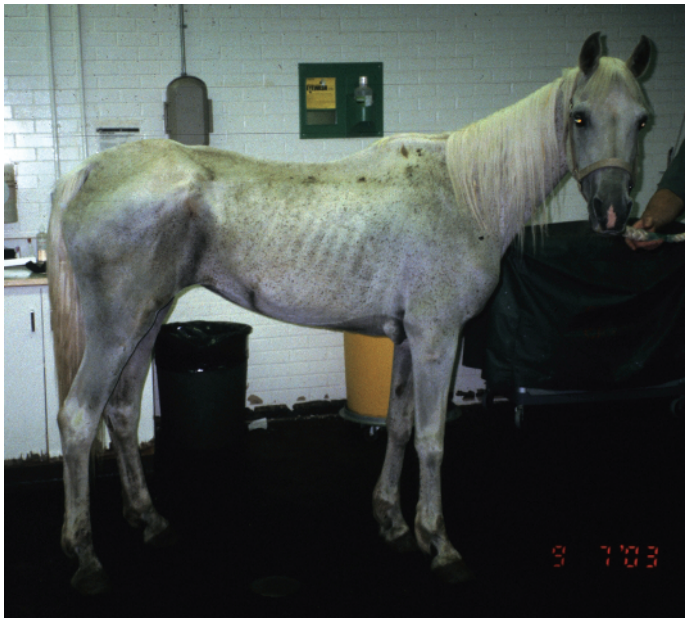


Figure 1.160 A horse affected with idiopathic inflammatory bowel disease (basophilic enterocolitis). Note the severe weight loss. Affected horses are usually presented with weight loss, ill thrift, diarrhea (fig. 1.161) and hypoproteinemia.

Figure 1.161 A horse affected with idiopathic inflammatory bowel disease. Note the loose feces (cow-pie-like diarrhea).

Antibiotic Induced Colitis

Figure 1.162 A horse affected with antibiotic induced colitis. Note the watery diarrhea. Clinical signs are similar to those seen in colitis caused by different reasons. Clinical signs include diarrhea, fever, and endotoxemia. Antibiotic induced colitis can be caused by treatment with most antibiotics and is suspected to be bacterial in origin.

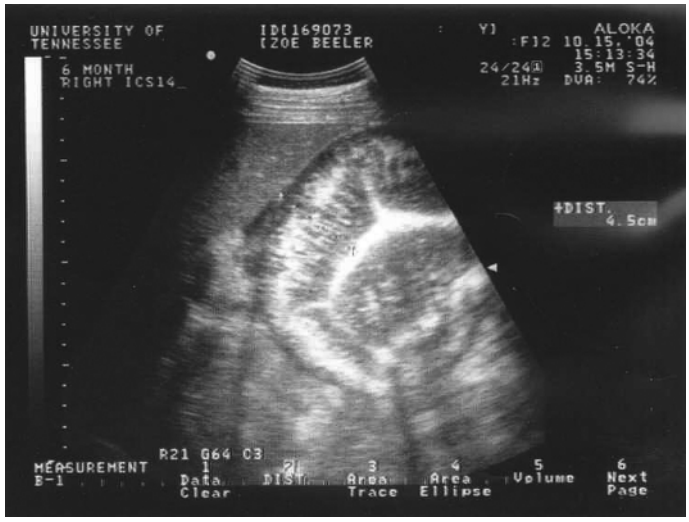


Figure 1.163 Ultrasonographic image of the right dorsal colon of a 6-month-old foal with antibiotic induced colitis. Dorsal is to the left. A small piece of the liver is visible on the left side of the image. The right dorsal colon with tremendous edema is visible on the right. Thickened large colon is usually seen on ultrasonography in cases of colitis.

Abdominal Abscessation

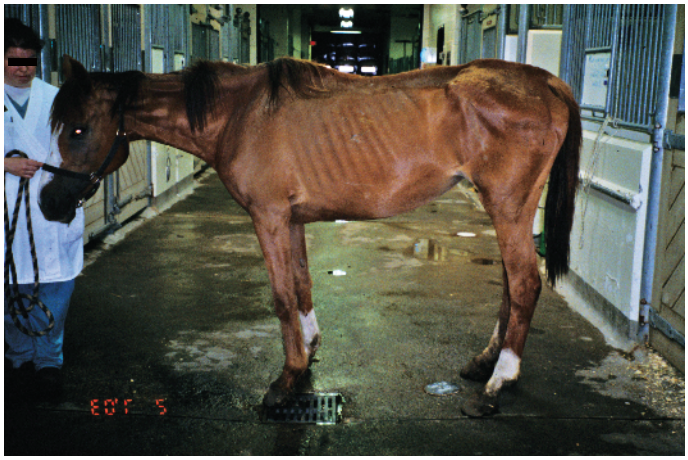


Figure 1.164 An adult horse affected with abdominal abscessation. Note the severe weight loss. Abdominal abscessation occurs as a sequel to respiratory infection, peritonitis, foaling accidents, foreign body penetration of the small intestine, verminous arteritis, umbilical infections, and septicemia. Clinical signs include anorexia, depression, weight loss, and intermittent colic.

