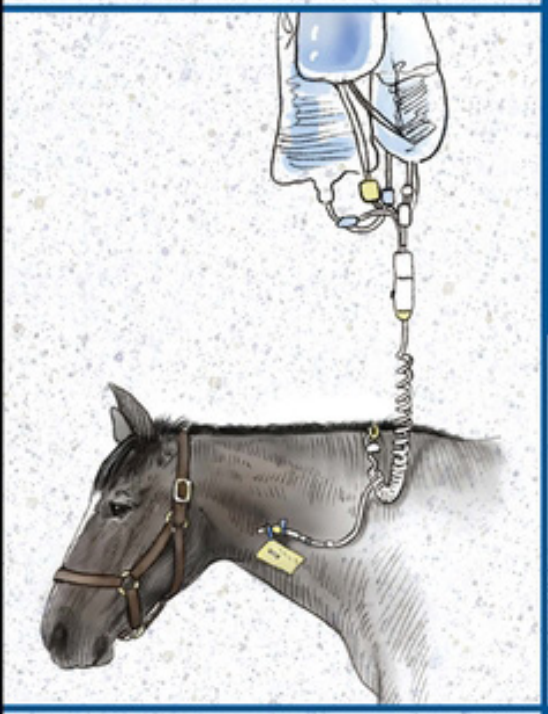
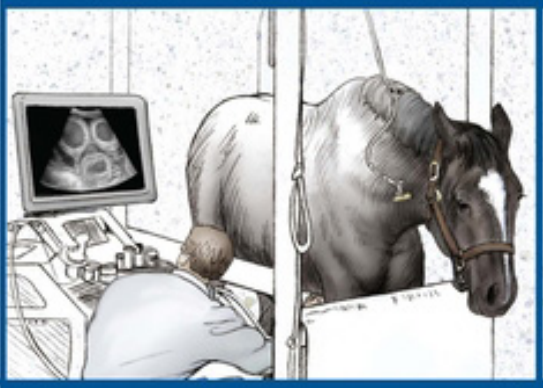
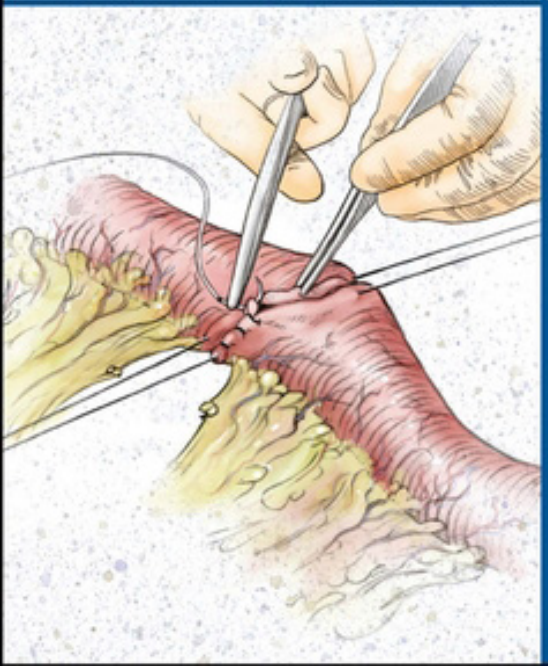
COLIC SURGERY IN THE HORSE







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Colic Surgery in the Horse

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Preface

Two roads diverged in a wood, and I
I took the one less traveled by. Robert Frost.

Three factors have encouraged me to write this book. Firstly, the methods and principals described have performed well in clinical use, although comparable or better techniques might well exist. The second reason is that many of the surgical methods described are not available in mainstream textbooks. The third, and probably the most important reason, is that some controversial opinions are proposed by the author, and if experiences of others support or refute these opinions, then the book has met an important goal. Examples of such are definition of chronic versus recurrent colic, multifactorial causes of postoperative reflux, failure of horses to vomit, interpretation of postoperative endotoxemia, limitations of prognostic indicators, indications for repeat celiotomy, and others.

The role of surgery in saving horses from a cruel and capricious disease is well known, but the failure of surgery in some cases prompts us to resort to aggressive pharmacological management postoperatively. The surgeon should recognize the possibility of a poor cost to benefit ratio for many such postoperative treatments, and cost alone can deny many horses access to a critical life-saving surgery. The relevant drugs are discussed throughout to achieve some balance in the role of all factors that can influence outcome. However, a minimalist approach to use of these drugs should be considered on a per case basis and not viewed as patient neglect nor as a threat to boundaries of humane treatment. However, pain control is always applied without shortcuts and can even include repeat celiotomy.

Although readers might question the omission of some surgical procedures or treatments that they see as critical to the outcome, the author has limited the descriptions to those with which he is most comfortable. Many old references are included because their retreat into the past could deny us some clues relevant to the future. The book is arranged in order so that the different diseases and the unique challenges of each precede the diagnostic and therapeutic sections. Pathophysiology precedes diagnosis and treatment because of its important influence on both. Client communications play a critical role in decision making, and the decision process relies heavily on the surgeon's input, the client's needs and the cost. The author encourages an optimistic approach to surgery, offering all affordable and reasonable options to give the horse a chance. The details of supportive and surgical treatments follow, along with aftercare tailored to address each surgery's unique risks.

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Many have helped me with this project, especially through kind words of encouragement. I thank Jeanne for her endless patience and support (married to a colic surgeon is not easy). I am grateful to those who gave me the opportunities that shaped my career, including Eleanor Green, Joe Di Pietro, Ted Valli, Fred Troutt, Bob Marshak, Charlie Raker, and Bill Donawick. I was inspired by my veterinary school professor, John O' Connor, whose calm demeanor and patience in surgery taught me that more than skill was involved in the outcome. Al Merritt stimulated my interest in gastrointestinal function and its relationship to disease, and most importantly, kindled my interest in research. My father shared with me his great fondness for horses that stimulated my desire to combat the worst of their diseases. I especially thank junior colleagues, faculty and house officers who provided innovative ideas, enthusiasm, and a fresh perspective. Most importantly, I am forever grateful to the house officers, technical staff, and students that cared for the wonderful horses that I had the privilege to treat.

I am very grateful to Cindy Brockett for the high quality and fast delivery of much of the printed product. I appreciate the contribution of my colleague, Luisito Pablo, to the Anesthesia chapter, which was written from the perspective of the horse anesthetized with colic. I thank Sally DeNotta for providing outstanding images to the chapter on diagnostic procedures. I thank Barbara Harmon for her wonderful cover artwork, which displays the progress of a horse through the throes of colic, followed by diagnostic and therapeutic procedures that produce the desired outcome, return to full function. This closely follows the written content, tracing the route from impending catastrophe to a full recovery.

I fully appreciate all the hard work, patience and guidance from the Wiley Blackwell team that ensured this book would reach completion and not become a Sisyphean failure. Erica Judisch, the Executive Editor at Wiley, was always supportive and helpful, even when I had doubts. I thank Jamila Niroop, Content Refinement Specialist, who guided me through the critical final stages.

1

Diseases of the Stomach

The stomach is rarely affected by diseases that require surgery and is more likely to become involved secondary to obstructions in other parts of the gastrointestinal tract. However, some primary diseases of the stomach can cause colic, and can be difficult to distinguish from surgical lesions in other sites.

Gastric Mucosal Diseases

These are the most common types of gastric diseases in horses and typically respond to medical treatment. However, many can be presented as colic and need to be distinguished from other diseases that display such signs. Duodenitis/proximal jejunitis (DPJ) probably has a component of gastritis but is covered in Chapter 2 because of small intestinal involvement.

Gastric Ulcers

Gastric ulceration is the most common disease of the equine stomach.[1–3] Equine Gastric Ulcer Syndrome (EGUS) is an early grouping of diseases characterized by ulcers in the terminal esophagus, nonglandular or squamous portion of the stomach, the glandular portion of the stomach, and proximal duodenum (Figure 1.1).[4] Reported prevalence of ulcers in mature horses varies from 17% in a population of Swedish horses at necropsy, 82–91% in a racehorse population, 71% in Thoroughbred broodmares at pasture,[5] 58% in show horses,[6–11] and 53% in pleasure horses not in active training.[12] A prevalence of 91% recorded in horses in race training can increase to 100% in those horses actively racing.[6,13,14] The prevalence of gastric ulcers in foals is 25–50%.[15] Within the syndrome of EGUS, two different disease entities are currently recognized (Figure 1.1): equine squamous gastric disease (ESGD) and equine glandular gastric disease (EGGD).[1,3,16] This distinction is based on different locations, rates of prevalence, risk factors, and responses to treatments.[16] Compared with a prevalence of 50–93% for ESGD, a prevalence of EGGD ranges from 8% to 63%.[10,16]

Pathophysiology

Although typically regarded as a single-compartment organ, the stomach can be divided into the fundus dorsally, the body in the mid-third, and then the pyloric antrum in the most distal third.[1] The dorsal third is covered with thick cornified, stratified squamous epithelium that is nonglandular and continuous with esophageal mucosa (Figure 1.2). The remainder of the stomach is covered with glandular mucosa (Figure 1.2).[17] The glandular portion contains mucous neck cells, chief cells (produce pepsinogen), parietal or oxyntic cells (produce HCl), and enterochromaffin-like cells (ECL; produce histamine).[18] The lining of the most distal part or antrum is arranged in prominent folds and has the largest concentration of gastrin-producing cells or G-cells (stimulate acid production).[18] Secretion of HCl by the glandular part of the stomach and the resulting acidity (and pepsin activation) are considered the dominant causes of gastric ulcers, with little if any contribution from short-chain fatty acids, lactic acid, and duodenal bile salts.[3] The associated injury to tight junctions could increase paracellular permeability and allow acid to diffuse into the intercellular space and induce cell damage.[19] Unlike human gastric ulcers, a bacterial cause (e.g. *Helicobacter pylori*) has not been identified in horses.[20–22]

The concentration of acid in gastric fluid is determined by the rate of its secretion, neutralization with exogenous or endogenous buffers, dilution and neutralization with salivary secretions and food, and loss through the pylorus.[23] Horses secrete HCl almost continuously,[24] and gastric pH is likely to be lowest during periods when horses are not eating and gastric acid is not buffered by breakdown products of food.[3] Contents of the nonglandular region have a pH of approximately 5.4, compared with a pH of approximately 2.6 in pyloric contents,[25] which suggests poor mixing

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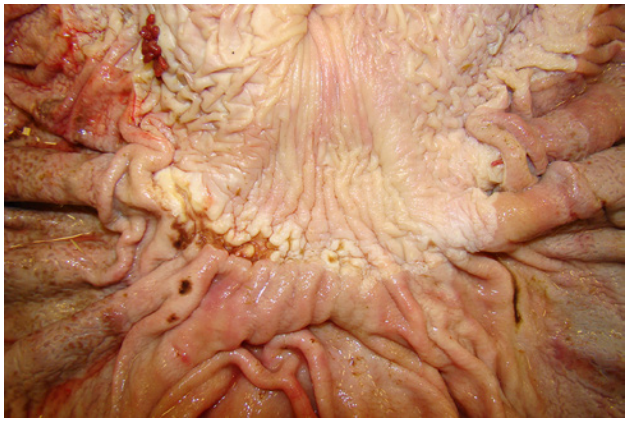

Figure 1.1 Equine squamous gastric disease (ESGD) and equine glandular gastric disease (EGGD) to left and bottom of necropsy image.