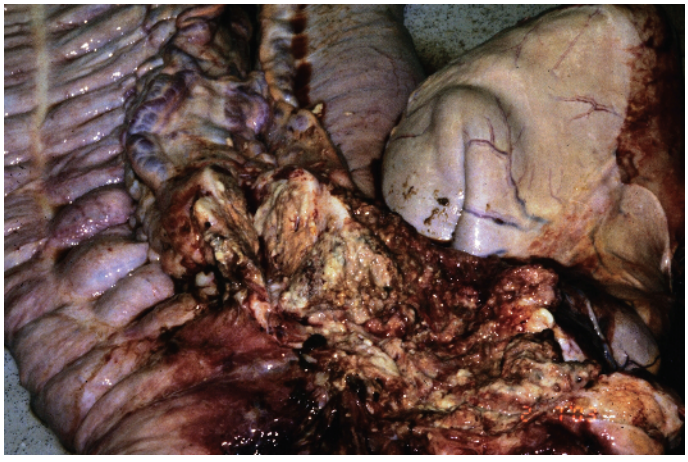


Figure 1.165 Postmortem examination of an adult horse affected with abdominal abscessation. Most abdominal abscesses occur in or around the mesentery.

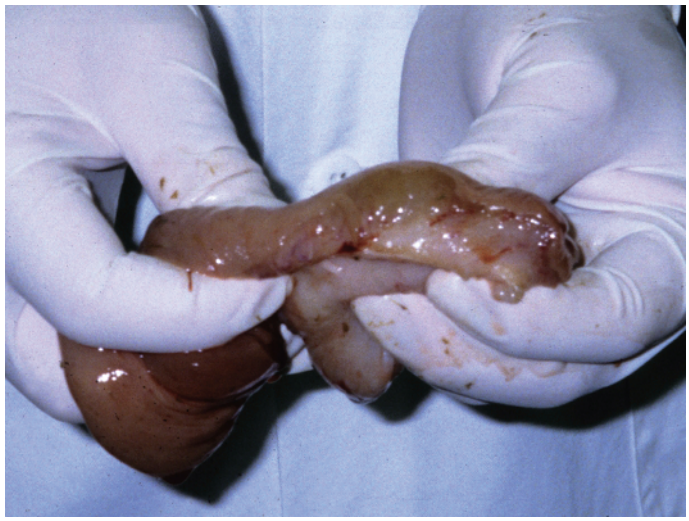


Figure 1.166 Thickened large colon in a horse affected by abdominal abscessations that were pinging on the lymphatic vessels; this resulted in edematous and thickened intestines.

Abdominal Adhesions

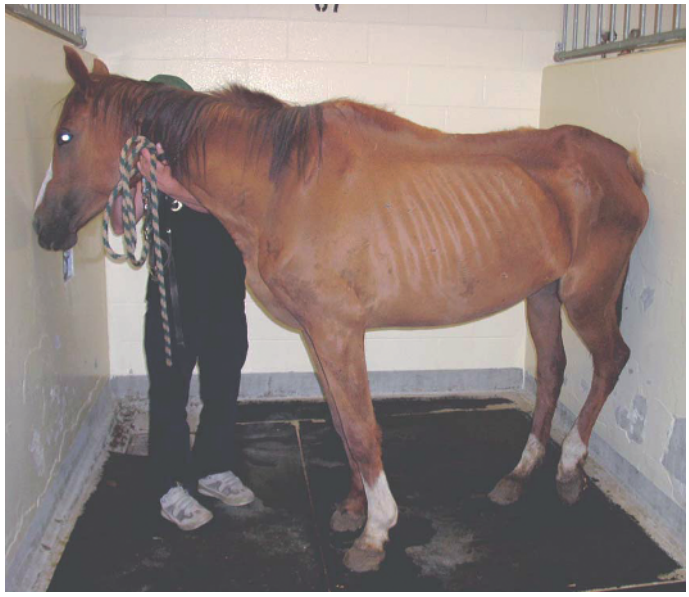


Figure 1.167 An adult horse with severe weight loss caused by abdominal adhesions. Abdominal adhesions usually occur secondary to inflammatory and traumatic injuries to the intestine. It is also seen in horses that have had exploratory laparotomy and reproductive surgeries. Clinical signs include colic due to intestinal obstruction, chronic weight loss, and sometimes soft feces (diarrhea) as in fig. 1.168.

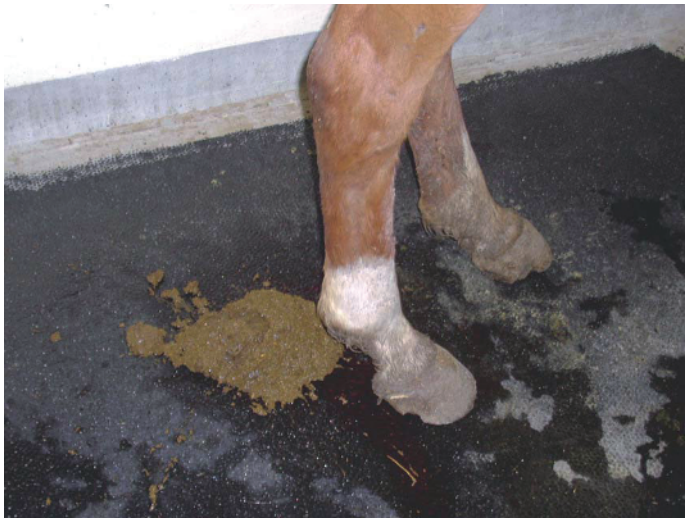


Figure 1.168 An adult horse with abdominal adhesions. Note the diarrhea and "cow pie feces."

Peritonitis

Figure 1.169 An adult horse affected with peritonitis. Although the abdomen seems full, the horse has lost weight. Depending on the cause, peritonitis can be localized or diffuse, septic or nonseptic, primary or secondary. Primary causes include intestinal or gastric rupture, abdominal abscess rupture, and leakage of bacteria to the abdomen from an infected uterus in postpartum mares. Usually it is diffuse and caused by contamination with intestinal bacteria. Clinical signs are variable depending on the primary cause and disease duration. They include depression, anorexia, fever, reluctance to move, splinting of the abdomen, and sensitivity to external pressure, colic, weight loss, signs of intestinal ileus, and endotoxemia. Cases with peracute peritonitis may die in hours. The disease is confirmed by abdominocentesis (figs. 1.170 and 1.171). Abdominal ultrasonography is also helpful in the diagnosis (figs. 1.172 and 1.173).

Figure 1.170 Abdominocentesis in a horse affected with peritonitis. Note the serosanguinous abdominal fluid. Normal peritoneal fluid is clear and straw-colored and has low protein content. In case of peritonitis, the fluid is usually cloudy and blood tinged (serosanguinous). Peritonitis cannot be diagnosed based on the gross appearance of the peritoneal fluid, and microscopic (cytological) examination of the fluid should be done.

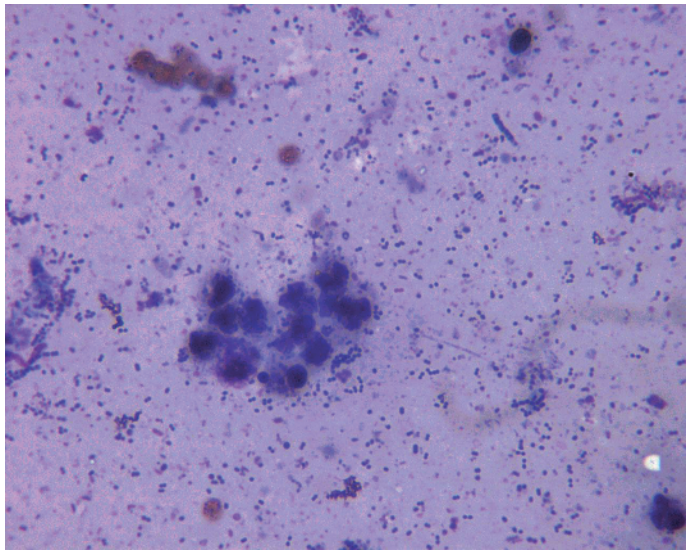


Figure 1.171 Microscopic (cytological) examination of a smear made from peritoneal fluid of a horse with diffuse peritonitis secondary to intestinal rupture. Note the presence of bacteria in the cytoplasm of the neutrophils.

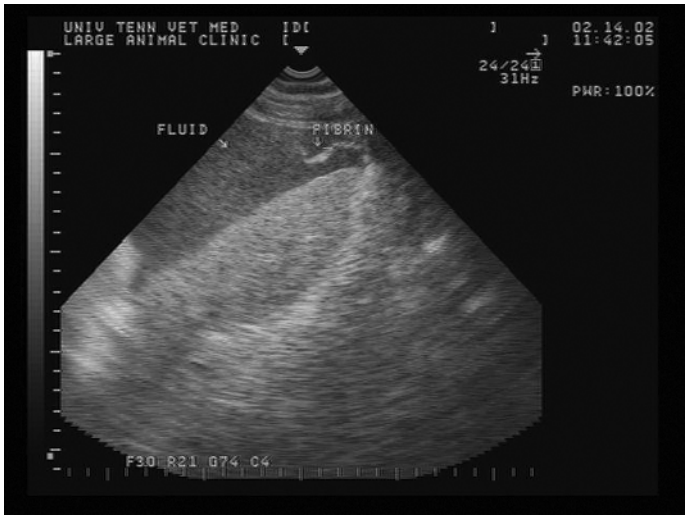


Figure 1.172 Ultrasonographic image of the abdomen in a horse affected with peritonitis (dorsal is to the left). Note the cellular fluid and fibrin tag.

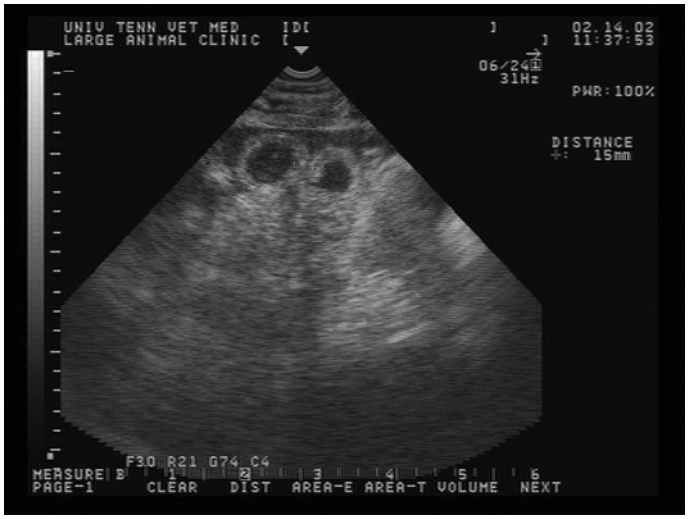


Figure 1.173 Ultrasonographic image of the abdomen in a horse affected with peritonitis (dorsal is to the left). Note the thickened small intestine shown on the top of the picture, surrounded by cellular fluid.