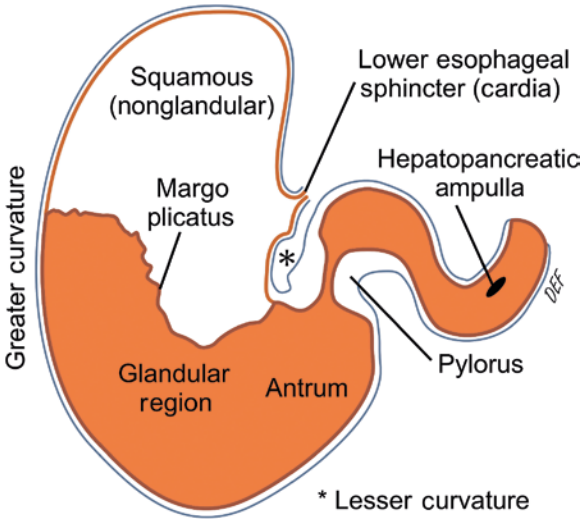

Figure 1.2 Anatomy of the interior of the stomach of the horse, displaying the sites relevant to gastric ulceration.

between these segments. However, this is not a consistent finding, and variation is considerable during interdigestive periods.[25] Feeding reduces gastric pH from near neutrality to about 2.5 over an eight-hour period with some dependence on the type of feed and feeding regimen.[19] Withholding food from horses decreases gastric pH rapidly and exposes the nonglandular mucosa to acid.[26] Intermittent feeding can increase severity of nonglandular ulcers and provides a useful model to produce these lesions.[27] More recent evidence suggests that the pH in the proximal part of the stomach can follow a circadian rhythm, with a significant decrease from 01.00 to 09.00 hours compared with 13.00 to 20.00 hours.[28] In the glandular (distal) part of the stomach, pH remains stable throughout the day.[28,29]

In ESGD, ulcers develop along the margo plicatus (Figure 1.3), especially along the lesser curvature of the

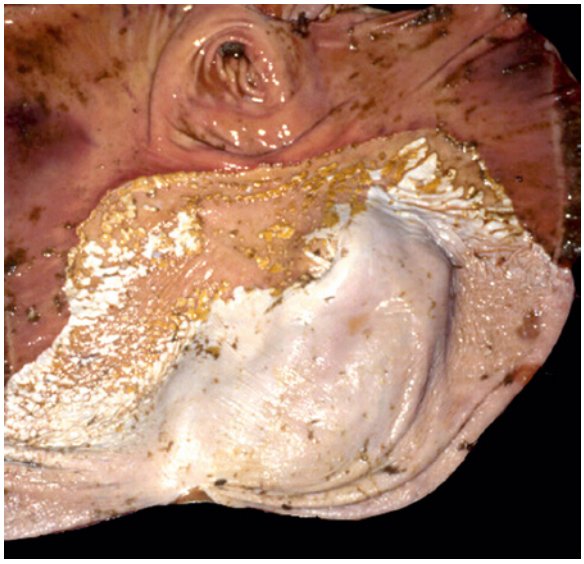

Figure 1.3 Extensive ESGD extending along the margo plicatus at the lesser curvature of the stomach in a horse with depression, inappetence, and colic.

stomach,[10] a predilection site attributed to the absence of critical protective mucous and HCO_3- secretion on the surface of these cells.[4] Galloping could increase the risk of damage to this site through abdominal compression, which would force acidic contents dorsally into the poorly protected nonglandular portion of the stomach.[30] By comparison, the distal portion of the stomach is lined by glandular mucosa with extensive protective mechanisms from mucous and HCO_3- secretions.[4] Most of these glandular mucosal ulcers (EGGD) are labeled as erosions on histological examination[2] and might be capable of repair through rapid restitution of damaged epithelial layers, even in the absence of any pharmacologic intervention.[4]

Gastric Ulcers and NSAIDs

Gastric mucosal injury by nonsteroidal antiinflammatory drugs (NSAIDs) has been documented in horses[31–33] and has been attributed to reduced mucus and HCO_3- production, decreased mucosal blood flow, neutrophil plugging of capillaries, and impaired healing.[34] The role of prostaglandins in phenylbutazone (PBZ) ulcers is unclear.[35–37] In adults and foals, PBZ has a greater ulcerogenic effect in the glandular mucosa and even the pyloric region than in the nonglandular mucosa,[33,35,36] whereas the nonglandular mucosa seems more susceptible to flunixin meglumine (FM) ulcers.[32] In a study on horses at necropsy, no difference was found between rate of gastric ulceration in horses treated with NSAIDs and those without.[10] This underscores the complex nature of gastric ulceration in horses and the many potential factors involved in any one case.

Because of the potential role of COX-2 in mucosal repair, COX-2 selective and nonselective NSAIDs could interfere with healing of gastric ulcers.[38] Both selective (firocoxib) and nonselective NSAIDs (PBZ) can induce gastric ulcers in both glandular and squamous portions but with greater severity in the glandular mucosa in horses treated with PBZ.[39] However, there is some controversy about the role of NSAIDs in the development of EGGD,[20] and PBZ can induce these ulcers in horses and ponies without decreasing basal glandular concentration of prostaglandin E_2.[37] Relevant to the more severe forms of colic, lipopolysaccharide (LPS) infusion can significantly increase the mean pH of gastric contents and decrease gastric concentration of H^+ and acid output compared with saline infusion.[40] These findings would suggest a protective effect from LPS against gastric mucosal injury that is mediated through overexpression of inducible COX-enhanced release of mucosal prostaglandin E_2.[40]

Risk Factors

The prevalence of ESGUS appears related to the intensity of exercise, which could explain the higher prevalence in Thoroughbred and Standardbred racehorses,[3,10] especially those actively racing and training.[9,13] Competing endurance horses also have a high prevalence (93%).[3] Although race training and racing could be risk factors for ESGD, EGGD does not appear to be associated with work intensity and duration in the same manner,[20] and ESGD is not limited to high-performance horses.[3] In a study on Thoroughbred broodmares at pasture, a 71% prevalence of gastric ulcers was recorded, mostly ESGD with a median score of 3.0/4.0, with no effect from pregnancy.[5] The role of stress from a severe GI or orthopedic disease in inducing gastric ulcers is difficult to assess,[15,23,41] although stress could increase the risk of EGGD.[2,3,16] In horses treated with abdominal pain, those with proximal enteritis had a higher prevalence of gastric ulceration than horses that had colic surgery.[42]

Intermittent feeding programs seem to put horses at greater risk of gastric ulcers compared with continuous feeding,[12,43–45] presumably because grazing/continuous feeding induces a continuous flow of saliva and food intake that buffers gastric acid ($pH \leq 4$) for most of the day.[4] Intermittent feed deprivation decreases proximal gastric pH in horses compared with fed horses throughout the day and evening.[29] In one study, however, pH was unchanged by paddock turnout compared with stall confinement.[28] The role of dietary management in horses with EGGD is controversial, and this disease can develop in horses under management conditions typically regarded as low risk.[20]

Feeding alfalfa hay with or without concentrates appears to be protective against gastric ulcers compared with horses fed other grass hay with or without grain and decreases the abdominal pain associated with EGUS.[3,46,47] Possibly, Ca^{++} and protein, both high in alfalfa hay, buffer gastric contents and thereby protect the nonglandular mucosa. However, weanlings fed alfalfa chaff ad libitum had an increased risk of EGGD in one study.[48] High-grain diets could produce volatile fatty acids during fermentation by gastric bacteria, which could contribute to gastric acidity.[4] Other risk factors include transport, oral hypertonic electrolyte solutions,[3] intermittent access to water, straw as the only forage, an interval > six hours between forage feeding,[49] and multiple handlers/riders.[50]

Clinical Signs

Gastric ulceration in adult horses can be clinically silent or cause highly variable and nonspecific clinical signs, such as poor appetite, weight loss, hair coat changes, poor performance, changes in attitude, depression, chronic diarrhea, behavioral changes, and colic.[1–4,51] Horses with EGGD are more likely to perform below expectations than those with ESGD.[2,3,52] In foals, clinical signs in severe cases include bruxism, salivation, dorsal recumbency, interrupted nursing, colic, diarrhea, and poor condition.[15,53,54] In one study, 92% of horses with clinical signs of gastric ulcers had diagnostic evidence on gastroscopy, whereas 52% without such signs had gastroscopic findings of ulcers.[53] These findings underscore the difficulty in establishing the role of EGUS as the cause of clinical disease. Perforating gastric ulcers are rare and can cause peritonitis, abscesses, and adhesions in adult horses and foals.[41,55,56]

Gastric Ulcers and Colic

Intermittent signs of mild colic, especially during and after eating, have been attributed to gastric ulcers.[4] Gastric ulcers should be considered a possible cause of idiopathic recurrent/chronic colic,[2] but signs can be vague and other differential diagnoses would need to be considered (Chapter 8). In one gastroscopy study, 55% of horses had signs of recurrent or acute colic.[51] In another, 82% of horses in race training had gastroscopic evidence of ulcers and 39% had signs consistent with gastric ulceration; however, colic was rare.[9] Gastric ulcers as the primary cause of abdominal discomfort were recorded in 28% of horses in another study.[57] Possibly severe ulcers are required to induce signs of colic (Figure 1.3).[51]

In one study, horses with DPJ seemed more disposed to gastric ulcers than those with other gastrointestinal lesions.[42] Horses that had medically responsive colic had significantly greater prevalence of gastric ulceration compared with horses that required surgical intervention, even when horses with DPJ were excluded.[42] The presence or absence of gastric ulcers was not associated with age,

breed, gender, duration of colic, NSAID use, or quantity of gastric reflux.[42] Horses with DPJ may be prone to gastric ulcers because they are not eating; they require repeated or continuous placement of a nasogastric tube, or the disease itself could induce gastritis and ulceration.[42] A significant association between gastric ulcers and diseases that cause colic has been demonstrated,[10] although a cause-and-effect relationship was not established.