Enterocutaneous Fistula and Parietal (Richter's) Hernia

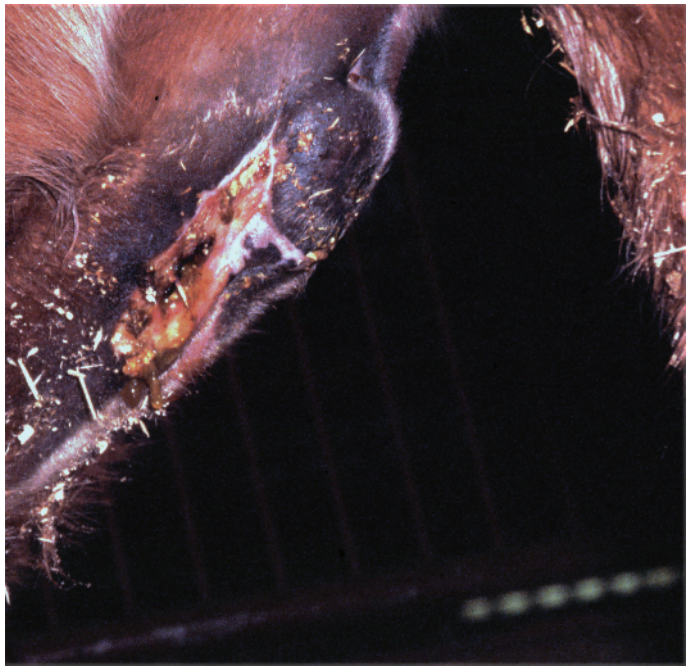


Figure 1.174a Enterocutaneous fistula in a horse as a sequela to parietal (Richter's) hernia. Note the drainage of digesta from the fistula. Parietal or Richter's hernia occurs when the small intestinal (ileum) incarceration, in case of umbilical hernia (fig. 1.174b), involves only a portion of the antimesenteric wall.

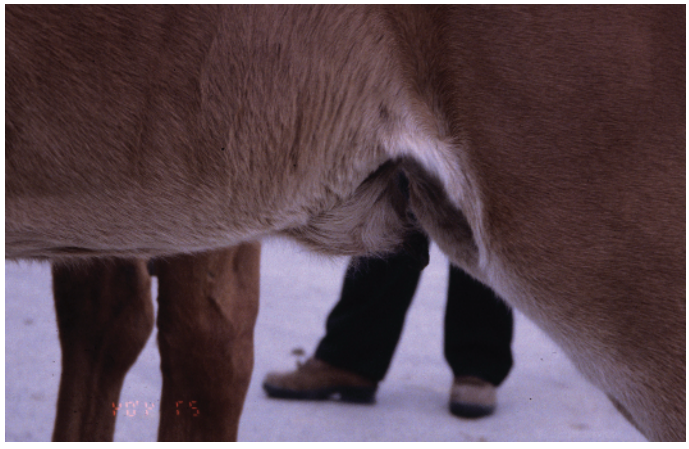


Figure 1.174b Umbilical hernia in a foal.

Omental Hernia

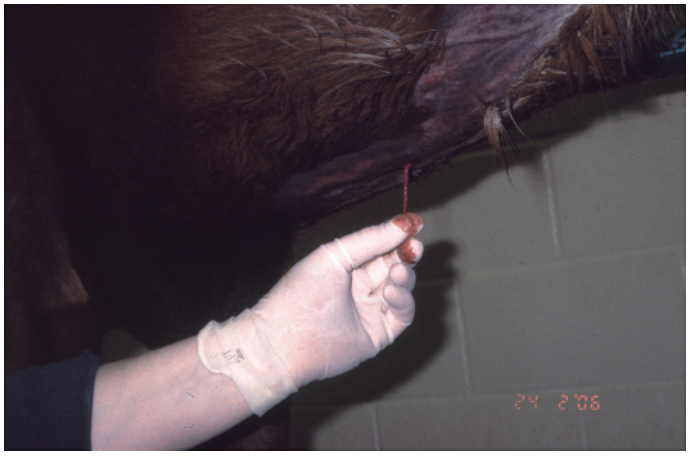


Figure 1.175 Omental hernia in a foal. It usually occurs subsequent to abdominocentesis using a teat cannula in a small percentage of foals. This is usually a benign complication. The prolapsed part of the omentum should be cut close to the skin.

Grass Sickness (Equine Dysautonomia)

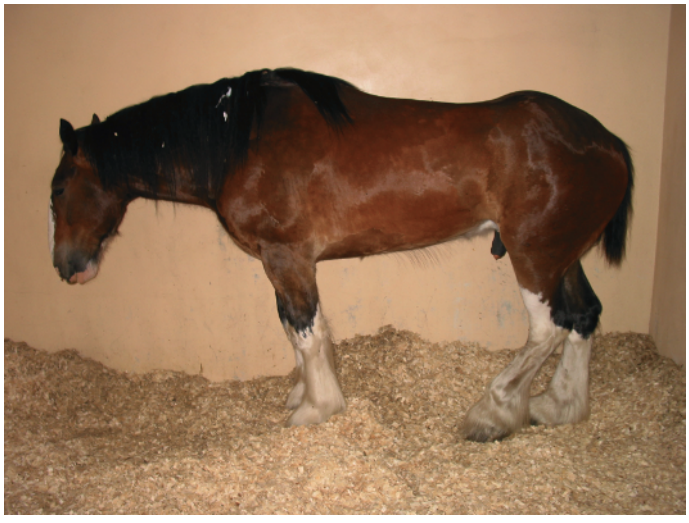


Figure 1.176 An adult horse affected with grass sickness. Note the dullness and weight loss. Grass sickness is a disease of unknown etiology. It is reported in Europe and Australia. A similar disease, *mal seco*, has been reported in Argentina and southern Chile. The disease is sporadic and often fatal. Grass sickness is an acquired degenerative neuropathy that mainly affects the autonomic and enteric nervous system. It has three clinical forms: acute, subacute, and chronic forms. Clinical signs of the three forms overlap from one form to the other. Clinical signs include depression, dullness, fever, "tucked up" appearance (fig. 1.177), weight loss, dysphagia (fig. 1.178), signs of intestinal ileus and large colon impaction (fig. 1.179), nasogastric reflux, abdominal pain (fig. 1.180), piloerection (fig. 1.181), patchy sweating (fig. 1.182), muscle fasciculation, rhinitis sicca (figs. 1.183 and 1.184), gait abnormalities, narrow base stance, leaning against the walls (fig. 1.185), ptosis (fig. 1.186), pica, and penile prolapse and paralysis (fig. 1.187). Antemortem diagnosis of grass sickness can be confirmed only by histological examination of ileal biopsy obtained via laparotomy. There is no curative treatment for grass sickness. (Image courtesy of Dr. R. Scott Pirie, University of Edinburgh, Scotland)

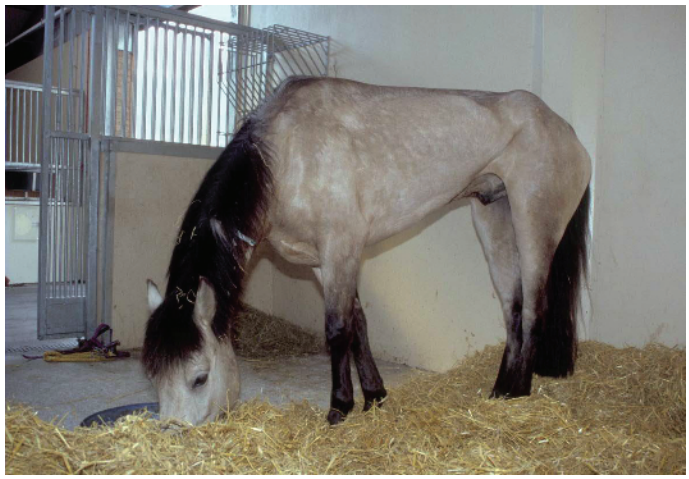


Figure 1.177 A “tucked up” appearance and weight loss in a horse affected with grass sickness. (Image courtesy of Dr. R. Scott Pirie, University of Edinburgh, Scotland)

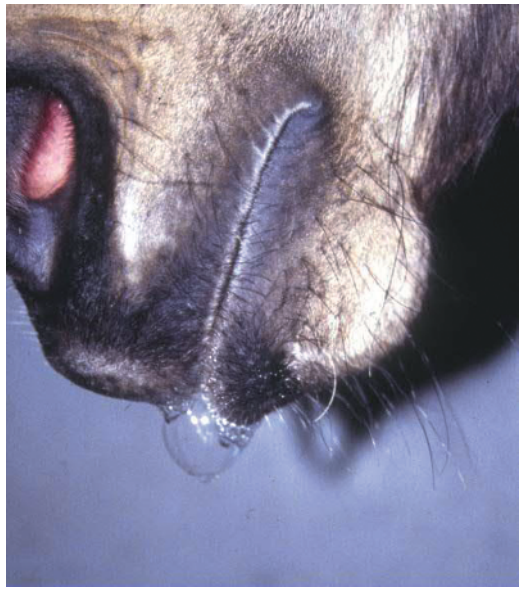


Figure 1.178 Drooling of saliva and dysphagia in a horse affected with grass sickness. (Image courtesy of Dr. R. Scott Pirie, University of Edinburgh, Scotland)

Figure 1.179 Large colon impaction found during postmortem examination of a horse affected with grass sickness. (Image courtesy of Dr. R. Scott Pirie, University of Edinburgh, Scotland)

Figure 1.180 Abdominal pain (colic) in a horse affected with grass sickness. (Image courtesy of Dr. R. Scott Pirie, University of Edinburgh, Scotland)

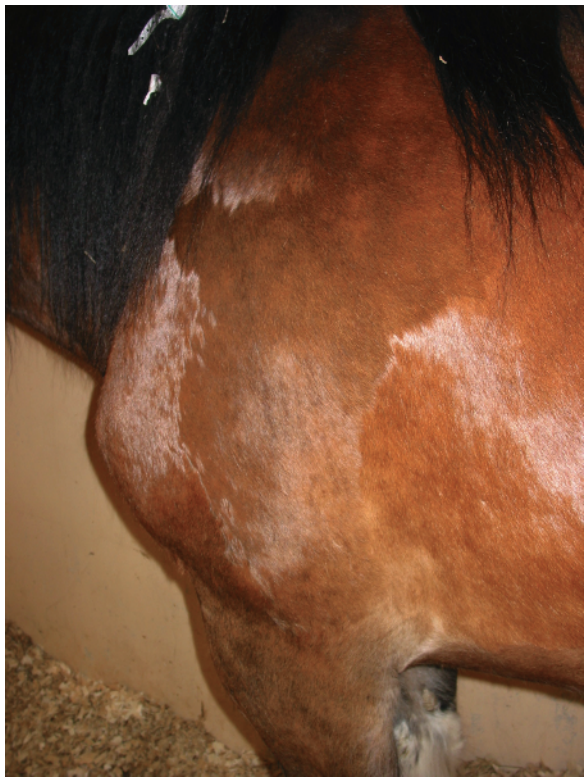


Figure 1.181 Piloerection (erection of hair) in a horse affected with grass sickness. (Image courtesy of Dr. R. Scott Pirie, University of Edinburgh, Scotland)