Gastric ulcers were diagnosed by gastroscopy in 38% of horses after enterolith surgery in one study.[58] These were probably present before surgery, either secondary to the chronic or subclinical pain caused by the enterolith or the result of housing and environmental factors associated with the development of both diseases (Chapter 4).[58] It is noteworthy that the prevalence of gastric ulceration in horses with a primary surgical lesion is low[10,42,58] compared with reports on prevalence for the general equine population (see above). Also, most intestinal diseases that cause colic are acute, whereas gastric ulcers tend to be chronic.

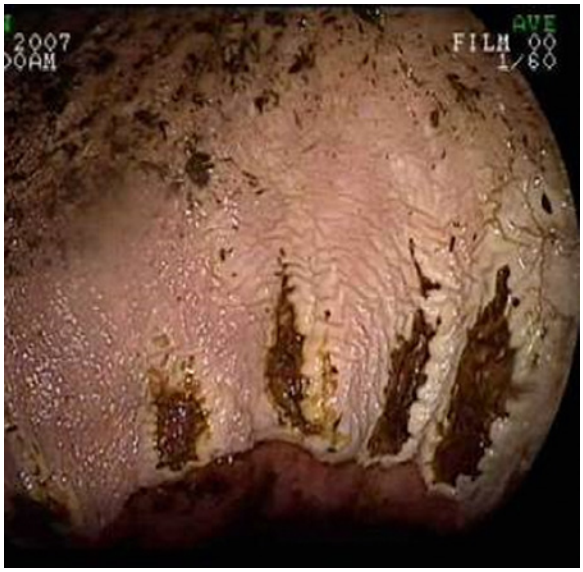

Figure 1.4 Endoscopic image of ESGD along the margo plicatus.

Diagnosis

Diagnosis is based on history, clinical signs, gastroscopic examination, elimination of other potential causes of colic, and response to treatment.[4,15] Gastroscopy is critical, allowing distinction between sites of mucosal injury (ESGD and EGGD), assessment of severity, and response to treatment.[2] Fasting before endoscopy could require up to 16 hours to ensure complete gastric emptying.[2] However, the duration of fasting for Thoroughbred racehorses on high-grain and low-roughage diets can be considerably shorter, with six to eight hours sufficient for complete gastric emptying (overnight and morning fast).[1] This improves owner and trainer compliance.[1]

The stomach is insufflated with air to improve access and to efface mucosal plications. The margo plicatus and the lesser curvature are examined closely, as these are common sites of squamous ulceration (Figure 1.4), and then the pyloric antrum and duodenum. The last two can be technically difficult to access (Figure 1.5), although most glandular ulcers arise in the pyloric antrum (Figure 1.6).[2] Hyperemia and similar mild changes in the glandular mucosa and thickened rugal folds should not be overinterpreted, whereas true lesions in this segment are evident as disruption of mucosal integrity.[2] Ulcers can be found in many parts and can be solitary or multiple, distinct or coalescing.[10,42] An ulcer scoring/grading system can allow correlation with clinical findings, monitoring of ulcer healing, and assessing the efficacy of treatment,[1,2,4,51] although supporting evidence of such a system for clinical relevance is weak.[3] Also, consistency between observers in assessing glandular lesions is good, whereas that recorded for

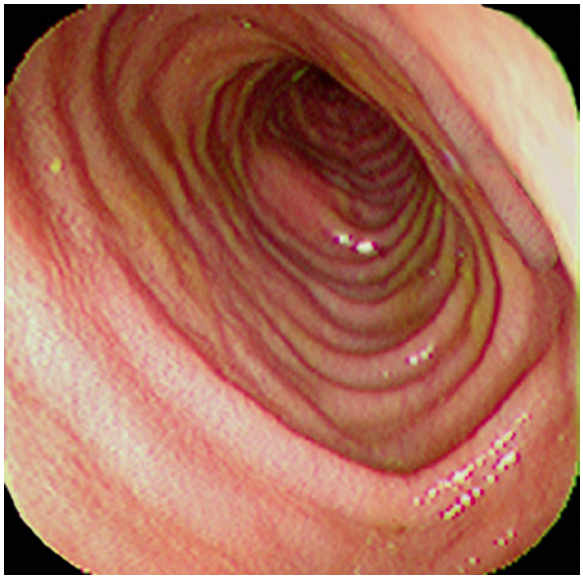

Figure 1.5 Normal endoscopic appearance of the duodenum, an important site of gastric outflow obstruction from ulceration and other causes. *Source:* Courtesy of Dr. Alfred M. Merritt.

nonglandular lesions is highly variable.[59,60] A grading system has not been developed for EGGD.[1] Lesions in the antrum can vary by degrees of ulceration (Figure 1.6), hemorrhage, hyperemia (Figure 1.7), and fibrinosuppuration and by a depressed, flat, or raised contour.[1]

Other tests that could support endoscopic findings are of limited value, such as assessment of anemia or hypoproteinemia.[4] The fecal occult blood test (FOBT), which uses specific equine monoclonal antibodies to albumin and

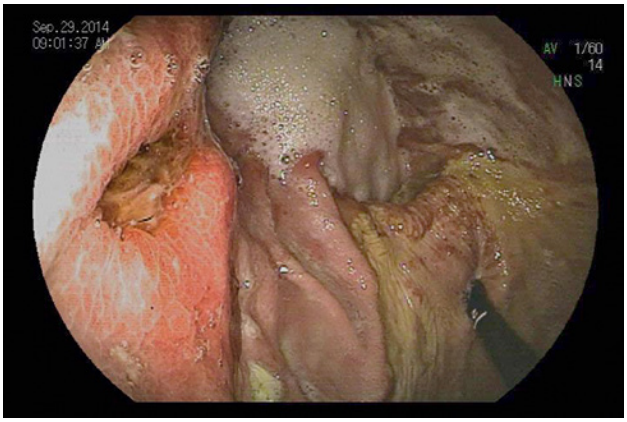

Figure 1.6 Endoscopic image of the cardia to the lower right and displaying EGGD in the glandular epithelium on the left. *Source:* Courtesy of Dr. Guy Lester.

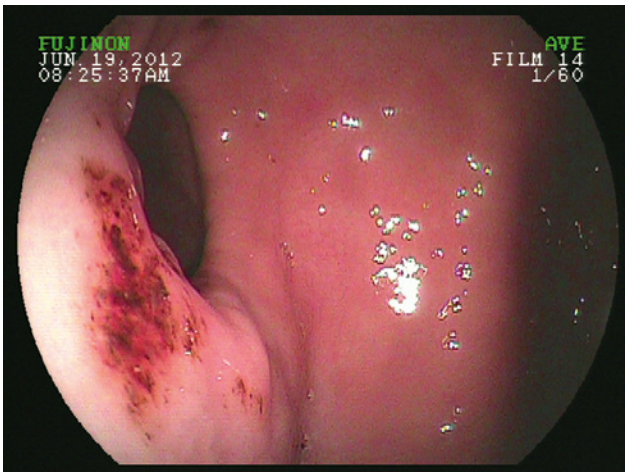

Figure 1.7 EGGD in the antrum. *Source:* Courtesy of Dr. Guy Lester.

hemoglobin in a simple commercially available kit, has poor specificity for EGUS[61] and is inferior to gastroscopy.[2] However, in one study, fecal albumin concentration was significantly associated with gastroscopic scores for glandular ulcers.[39] The sucrose permeability test was found to be neither sensitive nor specific for detecting EGUS in adult horses with naturally occurring disease.[62]

Treatment

Although equine ulcers can heal spontaneously, suppression of gastric acidity creates a suitable environment for repair and hastens the process.[4,13,14] The rate at which ulcers heal spontaneously can be reduced by antiulcer therapy (omeprazole),[13] although clinical improvement might be evident earlier.[59] A variety of drugs have been used to suppress gastric acidity and, in doing so, these drugs can also inhibit the acid-induced conversion of pepsinogen (the inactive zymogen) to the active pepsin (Figure 1.8). Antiulcer drugs might be required for several days to weeks, and some are required frequently per day,[63] which makes treatment expensive and compliance poor. Drugs that are available as a generic product at lower cost can be ineffective in such preparations.[64] Risk factors need to be identified if applicable and eliminated until clinical signs have resolved, including intensive training.[4] Protective management factors should be applied, such as turnout, feeding alfalfa hay, and rest (see above). Another important practical issue in treatment of gastric ulcers is identification of those ulcers that cause a clinical problem. Ulcer location is important, because the response to medical therapy is different between ESGD and EGGD.[16,20] If NSAIDs are implicated as the possible cause, they should be replaced with another form of treatment for pain (see next section).

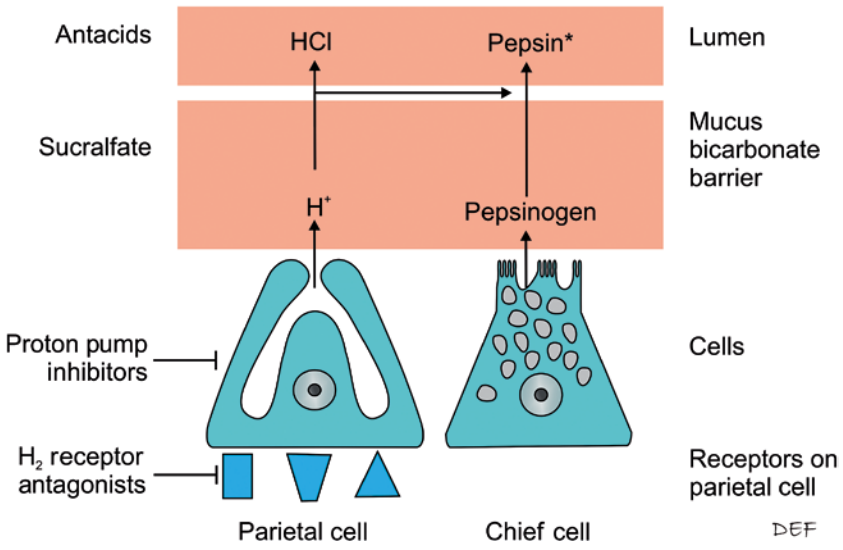

Figure 1.8 Anatomy of the gastric secretory cells, their products, and the effects of different methods of pharmacological management of gastric secretion. *Pepsin is converted from its zymogen, pepsinogen, by gastric acid in the lumen. Therefore, all inhibitors of lumen acidity indirectly block pepsin by inhibiting its formation.