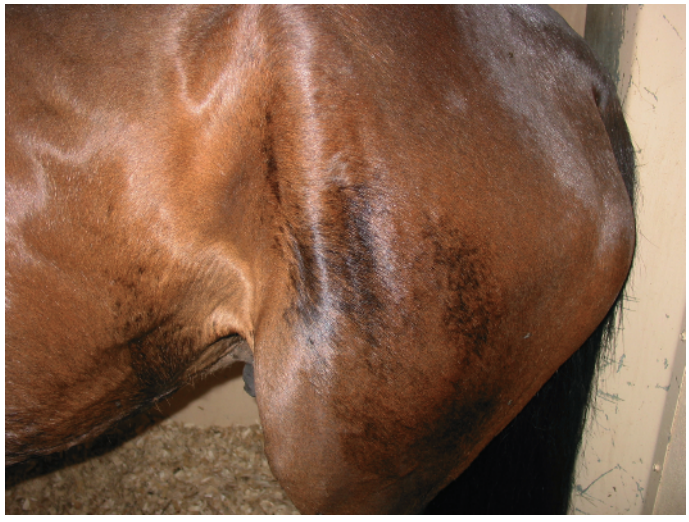


Figure 1.182 Patchy sweating in a horse affected with grass sickness. (Image courtesy of Dr. R. Scott Pirie, University of Edinburgh, Scotland)

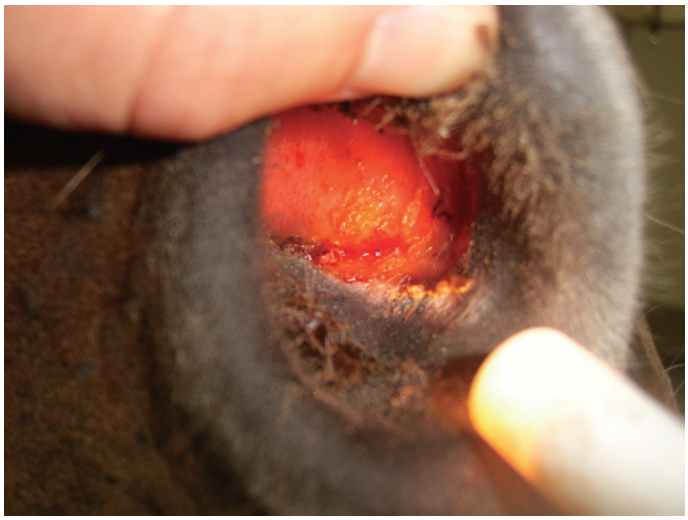


Figure 1.183 Rhinitis sicca in a horse affected with grass sickness. (Image courtesy of Dr. R. Scott Pirie, University of Edinburgh, Scotland)

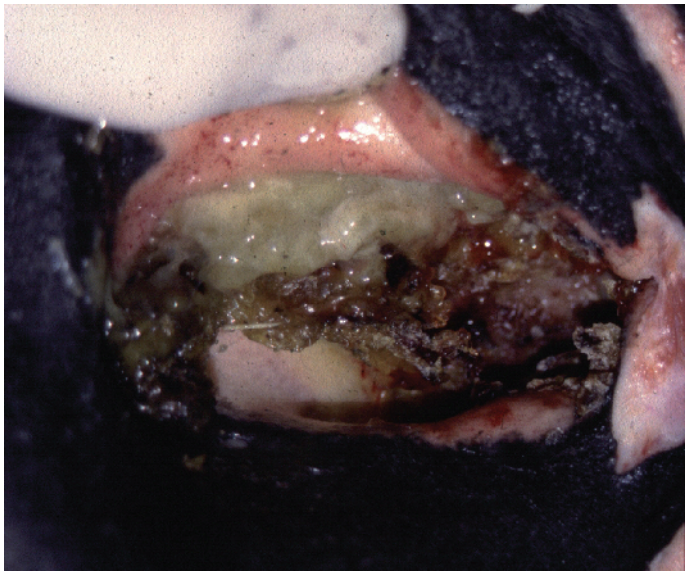


Figure 1.184 Rhinitis sicca in a horse affected with grass sickness. Note the accumulation of mucopurulent material in the nasal passages. (Image courtesy of Dr. R. Scott Pirie, University of Edinburgh, Scotland)

Figure 1.185 Narrow base stance and leaning against the walls in a horse affected with grass sickness. (Image courtesy of Dr. R. Scott Pirie, University of Edinburgh, Scotland)

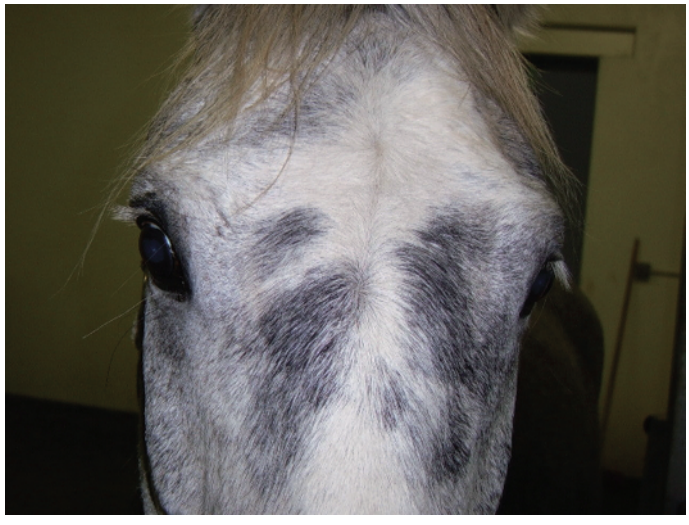


Figure 1.186 Ptosis (droopy upper eyelid), in a horse affected with grass sickness. (Image courtesy of Dr. R. Scott Pirie, University of Edinburgh, Scotland)

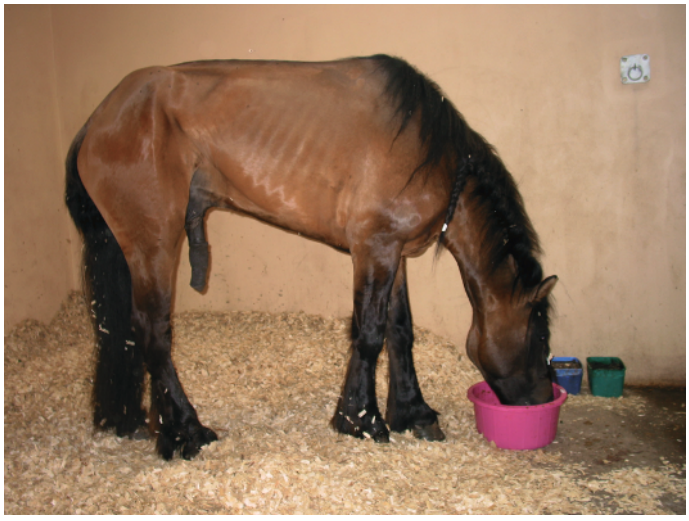


Figure 1.187 Penile prolapse and paralysis in a horse affected with grass sickness. (Image courtesy of Dr. R. Scott Pirie, University of Edinburgh, Scotland)

Hyperlipemia and Hyperlipidemia

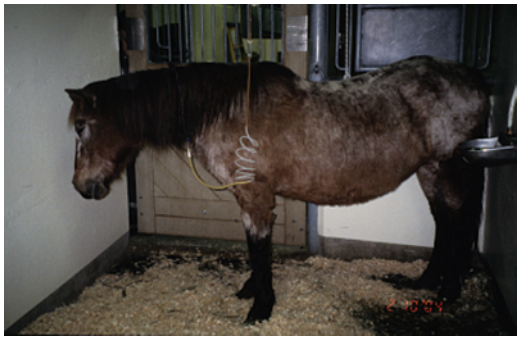


Figure 1.188 Hyperlipemia in a pony. Note the depression. Hyperlipemia/hyperlipidemia is caused by negative energy balance. The disease is mainly seen in ponies. It is characterized by serum triglyceride elevation. In hyperlipidemia, serum triglyceride elevation is up to 500 mg/dL, while it is much over that in cases of hyperlipemia. In cases of hyperlipemia, which is much more severe than hyperlipidemia, the plasma is milky and hepatic lipidosis is present (fig. 1.189). Clinical signs of hyperlipemia include anorexia, depression, weakness, icterus (fig. 1.190), and incoordination. Fatty and swollen liver is usually seen on postmortem examination (fig. 1.191).

Figure 1.189 Milky plasma in a pony affected with hyperlipemia.

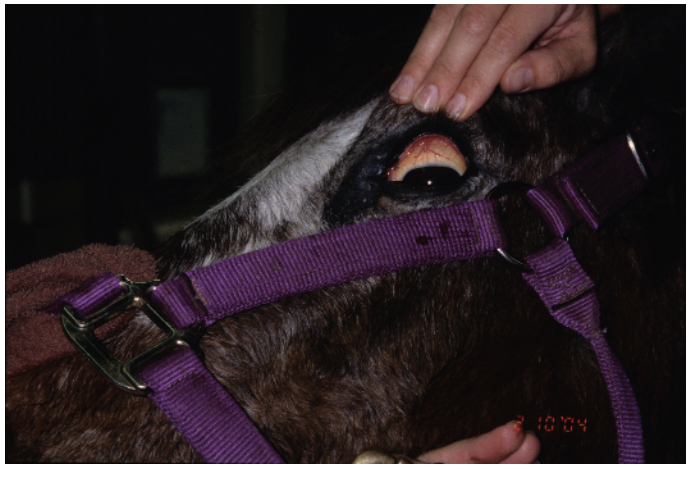


Figure 1.190 Icteric mucous membranes of a pony affected with hyperlipemia.

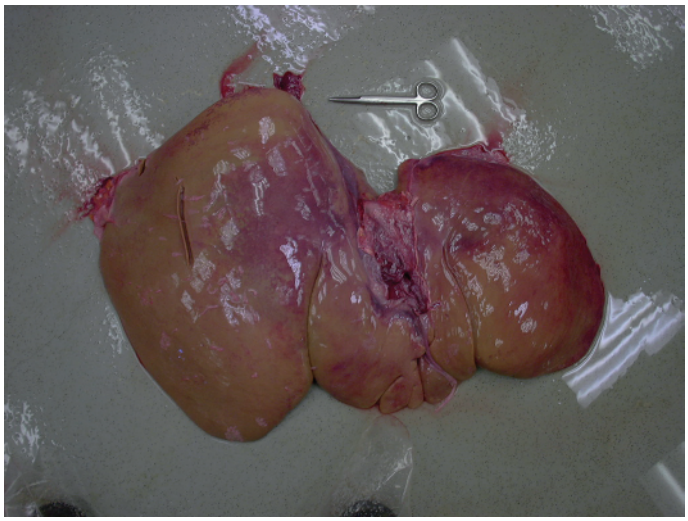


Figure 1.191 Fatty and swollen liver obtained during postmortem examination from a pony affected with hyperlipemia.