Pain Management of Gastric Ulcers Any NSAID could be contraindicated to treat gastric ulcer pain because of their potential to exacerbate the disease (see above), but ketoprofen and firocoxib might be safer than FM (Chapter 14).[4,32,39] Xylazine or detomidine, with or without butorphanol, can also be used to control pain,[4] but a perceived need for these drugs might suggest a more acute and severe disease than gastric ulceration.

Proton Pump Inhibitors (PPIs) Omeprazole (GastroGard™, Merial Limited, Iselin, NJ; 4 mg/kg PO, SID) is a substituted benzimidazole that is converted to sulfenamide in the canaliculus of the parietal cell and binds irreversibly to the parietal cell H^+/K^+ ATPase (proton pump).[4] In this way, the period of acid suppression outlasts plasma concentration of this proton pump inhibitor (PPI), so it is suitable for once-daily use.[65] Gastric acid degrades omeprazole and reduces its bioavailability and, therefore, omeprazole is formulated in granules that release it at a high pH.[64] Because acid is required to convert the zymogen, pepsinogen, to the active pepsin, this process is also disrupted by omeprazole (Figure 1.8).

Oral omeprazole at lower doses can significantly decrease gastric acid secretion and increase gastric fluid pH.[66,67] It significantly improved healing rates in horses with gastric ulcers induced by NSAIDs[68,69] and in naturally occurring gastric ulcers in Thoroughbred racehorses.[13] In a large study on horses in race training, omeprazole was superior to H_2 antagonists in improving performance, weight gain, attitude, appetite, and appearance.[4]

Compounded preparations of omeprazole do not improve healing of ESGD compared with GastroGard, possibly because of vehicle-related problems.[64] Enteric-coated granules of omeprazole suspended in a paste might have some modest superiority over GastroGard in the treatment of ESGD.[3] Omeprazole for 28–35 days of treatment at 4 mg/kg daily had a weaker effect on healing of glandular ulcers (25% healing) than it had with ESGD (healing rate of 78%).[16,20] Type of diet and duration of fasting might improve the performance of omeprazole.[70,71] The intravenous formulation of omeprazole[72] and the intramuscular form[73] lack the adverse effects and bioavailability issues associated with oral omeprazole[74] and could be used in horses with dysphagia, gastric reflux, duodenitis-proximal jejunitis and other diseases that would restrict oral administration.[4] A long-acting IM omeprazole can suppress gastric acid secretion effectively over a seven-day period and with high rates of success in clinical cases.[74] In a small number of cases, it was 100% effective for healing in horses with ESGD and 75% in those with EGGD.[74] Esomeprazole appears to be superior to omeprazole and other PPIs in human meta-analysis studies and has been used PO and IV in horses with efficacy comparable to that of omeprazole.[75] One concern with long-term use of PPIs in human patients is rebound acid hypersecretion and gastrin-induced hyperplasia.[76] Whether or not these responses apply to horses is unknown, although cost could determine the duration of treatment before such complications develop.

H_2 Receptor Antagonists Ranitidine (Zantac, Glaxo Inc., Research Triangle Park, NC) and cimetidine are histamine type 2 (H_2) receptor antagonists that block the potent stimulation of acid secretion by H_2 binding to the receptor site on the parietal cell (Figure 1.8).[14] Ranitidine is considered superior to cimetidine for acid suppression by these drugs.[15] At 6.6 mg/kg, PO, TID, for periods of two to three weeks, ranitidine produced a clinical and endoscopic improvement in adult horses with spontaneous EGUS.[77] Ranitidine can also reduce the severity of gastric ulcers induced by intermittent feed deprivation.[43] Limitations with ranitidine are variable oral bioavailability,[14,59,78] less effective inhibition of total acid output compared with omeprazole,[79,80] and lack of owner compliance.[4]

Sucralfate Sucralfate (Carafate®, Marion Merrell Dow, Inc., Kansas City, MO) is a hydroxy aluminum salt of sucrose octasulfate that forms a sticky, viscous gel at pH <4.0.[59] This gel adheres firmly to the base of ulcers for hours and forms an acid-resistant layer that protects against acid and pepsin.[59] Sucralfate also preserves and thickens the mucus layer, stimulates mucus and HCO_3- production, enhances restitution, and stimulates prostaglandin synthesis.[59] In a study on spontaneous gastric ulcers in foals, sucralfate at 22 mg/kg, PO, q 8 hours, did not improve ulcer healing over 14 days, compared with corn syrup-treated controls.[81] Sucralfate could be used as an adjunctive treatment for EGGD along with omeprazole.[82]

Misoprostol Concurrent administration of a PG analog prevented the clinical signs, hypoproteinemia, and gastrointestinal mucosal atrophy, ulcers and edema induced by PBZ in ponies (4.4 mg PBZ/kg, PO, q 12 hours, for 10 days).[83] Misoprostol (PGE_1) is a synthetic analog of prostaglandin E_2 that has been shown to protect against gastric ulcers induced by NSAIDs in human beings[84] and dogs[85] and might be as effective as PGE_2 in promoting repair of injured mucosa.[86] Misoprostol can be recognized by E-prostanol (EP) receptors but only binds to three of them, whereas PGE_2 binds to all four EP receptors.[86] At 5 µg/kg PO, misoprostol suppressed acid secretion in horses over baseline for the eight-hour test period.[87] In a recent study, misoprostol (5 µg/kg PO TID) for two weeks improved healing of PBZ-induced glandular lesions in horses.[88] These findings would support the use of such analogs as

misoprostol in treating or preventing NSAID ulcers in the equine stomach (and in the right dorsal colon; Chapter 4). Misoprostol might be effective as part of combined therapy with omeprazole as first-line treatment of EGGD[88] and was found to be superior to combined omeprazole–sucralfate for this disease in one study.[52]

Recent evidence suggests that misoprostol is well tolerated and is rapidly absorbed in horses following oral administration of a single 5 μg/kg dose.[89] This dose did not significantly inhibit ex vivo LPS-stimulated TNFα mRNA production in leukocytes, suggesting lack of an anti-inflammatory effect.[89] One horse displayed signs of abdominal discomfort (flank watching, lying down), soft feces and depression with this dose, and others had soft feces without colic or changes in physical exam parameters.[90] A five-day regimen of twice daily misoprostol at 5 μg/kg bwt orally did not disrupt pregnancy, nor adversely affect the general health and comfort of mares in mid-gestation pregnancy.[90] An oral misoprostol regimen to mares in early gestation (37–85 days) days was also associated with a relatively low risk to the pregnancy or to foals from those pregnancies.[91] Possibly misoprostol has a weaker effect on contraction of urogenital smooth muscle compared with PGE_2.[86]

Antacids and Others In a trial that compared different antacids in clinically healthy horses, only 30 g aluminum hydroxide/15 g magnesium hydroxide (Maalox TC, Rhone-Poulenc Rorer Pharmaceuticals Inc., Fort Washington, PA) increased gastric pH compared with controls.[63] However, the duration of effect was short enough to require repeated administration daily. Also, the effect on ulcer healing is unknown,[63] and the dose of magnesium hydroxide required could be toxic.[92] Prokinetic drugs are recommended for foals with delayed gastric emptying secondary to gastroduodenal ulceration, reflux, and ileus (Chapter 42), and metoclopramide and bethanechol have been used for this purpose.[15] Because bacteria are not involved in the development of EGUS, antimicrobial drugs are not recommended.[88] Adjunctive treatments, such as pectin–lecithin-based nutraceuticals, could contribute to prevention and treatment of EGGD and ESGD treated with omeprazole and other PPIs.[16,20] Gastric surgery can be effective for foals and adult horses with perforated gastric ulcers (Chapter 22).[55,93]

Gastroduodenal Ulcer Disease (GDUD)

Gastroduodenal ulcer disease (GDUD) is an insidious disease in foals caused by progression of ulcers in the esophagus, stomach, pylorus, and duodenum to deeper and more chronic injuries that cause gastric outflow obstruction and signs of colic (Chapter 9).[54,94–97] Adult horses can develop similar changes, but rarely.[98] Gastroduodenoscopy (Figure 1.5) provides the most useful information about the lesion and location, and ultrasonography can identify the caudal extension of the stomach, nature of its contents, and the appearance of the duodenum (Chapter 12). A bypass procedure is required in many cases, and prognosis is determined by site of obstruction (Chapter 22).

Gastritis

Primary gastritis is uncommon in horses and is usually caused by ingestion of chemicals, such as arsenic, lead, copper, mercury, cantharadin, phosphorus and nitrates, or by licking or swallowing counterirritants applied to the forelimbs.[99] Dioctyl sodium sulfosuccinate (DSS), a laxative for impaction colic in horses, can cause gastritis if given in high concentrations or to horses with delayed gastric emptying.[100] Other documented causes are uremic gastritis caused by oxalate nephropathy,[101] chronic granulomatous gastroenteritis and chronic hypertrophic catarrhal gastritis in old horses.[102–104] Chronic parasitic gastritis from *Trichostrongylus axei* and *Gasterophilus* spp. infestation is rare under current management systems.[104]

Conditions that delay gastric emptying, cause prolonged gastric distension and stasis, or allow reflux of duodenal contents, bile, and pancreatic secretions into the gastric lumen can also damage the gastric lining.[105] The consequences of gastric stasis are bacterial overgrowth, which causes volatile fatty acid production and deconjugation or dehydroxylation of bile salts refluxed from the duodenum, all of which could damage the gastric mucosa.[4] These changes must be interpreted with due regard for the observation that reflux of duodenal contents into the stomach is probably common in horses, particularly during cessation of antral motility.[105]

Cantharadin/Blister Beatle Toxicity

A well-known form of gastritis in horses is caused by ingesting cantharidin in hemolymph from the male of three species of striped blister beetles (*Epicauta*) (Figure 1.9).[106,107] The beetles are incorporated into alfalfa hay, and five to six of them can cause gastritis and colic in an adult horse.[106,108] Cantharidin is absorbed through the gastrointestinal tract and skin and can cause severe inflammation along its route of excretion through the urinary system.[107] It disrupts cell membranes and causes inflammation, necrosis, ulceration, and erosions in the upper alimentary tract, and the excreted toxin causes severe urinary tract inflammation.[107]

Figure 1.9 Blister beetles found in alfalfa hay. *Source:* Courtesy of Dr. Robert J. MacKay.