Recommended Readings

Abutarbush SM. Dysphagia in horses. Large Animal Veterinary Rounds 4(2), 2004.

Abutarbush SM. Use of ultrasonography to diagnose large colon volvulus in horses. J Am Vet Med Assoc. 228(3):409–13, 2006.

Abutarbush SM, Carmalt JL. Endoscopy and arthroscopy for the equine practitioner. Made Easy Series. Jackson, WY: Teton NewMedia, 2008.

Abutarbush SM, et al. Clinical West Nile virus infection in 2 horses in western Canada. Can Vet J. 45(4):315–17, 2004.

Abutarbush SM, Carmalt JL, Shoemaker RW. Causes of gastrointestinal colic in horses in western Canada: 604 cases (1992–2002). Can Vet J. 46:800–805, 2005.

Abutarbush SM, Naylor JM. Comparison of surgical versus medical treatment of nephrosplenic entrapment of the large colon in horses: 19 cases (1992–2002). J Am Vet Med Assoc. 227:603–5, 2005.

Abutarbush SM, Shoemaker RW, Bailey JV. Strangulation of small intestines by a mesodiverticular band in 3 adult horses. Can Vet J. 44(12):1005–6, 2003.

Alder HA, Arey LB, Barr ML, et al. Dorland's illustrated medical dictionary, 24th ed. Philadelphia: WB Saunders Co, 1965.

Baum KH, Halpern NE, Banish LD, Modransky PD. Dysphagia in horses: the differential diagnosis—part II. Compendium Contin Educ Pract Vet. 10(12):1405–10, 1988.

Baum KH, Modransky PD, Halpern NE, Banish LD. Dysphagia in horses: the differential diagnosis-part I. Compendium Contin Educ Pract Vet. 10(11):1301–7, 1988.

Brown CM. Problems in equine medicine. Philadelphia: Lea & Febiger, 67–80, 1989.

Burba DJ, Moor RM. Renosplenic entrapment: a review of clinical presentation and treatment. Equine Vet Educ. 9:180–84, 1997.

Cohen ND. Neurologic evaluation of the equine head and neurogenic dysphagia. Vet Clin North Am Eq Pract. 9(1):231–40, 1993.

Craig DR, Shivy DR, Pankowski, Erb HN. Esophageal disorders in 61 horses results of nonsurgical and surgical management. Vet Surg. 18(6):432–43, 1989.

Dart AJ, Snyder JR, Pasco JR, Farver TB, Galuppo LD. Abnormal conditions of the equine descending (small) colon: 102 cases (1979–1989). J Am Vet Med Assoc. 200:971–78, 1992.

Edwards GB. Duodenitis-proximal jejunitis (anterior enteritis) as a surgical problem. Eq Vet Edu. 12:411–14, 2000.

Greet T. Dysphagia in the horse. In Practice 11(6):256–62, 1989.

Hance SR, Noble J, Holcomb S, Rush-Moore B, Beeard W. Treating choke with oxytocin. Am Assoc Eq Pract Proceedings 43:338–39, 1997.

Heath SE, Artsob H, Bell RJ, Harland RJ. Equine encephalitis caused by snowshoe hare (California serogroup) virus. CVJ 30:669–71, 1989.

Hillyer M. Management of oesophageal obstruction ("choke") in horses. Practice 17(10):450–56, 1995.

Howarth S, Lane JG. Multiple cranial nerve deficits associated with auditory tube (guttural pouch) diverticulitis: three cases. Eq Vet Edu. 2(4):206–7, 1990.

Jones SL, Zimmel D, Tate LP, Campbell N, Redding WR, Carlson GP. Dysphagia caused by squamous cell carcinoma in two horses. Compendium Contin Educ Pract Vet. 23(11):1020–24, 2001.

Klohnen A, Vachon AM, Fischer AT, Jr. Use of diagnostic ultrasonography in horses with signs of acute abdominal pain. J Am Vet Med Assoc. 209(9):1597–601, 1996.

MacKay RJ. On the true definition of dysphagia. Compendium Contin Educ Pract Vet. 1988(11):1024–28, 2001.

Mair T, Divers T, Ducharme N. Manual of equine gastroenterology. London: WB Saunders, 63–67, 2002.

Modransky PD, Reed SM, Barbee DD. Dysphagia associated with guttural pouch empyema and dorsal displacement of the soft palate. Equine Practice 4(8):3–38, 1982.

Murray MJ, Smith BP. Diseases of the alimentary tract. In Smith BP, ed. Large animal internal medicine. 3rd ed. St Louis: Mosby, 593–789, 2002.

Pascoe R. Differential diagnosis of diseases of horses. University of Sydney Post Graduate Foundation in Veterinary Sciences, 190–95, 1994.

Radostits OM, Gay CC, Blood DC, et al. Diseases of the alimentary tract—1 Veterinary medicine: a textbook of the diseases of cattle, sheep, pigs, goats, and horses. 9th ed. London: WB Saunders, 169–258, 2000.

Swerczek TW. Toxicoinfectious botulism in foals and adult horses. JAVMA 176(3):217–20, 1980.

Wagner PC, Rantanen NW, Grant BD. Differential diagnosis of dysphagia in the horse. Modern Vet Pract. 60(12): 1029–33, 1979.