Risk Factors

Modern methods of harvesting alfalfa, with cutting and crimping in a single operation, increase the risk of including a large number of beetles in individual bales or flakes.[107] Hay cut from field borders, between May and September, and as second and third cuttings, is at greatest risk of contamination because of coincidence with greatest beetle activity.[106,108] The risk is reduced by obtaining hay from a reputable source, spraying infested areas, and checking hay carefully, recognizing that beetle distribution in the crop will be very erratic. Cantharadin is heat stable and can survive the processing that produces dehydrated alfalfa pellets.[108] Blister beetle poisoning might be more common with hay harvested in Nebraska, Kansas, Colorado, Oklahoma, Texas, and Louisiana, but has been reported in most of the eastern parts of the United States.[107,108]

Clinical Signs

The clinical findings in horses suspected of blister beetle poisoning can be diagnostic but are not constant or specific.[107] Onset of signs can be rapid, and severely affected horses can die within a few days, usually with signs of severe shock.[107] The most consistent clinical signs in decreasing order of frequency are abdominal pain, fever, depression, elevated heart and respiratory rates, and congested mucous membranes with slow capillary refill time.[107] Sweating, fever, hypersalivation, intestinal hypomotility, soft feces, and bloody feces can develop, and affected horses can repeatedly splash their muzzles in water without drinking.[107] Oral mucosal lesions can develop but are not common. Signs related to the urinary tract can include frequent attempts to urinate, small volumes of urine, pain on urination, and hematuria, with obvious blood clots.[107]

Diagnosis

Diagnosis of cantharidin ingestion is based on clinical signs, recent alfalfa hay consumption, hay harvested in certain states (see above), dead blister beetles in the hay supply, and marked and sustained hypocalcemia and hypomagnesemia.[107] Horses become hemoconcentrated and develop leukocytosis, with increases in nonsegmented neutrophils in severely affected cases.[107] The BUN can be elevated during the early stages. Serum concentrations of most electrolytes are normal except for a severe hypocalcemia in some horses, which can correlate with severity of the disease.[107] Hypocalcemia can be responsible for the synchronous diaphragmatic flutter seen in some horses, but the mechanism by which cantharidin causes hypocalcemia is unknown.[107] Urine specific gravity can be low, and red blood cells can be found in the urine.[107]

At necropsy, the nonglandular epithelium can be sloughed over a large area and is easily detached.[107] Small intestinal changes range from mucoid or watery flocculent contents with some patchy pseudomembranous inflammation.[107] The wall of the small and large intestines can be grossly edematous and hemorrhagic and ulcers and hemorrhage can be found in the bladder.[107] Other changes include renal tubular necrosis and evidence of ventricular myocardial necrosis.[107]

Treatment

Recommended treatments include intravenous fluids to offset the effects of shock, activated charcoal by stomach tube, analgesics, and calcium borogluconate.[107,109] Although intragastric mineral oil has been recommended to solubilize cantharidin and facilitate its elimination,[107] recent studies in rats suggest that mineral oil increases cantharidin absorption, which could increase morbidity and fatality rates.[109]

Prognosis

Prognosis for cantharidin toxicosis is dose-related. Mortality is 50%, less than 10% of affected horses have neurologic signs, affected horses can die quite suddenly, and death is preceded by signs of severe shock.[106–108]

Gastric Obstruction and Stasis

Many of the mucosal diseases described above can cause gastric obstruction through a functional response to inflammation, or in some cases to physical obstruction from deep invasion of the inflammatory process (e.g. GDUD). The following are functional and physical obstructions without apparent predisposing mucosal injury.

Gastric Distention

Gastric distention typically develops secondary to small intestinal obstruction, or in association with ileus, DPJ, or grass sickness (Chapters 2 and 6). It can also be associated with large colon obstructions (Chapter 4).[110,111] Gastric distention can also be caused by aerophagia in cribbers.[99]

Horses can develop a primary gastric distention from consuming excessive amounts of readily fermentable carbohydrates, such as wheat, oats, and barley, or beets and freshly chopped grass.[99,103,104,110,112] A diverse bacterial community resides in the normal equine stomach, presumably organisms that can tolerate the acidic environment,[19] mostly gram-positive cocci, such as lactobacilli and other lactate-producing organisms.[112–114] Bacterial fermentation can explain the high rate of volatile fatty acid and lactate production in the stomach, especially after consumption of readily fermentable carbohydrates.[19,25] Such fermentation is supported by a large region of nonglandular epithelium that provides a locally stable pH, buffered by the high concentration of HCO_3- in saliva.[19,25,112] Volatile fatty acids produced by gastric fermentation could inhibit gastric emptying, thereby retaining gastric contents, with further bacterial fermentation, gas production, gastritis, and ulceration.[19] Most of the lactic acid produced from the stomach is absorbed in the small intestine, but some could escape and lead to cecal and colonic mucosal injury and laminitis.[115]

Clinical Signs

Gastric distention usually causes acute abdominal pain. Although the "dog-sitting" position has been regarded as pathognomonic, it is not (Figure 1.10).[99] Elevation of the left side of the rib cage has been described,[99] but is rare because the stomach is located so deep within the abdomen.[110] Teeth grinding and constant salivation can be observed in foals, and frothy, foul-smelling, green ingesta is regurgitated and expelled through the nostrils in some cases.[81,110,116] Spontaneous regurgitation through the nostrils is rare and provides some relief but can be associated with imminent gastric rupture.[99,116] Persistent spontaneous reflux through the nostrils can cause aspiration pneumonia.

Figure 1.10 Dog-sitting posture while rising from recumbency can be erroneously attributed to gastric or cranial abdominal pain. This rare posture can be seen in horses without colic (this horse) and with colic from various causes.

Diagnosis

Gastric distention can be diagnosed by reflux of large volumes of fluid and gas under pressure through a nasogastric tube, and this provides the horse some relief. The introduction of the nasogastric tube often meets with considerable resistance in the lower esophagus because of increased lower esophageal sphincter (LES) tone in response to increased intra-abdominal and intragastric pressure.[99] The volume of fluid removed can exceed the anatomical capacity reported (5–15 L) up to 60 L in an extreme case.[116] Because gastric dilation is typically secondary to another lesion, other diagnostic procedures are indicated to identify them (Chapter 12).

Treatment

Gastric distention with liquid and gas can be relieved through a nasogastric tube, but this is only temporary in cases of secondary gastric distention. Surgery is treatment of choice for any such disease or other physical obstruction to gastric outflow. Any associated pain and fluid, electrolyte, and acid–base imbalances should be treated appropriately (Chapters 14 and 15).

Gastric Impaction

Gastric impaction is a rare cause of colic in horses (1.4% of hospitalized horses in one study) but can present with acute, chronic, or recurrent colic in the presence or absence of other gastrointestinal diseases.[117–119] Affected horses can be of a wide age range and with no gender predisposition.[117–119] Two types of gastric impactions have been described in horses, one of which develops in an otherwise normal stomach and responds to treatment (usually medical) with low risk of recurrence (this section).[118] Feed material constitutes the impaction in these cases, rarely ingested foreign matter.[120] In the second type, an insidious and chronic disease develops, presumably because of alterations in gastric motility that allow high volumes of food material to accumulate and cause progressive gastric enlargement.[118,119] This form of chronic impaction has a guarded prognosis and will be

discussed separately. Possibly, both types constitute a single disease with a wide variation in severity at the time of diagnosis.

Pathogenesis

Some derangement of normal gastric motility probably predisposes to gastric impaction. When food is swallowed, the gastric fundus progressively relaxes through a vagally mediated response called "receptive relaxation."[121,122] As the stomach receives and stores food in the expanded fundus and body, the antrum triturates, mixes, and expels food through the pylorus.[23] During this process, simultaneous contractions of the terminal antrum and pylorus force some liquid into the proximal duodenum and deliver solid ingesta back into the gastric body for further digestion.[23] This process favors retention of solid material so that particulate matter is retained, even after 1.5 hours, whereas 75% of a liquid meal has left the stomach half an hour after intake.[123] Such retention can dehydrate normal gastric contents to the point that they achieve approximately 20% dry matter (personal observation). Presumably, some derangement in this process could increase retention of solid contents while eliminating liquid, thereby creating a gastric impaction.

The classification of gastric impactions as primary or secondary can be difficult.[117] Primary gastric impaction can be caused by normal dietary components or by ingesta that swells or forms a mass after consumption, such as persimmon seeds, mesquite beans, shavings for bedding, wheat, barley, and sugar beet pulp.[99] Possible predisposing causes are dental abnormalities, inadequate water consumption, rapid food intake, and abnormal gastric motility or secretion.[124–128] Foreign material is more likely to obstruct the small intestine (Chapter 2) than the stomach (see below).[120,129,130] Foals can form gastric and intestinal trichophytobezoars from ingested tail and mane hairs from horses and other animals.[120] Secondary impaction can be caused by liver disease, specifically from ragwort poisoning (*Senecio jacobaea*).[131,132] Gastric contents can become dehydrated and impacted secondary to severe intestinal obstruction, such as large intestinal impaction or displacement, or small intestinal strangulation.[117] Such secondary impactions can be incidental findings at surgery for the primary disease and usually do not require treatment at surgery. In one study, horses with gastric impaction associated with other intestinal abnormalities had more severe colic than those without concurrent lesions.[117]

Gastric impaction with persimmon seed phytobezoars (diospyrobezoars) in human beings and horses is caused by ingestion of the tropical fruit from *Diospyros virginiana*, which covers a broad range, but predominantly in the southeastern United States (Figure 1.11).[132–134] Horses are more likely to engorge on ripe persimmons in fall and early winter, and the water-soluble tannin in the fruit probably polymerizes in gastric acid to form an adhesive coagulum.[130,134] This combines with indigestible components, cellulose, hemicellulose, and lignin, with protein to form a solid impaction (Figure 1.12).[130,133–135] The seeds of the persimmon create an abrasive surface on the phytobezoar that causes gastric ulcerations and perforation.[133]

Figure 1.11 Fruit of the persimmon on the tree and ripened (top inset).

Clinical Signs

Gastric impaction can cause nonspecific clinical signs of long duration,[125,126,128,133,135] but some horses can have a more acute and severe clinical presentation.[124,136] Affected horses can have a normal attitude[125] or be dull, lethargic, or depressed,[125,126,130,135] and signs of colic can range from none to mild, with frequent periods of prolonged recumbency and inappetence.[125,126,130,133,137] Recurrent signs of colic were reported in 35% of horses in one study,[128] and a long-term history of recurrent colic with or without exercise intolerance has also been reported.[117] In one study of 13 horses with persimmon impaction, the most common signs were colic (7 of 13), anorexia, weight loss, and diarrhea.[132] The wide variability in signs could be attributed to

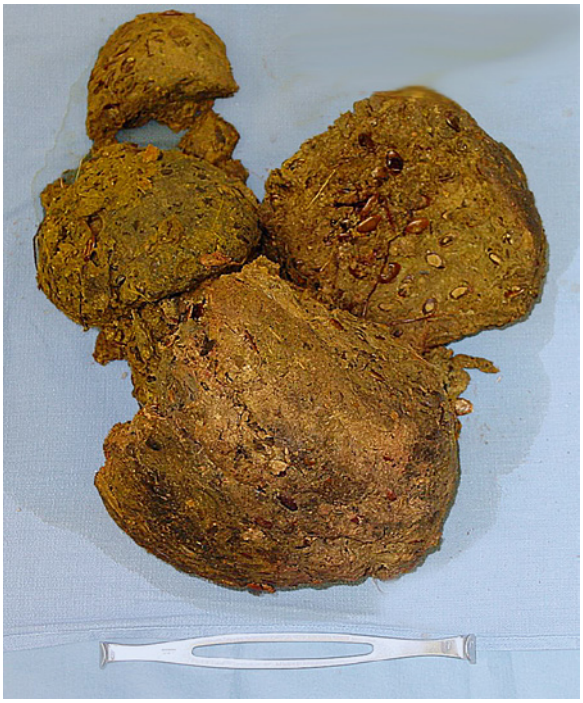

Figure 1.12 Gastric contents with obstruction caused by persimmon. Seeds can be seen scattered throughout the gastric contents. *Source:* Courtesy of Dr. Amanda House.

the impaction behaving as a valve, with a greater degree of antral and pyloric obstruction in horses with severe signs of colic, and milder and more protracted presentations if these segments are minimally obstructed.[132]

The admitting heart rate can range from 36 to 60 beats/min with a mean of 48 beats/min.[117] Intestinal sounds are reduced,[128] and fecal output is absent or scant.[125,126] Some affected horses might try to eat, but drop food from the mouth,[125,126] have frothy material at the mouth[126] or nostrils,[124] or fluid drainage from the nostrils.[130] Orange-tinged nasal drainage in a horse that had access to persimmon fruit is highly suggestive of this type of impaction.[134] A history of weight loss, with normal appetite, with or without colic,[132,134] has been reported. Weight loss can be attributed to reduced appetite, chronic pain, and intervals of fasting during colic episodes.[132] Another presentation is an overweight horse[135,138] or pony,[124–126,135] suggesting that a voracious appetite can contribute to this disease.

Diagnosis

History of access to the persimmon fruit (Figure 1.11) is an important clue. A diagnosis of primary gastric impaction can be difficult because of the highly variable clinical signs and absence of diagnostic features on rectal palpation, and abdominocentesis. Diagnosis also requires evidence that other primary gastrointestinal abnormalities are absent, sometimes accomplished at exploratory celiotomy. Confusion with choke can arise because of similar clinical signs in some acute cases.

Gastroscopy is used with care to avoid overdiagnosis.[139] A stomach full of ingesta on gastroscopy is not consistently diagnostic of impaction, even after a 24-hour fasting period,[110] and gastric distention is difficult to assess by this method.[127] Even a 16-hour fast might not allow gastric emptying in all horses, and rate of gastric emptying could be diet-[121,140] and fasting-related.[141] Reflux through a nasogastric tube is rare in most cases[125,126,136] with the exception of diospyrobezoar impaction,[129,132,134] and gastric contents are more likely to be dehydrated, as in normal horses.[140]

Time of year and known access to or witnessed consumption of persimmon fruit should prompt a gastroduodenoscopic examination.[132] Vigorous irrigation of the impacted mass with water might be required to expose the seed mass beneath other material on gastroscopy (Figure 1.13).[129] The seeds are oblong, flat, and brown, measure approximately 1.5 cm long,[129] and can be seen on masses of food material and on the gastric mucosa.[130] Gastroscopic examination can also demonstrate an associated hyperkeratotic squamous epithelium[129] and gastric ulcers, either primary or secondary to mucosal injury from an abrasive bezoar. Duodenoscopy should also be performed to identify any bezoars impacted in the duodenum.[132,142] Unlike other types of phytobezoars, diospyrobezoars can also obstruct the small intestine with

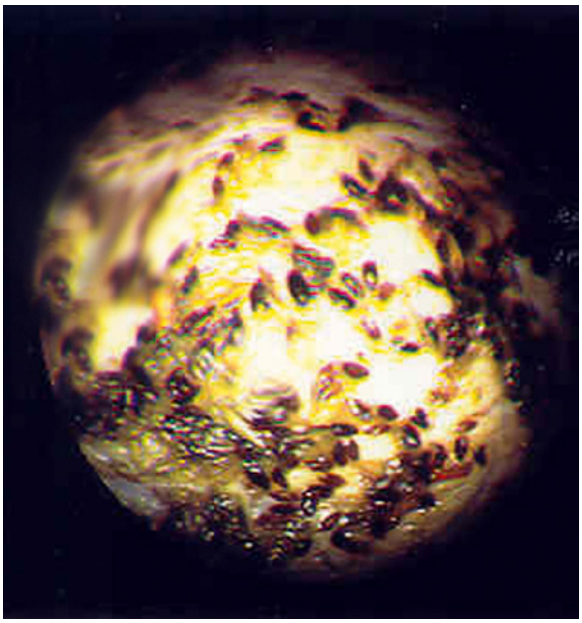

Figure 1.13 Endoscopic image of persimmon seeds on gastric contents. *Source:* Courtesy of Dr. Amanda House.

signs referable to that site (Chapter 2).[129] Horses can develop hypochloremia, possibly caused by decreased enteral absorption of Cl−.[132]

On palpation per rectum, a gastric impaction can be felt in some cases,[126] with caudal medial displacement of the spleen.[117,136] On ultrasound examination, the stomach can be evident over a larger area than normal,[117] with a subjectively thinned wall,[128] and the spleen might be displaced ventrally and caudally.[136] Diagnosis is made in some horses by exploratory celiotomy in the absence of a preoperative diagnosis.

Treatment – Medical

Medical treatment for gastric impactions from normal dietary components involves feed deprivation, analgesics, intravenous fluids, and repeated gastric lavage with water and electrolytes (Chapter 17) through a stomach tube (2–10 L each time, 1–8 times daily [median of 5 times]).[128] Impactions caused by diospyrobezoars and trichophytobezoars are difficult to resolve by lavage because the components are bound within the substance of the mass. Response to medical treatment should be closely followed by gastroduodenoscopy.[132] If a solid component of a gastric impaction breaks off a persimmon obstruction, it could obstruct the small intestine.[129] Endoscopic methods and use of forceps intended to fragment impactions into smaller components could also lead to small intestinal obstruction, especially if the impaction includes foreign material or hair.

Although usually used for treatment of diospyrobezoars, carbonated cola soft drinks of the regular, diet, or caffeine-free diet (preferred) varieties can be effective in treating other types of gastric bezoars in human patients.[133,143] However, the efficacy in horses for any type of gastric impaction is difficult to determine.[132] Presumably, sodium bicarbonate is mucolytic, and the carbon dioxide bubbles can physically disrupt phytobezoar fibers.[132] Also, carbonic and phosphoric acid in colas produce pH ranges from 2.3 to 2.5, which could contribute to break down of phytobezoars.[132,133] Based on the human dose, a horse should receive volumes of 20–24 L a day of decaffeinated cola.[132] Treatments can require oral administration of 355 mL to 2 L of cola every 12 hours for 8 weeks, mixed in the feed, or by intermittent intragastric infusion of 700 mL every 12 hours for 3 days.[132] Intragastric constant rate infusion has also been used (1 L of cola/h for one to three days).[132] Caffeine toxicosis and laminitis from an excess of nonstructural carbohydrates can be prevented by using caffeine-free diet colas.

Other potential intragastric treatments include pancreatic enzymes, papain, cellulase, mineral oil, sodium bicarbonate,[133] and acetylcysteine.[132] Intragastric mucosal protectants and antiulcer medications (see above) are used to prevent ulceration from the disease and its treatments.[132] Mineral oil might bypass a gastric impaction,[125,129] without permeating it. As the impaction resolves with medical treatment, pelleted feed or soft mashes can be offered, with gradual increase in hay.[117,132] Because gastric impaction causes anorexia and requires protracted feed deprivation, affected horses should be monitored and treated for hyperlipidemia as needed (Chapter 44), especially ponies and overweight horses.[137]

Treatment – Surgical

Failure of medical treatment could indicate the need for exploratory surgery to remove the gastric impaction (Chapter 22). Gastric impaction secondary to small intestinal obstruction usually resolves without treatment, after surgical correction of the primary small intestinal disease and appropriate intravenous fluid therapy. However, anastomotic impaction[144,145] or postoperative gastric rupture[117] can follow in such cases and prompt the concern that medical treatment of the impaction should be considered postoperatively, based on surgeon's judgment of severity.

Prognosis

Regardless of cause, a primary gastric impaction (no other predisposing digestive tract disease) can require protracted and aggressive medical treatment. Time from hospital admission to endoscopic resolution of obstruction with diospyrobezoars in one report ranged from 3 to 84 days, with a median of 24 days and a survival rate of 62%.[132] With impactions from dietary components, resolution with medical treatment required one to five days (median two days) in one study[128] and two days to two weeks in another.[117] More deaths from diospyrobezoars are caused by enteric rather than gastric impaction, probably because of a more complete obstruction with resulting mucosal damage when not treated promptly.[132] Other causes of death in affected horses can include recurrence of impaction and spontaneous gastric rupture. Gastric impactions amenable to medical treatment can have a better prognosis than those treated by surgery.[132]

Chronic Gastric Impaction

A rare massive and chronic form of gastric impaction has been reported in Europe, with growing evidence that the same disease, or possibly a milder version, occurs in the United States (Figure 1.14). It could also be the same disease described above for impaction with feedstuff, but later in its course, possibly because of an early misdiagnosis or gradual development of gastroparesis. The stomach can achieve considerable size over a long period of time before causing any apparent clinical signs or interfering with performance.[118,119] Affected horses have a mean age of eight

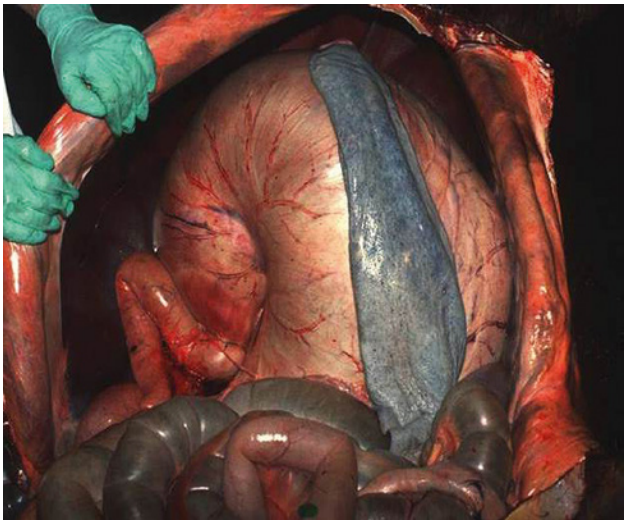

Figure 1.14 Enormously enlarged stomach from chronic impaction. Note the stomach occupies most of the cranial part of the abdomen. *Source:* Courtesy of Dr. Wolfgang Scheidemann.

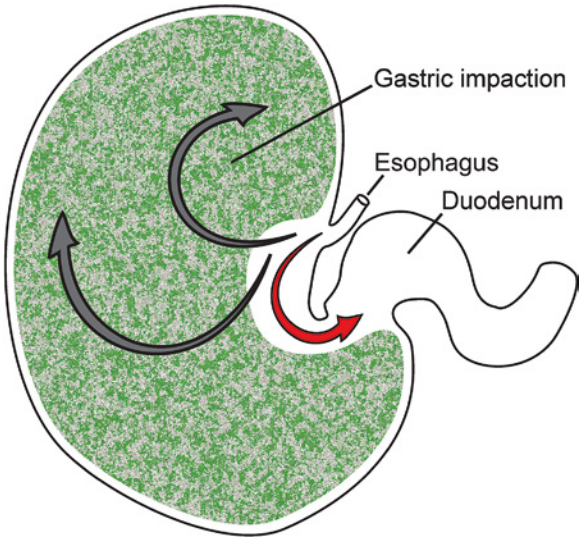

Figure 1.15 Solid ingesta is retained in the gastric fundus (gray arrows) while more liquid contents pass along the lesser curvature into the duodenum (red arrow).

years and with no gender predisposition.[118] Larger breeds tend to be over-represented,[118] such as draft breed horses, Friesians and warmblood horses. Some affected horses develop secondary intestinal obstruction and colic that necessitate referral.[118,119]

Pathologic Changes

As this chronic impaction slowly develops, it enlarges considerably along the greater curvature, while liquid contents seem to pass unimpeded along the lesser curvature to the duodenum (Figure 1.15).[119] Accompanying gastric changes include large chronic ulcers and adhesions to the

Figure 1.16 Chronic gastric impaction at necropsy displaying the intensely dehydrated and impacted material in the open stomach. *Source:* Courtesy of Dr. Wolfgang Scheidemann.

diaphragm, spleen, and left body wall,[110,118] possibly caused by transmural inflammation from the ulcers.[119] The ulcers are probably secondary to the impaction. The stomach can approach 80 cm in diameter,[110] and contents can weigh between 15 and 122 kg,[118,119,136] compared with 8 kg in unaffected horses. Fundic contents are usually foul-smelling, fermenting material, with recently ingested material along the lesser curvature between the cardia and pylorus.[110,118] The impaction is dry and hard and packed in layers or laminations (Figure 1.16).[119]

The fundic wall undergoes transmural muscular hypertrophy ranging in thickness from 10 mm to 35 mm,[110,117,118] compared with ≤5 mm in normal horses.[117] Focal fibrosis of the stomach wall and focal myositis are the main histological findings, and thickening can be found rarely in some horses in the distal esophagus or pylorus.[117] Of the three incomplete layers of the stomach wall, the internal oblique fibers, which are arranged in two layers, are predominantly affected.[117] Muscular hypertrophy of sections of large colon and small pale liver in some horses can be attributed to pressure from the enlarged stomach on adjacent viscera.[118] Gastric rupture is regarded as a rare complication (see below), despite the tension applied by the impaction.[119] The lack of pain despite severe distention of the gastric fundus has been attributed to damage to the intrinsic nerve supply of the fundus.[118] Also, the large-horse breeds at risk tend to be stoic, which would allow them to develop a large impaction over time with a low clinical response.

Clinical Signs

Clinical signs include mild intermittent colic for months, despite normal appetite and defecation and successful participation in athletic events during this time.[110] Overt signs

of colic are not a consistent feature of this disease, although recurrent colic episodes can be seen postprandially.[118] Other signs are bruxism and salivation.[118] Attentive owners might notice the horse burp, which is accompanied by a sour smell from the nostrils.[119] In one successful dressage horse, gastric enlargement was followed over four years before clinical signs developed.[119] Body condition can be good, possibly because liquid nutrients can bypass the impaction (Figure 1.15), and cardiovascular parameters are normal at admission, as are intestinal sounds and pattern of defecation.[110] A history of insidious weight loss over two years has also been reported.[110] Performance eventually declines.[118] In general, gastric obstructive diseases cause a variety of unusual clinical signs, some of which are identified in hindsight. Secondary gastric rupture causes dramatic clinical manifestations of septic shock (see below).

Diagnosis

Diagnosis starts with history of a slowly progressing disease in a large-breed horse, without overt colic in the early stages.[118] If large enough, the convex caudal portion of the impacted stomach and its smooth contents can be palpated per rectum cranial to the caudally displaced spleen.[110,119] Spasmolytic agents might be required if deep palpation is necessary (Chapter 12). A large impaction can also elevate the left thoracic wall to create a distinct bulge on the left side compared to the right.[119] Passage of a nasogastric tube encounters resistance against a hard mass so the tube cannot be fully advanced.[120] Lavage of impacted contents fails to remove much material.[119] Ultrasonography is the most helpful diagnostic method, with additional information provided by gastroscopy and radiography. The dilated stomach can be seen cranially as far as the 8th or 7th intercostal space and caudally to the 14th and 15th spaces with ultrasound examination.[119] On gastroscopy, the stomach remains full after starvation, and food particles can be seen adhering to the fundic mucosa.[119]

Treatment and Prognosis

Treatments that are effective with acute cases of gastric impaction are less successful with this disease, which earns it a guarded prognosis.[118] Gastric lavage at best provides temporary reduction in impaction size.[119] Although surgical evacuation is possible (Chapter 22), continued gastroparesis and permanent expansion of the gastric wall can lead to reimpaction.[118,119] Partial resection of the atonic stomach has been reported but was unsuccessful.[119] Successful resolution and complete recovery have been reported after gastrotomy.[136]

Neoplasia and Miscellaneous Masses

Gastric neoplasia is rare in horses, with squamous-cell carcinoma (SCC) in the nonglandular portion of the stomach the most common type.[110] Affected horses usually have a vague history and clinical signs and are older than 6 years (mean of 10.7 years).[110,146] Rarely, the tumor involves the glandular mucosa and produces squamous metaplasia.[147] Gastric carcinoma produces large, cauliflower-like masses that occupy the lumen to varying degrees.[147] Metastasis produces neoplastic nodules on the parietal peritoneum, liver, spleen, adrenal glands, kidneys, mesenteric and other lymph nodes, lungs, mesentery, omentum, diaphragm, pleura, pericardium, and testes.[147,148] Other gastric tumors include adenocarcinoma of the glandular portion, leiomyoma, leiomyosarcoma, mesothelioma, and lymphosarcoma.[146–149]

A pyloric mass composed of a central connective tissue core and an extremely hyperplastic pyloric glandular region with a heavy cellular infiltrate caused intermittent signs of colic, progressive weight loss, and gastric reflux in a 12-year-old stallion.[150] A large pedunculated mass identified as an inflammatory polyp was the likely cause of pyloric obstruction and associated large gastric impaction in a 13-year-old Percheron gelding.[151] In an adult mare with recurrent colic, then sudden uncontrollable colic, a large mass composed of adenomatous polypoid gastric hyperplasia from the glandular portion of the stomach formed the leading edge of a 20-cm-long intussusception of the pylorus into the duodenum.[152] A similar gastric hyperplastic polyp caused a pyloric outflow obstruction in another horse but without intussusception.[153] A large mass of granulation tissue (10.5 kg) attached to the squamous epithelial lining of the stomach in a four-year-old stallion caused chronic anorexia, weight loss, diarrhea, ventral edema, and malabsorption, without an apparent effect on gastric emptying.[154] Presumably, a sense of "fullness" from such a large intragastric mass caused inappetence.[154] A similar mass emanating from the greater curvature of the stomach and proposed to be secondary to a penetrating gastric injury caused recurrent colic then acute colic in a mature mare.[155]

Clinical Signs and Diagnosis

In one study, abdominal pain was reported in 21% of horses with gastric neoplasia caused by SCC, mesothelioma, and leiomyoma, and was attributed to impaired gastric outflow and/or abdominal metastasis.[148] However, for SCC, anorexia, depression, and weight loss over two to six weeks are the most consistent clinical findings.[156] Ptyalism, difficulty in eating and drinking, and difficult passage of a nasogastric tube are evident if the distal esophagus is involved.[148] Anemia and low-grade fever are common,[146] and anemia can be attributed to hemorrhage from the tumor or to depressed erythrogenesis. Hypercalcemia of malignancy has also been reported as a component of the paraneoplastic syndrome with this disease.[148] On palpation per rectum, intra-abdominal metastases can be identified as scattered nodules on the serosal surfaces of viscera and on the

peritoneum. Abdominocentesis can yield normal fluid or fluid with single or clumped exfoliated squamous cells.[157] The tumor can be seen on gastroscopy,[158] and ultrasonographic examination of the left cranial abdomen can demonstrate thickening and abnormal echogenicity of the stomach wall.[156] Euthanasia is indicated upon diagnosis. Gastric masses that block gastric outflow obstruction can cause intermittent or acute colic that could be diagnosed by gastroscopy, ultrasound examination, or surgery.

Pyloric Stenosis

Obstructive pyloric diseases are rare causes of gastric outflow obstruction that affect neonatal foals (Chapter 9) to horses of any age.[159–163] Acquired pyloric stenosis is usually secondary to gastritis, gastric ulceration, and neoplasia and is the focus of this section as a cause of gastric outflow obstruction (see Chapter 9 for foals).

In a multicenter study in Europe, 47 horses were identified with a chronic severe pyloric stenosis, with an age range of 0.8–16 years (median age 3 years) and mostly warmbloods of any gender.[162] These represented 0.07% of all cases admitted to one hospital over a six-year period.[163] The most common presenting signs were poor body condition, slow eating, selective appetite,[163] frequent recumbency, and poor performance.[163] Recurrent colic was reported in 74%[162] of cases, and hypoalbuminemia was the most common laboratory abnormality. Physical examination findings were highly variable.[163] Gastroscopy and/or ultrasonography demonstrated slow gastric emptying, and endoscopy revealed ulcers in the terminal portion of the esophagus.[162] All horses had severe squamous ulceration and pyloric lesions that were inflamed, ulcerated, and coated with fibrin.[162] One horse also had perforation of a duodenal ulcer.[163] Response to treatment with antiulcer medication was satisfactory in most cases, although recurrence was high and only 41% of horses survived long-term.[162] Four horses required a gastrojejunostomy.[162] Horses ≤3 years of age had a poorer prognosis than older horses.[162]

Foreign Bodies

Horses are unlikely to ingest foreign bodies, although pieces of wood, stick (Figure 1.17), shavings from bedding, bone, needles, stones, carpet, and bezoars have been found in the horse's stomach.[19,120] Fragments of broken nasogastric tubes have been removed from the stomach (Figure 1.18),[164] although endoscopic methods should be attempted before gastrotomy (Chapter 22). Young horses can ingest strands of cord that unravel from rubberized fencing in sufficient quantities to cause large intestinal obstruction (Chapter 4) and obstruction in the distal portion of the stomach.[165] This obstruction can cause

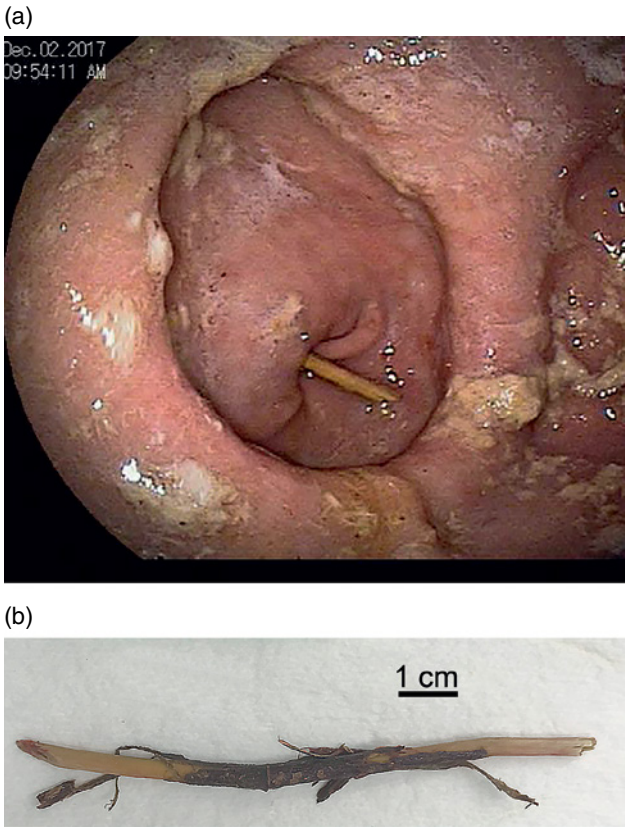

Figure 1.17 (a) Gastroscopic image demonstrating a stick passing through the pylorus. (b) Same stick removed by gastroduodenoscopy. The end to the left had punctured the stomach near the pylorus and caused mild peritonitis.

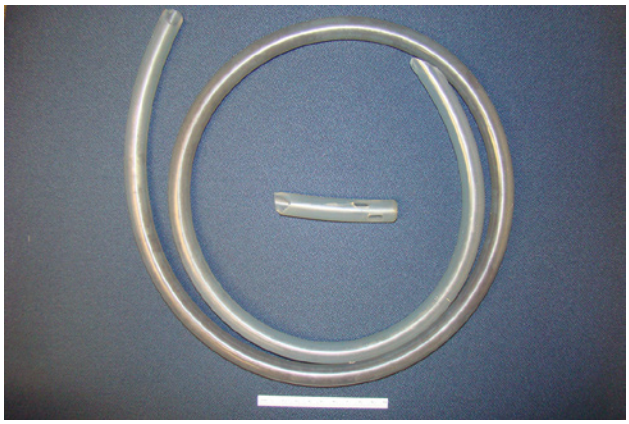

Figure 1.18 Fragments of nasogastric tube removed by gastrotomy.

anorexia, weight loss, lethargy, intermittent, acute abdominal pain and an abnormal glucose-absorption test from the delayed gastric emptying.[165] A gastric trichophytobezoar was removed successfully by gastrotomy in an eight-week-old foal,[120] and a perforating briar of a blackthorn plant (*Prunus spinosa* spp.) in the pylorus was removed by gastrotomy in a mature horse.[166] Ingested wire can

perforate the stomach wall and lodge in the spleen and other sites to cause multiple abscesses and peritonitis (Chapter 6).

Gastric Rupture

Gastric rupture can follow any form of primary or secondary gastric obstruction and is the reason that such obstructions should receive prompt attention. The reported prevalence of gastric rupture in horses is 1–6% of necropsy findings, 3–5% of colic cases,[110,167–169] and 11% of deaths following exploratory celiotomy for colic.[170] Gastric rupture is a common type of ruptured viscus in horses, approximately three times more common than cecal rupture.[171]

Primary or idiopathic rupture refers to cases in which no predisposing cause can be identified,[119] and accounts for 17–60% of horses with gastric rupture.[110,169,170] It can be associated with grain overload, ulcerative gastritis, or feeding by nasogastric tube.[170] Secondary gastric ruptures are more common than primary and are caused by small intestinal obstruction (especially in proximal segments), proximal enteritis, grass sickness, or postoperative ileus.[110] Secondary gastric rupture is rare, presumably because of prompt recognition and effective decompression by nasogastric tube. A predisposing mural weakness is not required, except in the rare gastric ulcers that rupture in foals.[55,56] Gastric bezoars are rare causes of gastric rupture.[172]

Pathogenesis

A popular explanation for the horse's apparent predisposition to gastric rupture is failure to relieve gastric pressure through vomition, an act considered impossible in the horse because of the oblique entry of the esophagus into the stomach[173] and an overly powerful cardia[110] or LES. This explanation is based on cadaver studies, which demonstrated that the equine cadaver stomach would rupture under pressure from injection of fluid or gas through the pylorus, apparently because the cardia prevents escape into the esophagus and relief from the distention.[174,175] However, cadaver studies inform little about such a complex physiological event as vomiting in the live animal. The LES pressure reported for the horse *in vivo* is similar to that recorded for species that can vomit,[176,177] which eliminates an extremely powerful LES as the reason for failure to vomit. A more plausible reason is revealed by examining the vomiting reflex, which would be absent in a cadaver study. This requires an intact nervous system, responses from the chemoreceptor trigger zone and emetic center, and orderly sequence of specific motility events in the LES, gastric fundus, and duodenum, combined with abdominal contractions.[23] Pharmacological studies have demonstrated that the vomiting center in the horse may be poorly developed or have a higher threshold to noxious stimuli than in other species.[178–180] On a teleological basis, vomiting probably has no role in normal digestive function of the horse.

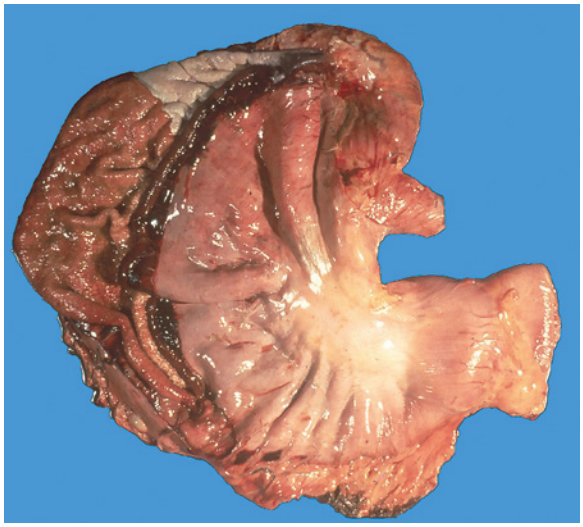

Figure 1.19 Typical appearance of a gastric rupture along the greater curvature with hemorrhage along the torn edges, indicating an antemortem tear.

In most horses, the gastric rupture is along the greater curvature (Figure 1.19), with rarer sites between the greater and lesser curvature, on the lesser curvature,[170] or transversely.[119,181,182] Antemortem ruptures can be recognized by hemorrhage along the margins of the tear[169,182] with a longer tear in the seromuscular layer than in the mucosa (Figure 1.19).[119,170] In one study, all gastric ruptures in foals less than three months old were associated with gastric ulcers, in which seromuscular tears did not extend beyond the limits of the mucosal tear.[170] In most cases, the rupture is immediately full thickness, although a biphasic rupture has been described, in which the mucosa tears hours or even days after the serosal tear.[99,119] These can be amenable to surgical repair,[181] but difficulty in diagnosis can deny this opportunity.[119]

Risk Factors

In earlier studies, age, breed, and gender risk factors were not identified for gastric rupture, although geldings might have been over-represented and stallions underrepresented.[170] A more recent study has identified a significant breed association with Friesian and draft breeds with primary gastric rupture compared with the colic population.[180] In the same comparison, Quarter Horses were underrepresented, but Thoroughbred horses were