

# Overview of Equine Gastroduodenal Ulceration

Michael J. Murray, DVM, MS, Dipl. ACVIM

Just as the term colic describes a clinical presentation and encompasses a large number of disorders, gastroduodenal ulceration describes a clinical finding, the cause of which is likely to be multifactorial and different from case to case. Within the umbrella term gastroduodenal ulceration are included symptomatic and asymptomatic cases, focal or multifocal ulceration involving the squamous or glandular mucosal linings of the stomach, gastritis, gastric emptying disorders, duodenitis, duodenal ulceration, and complications resulting from these disorders. Author's address: Marion duPont Scott Equine Medical Center, Virginia-Maryland Regional College of Veterinary Medicine, Leesburg, VA 20177. © 1997 AAEP.

## 1. Introduction

Gastric ulceration affects large numbers of foals, yearlings, and adult horses, and different clinical syndromes and lesion distributions occur in each group. Gastric ulceration may occur as a primary problem, or it may occur secondary to another intestinal disorder. Duodenal ulceration occurs primarily in foals, although it has been diagnosed in yearlings. Duodenal ulceration is an uncommon finding in adult horses.

## 2. Pathogenesis

In consideration of possible pathogenic mechanisms, the anatomic location of the ulcer must be taken into account. Lesions in the gastric squamous mucosa result primarily from excessive acidity, whereas gastric glandular lesions result primarily from defective mucosal protection. In general, ulceration is considered to result from an imbalance of aggressive and protective factors. The principal relevant aggressive factors are hydrochloric acid and pepsin, whereas relevant protective factors include the mucus-bicarbonate barrier, prostaglandin E<sub>2</sub> (PGE<sub>2</sub>),

mucosal blood flow, cellular restitution, and growth factors that promote angiogenesis and mucosal proliferation. Gastric motility also is important, because delayed gastric emptying and prolonged gastric contractions have been implicated in the pathogenesis of ulcers. Mucosal protection of the duodenum relies on an alkaline or neutral pH, with factors such as PGE<sub>2</sub> and mucosal blood flow of probable relevance.

The squamous mucosa of the equine stomach lacks a mucus-bicarbonate layer, and it has minimal resistance to exposure to hydrochloric acid.<sup>1,2</sup> The gastric glandular mucosa comprises multiple cell types and functions and has elaborate mechanisms for protecting against peptic injury.<sup>3</sup> Horses secrete acid even when not eating, and gastric pH can fall below 2.0 soon after a horse stops eating.<sup>4,5</sup> Twenty-four-hour gastric acidity was significantly less in horses with hay available compared with horses deprived of feed.<sup>5</sup> Intermittent periods of feed deprivation can induce damage to the gastric squamous epithelium within hours to days,<sup>2</sup> as a result of increased gastric acidity. Bleeding gastric squamous mucosal ulcers can develop in as short as 48 h.

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## NOTES

Concentrate feeding may contribute to ulcers by increasing serum gastrin levels<sup>6</sup> (and presumably acid secretion), by reducing the horse's roughage intake, and, most importantly, by reducing the amount of time the horse spends eating. Thus, feeding management may play a pivotal role in the pathophysiology of gastric ulcers in horses. In fact, we have found that horses that are turned out onto pasture full time typically have no gastric lesions.

The high prevalence (50%) of gastric ulceration, particularly in the squamous mucosa, in young foals may be associated with gastric developmental changes that occur in the first days and weeks of life.<sup>1,7</sup> At birth the equine gastric squamous epithelium is thin and not highly keratinized.<sup>1</sup> Within days the mucosa becomes hyperplastic and parakeratotic. Desquamation of the squamous epithelium can be observed endoscopically in the first month of life in foals. Histologically, desquamation appears to involve separation of the superficial cornified epithelial layers. Increasing gastric acidity temporally parallels the proliferation of gastric squamous epithelium, with minimal acidity during the first few days of life and marked acidity present by 7–14 days. It is possible that the developing epithelium is less resistant to acid than more mature gastric squamous epithelium, thus predisposing it to peptic injury.

Illness appears to be a risk factor for foals developing glandular mucosal ulcers, because foals that were sick or had a painful musculoskeletal condition had a greater prevalence of glandular lesions compared with normal foals.<sup>8</sup> The lesions presumably are associated with stress, and people in intensive care units are known to be at high risk of developing gastric ulcers. The precise mechanism of stress ulceration is not known, but decreased mucosal blood flow is probably a primary factor. Physiologic stress associated with illness should be differentiated from psychological stress, which is popularly believed to be associated with peptic disease in people. In fact, such stresses are not correlated with peptic lesions in people, although psychologic stress may be associated with symptoms of dyspepsia.<sup>9</sup>

Much attention has been directed toward *Helicobacter pylori* as a cause of peptic disease in people.<sup>10,11</sup> *H. pylori* is now generally accepted as being the principal cause of peptic ulceration and gastritis in people. The organism is found on the surface of the glandular mucosa, just beneath the mucus layer. Infection with *H. pylori* induces an inflammatory response, particularly in the gastric antrum, and clinical signs are thought to usually develop months to years after infection. There is no evidence to date of *H. pylori* or related *Helicobacter* spp. infection in horses. Most gastric lesions in horses occur in the squamous mucosa, and *Helicobacter* spp. do not colonize alimentary squamous epithelium. Most lesions in the glandular mucosa of foals and adult horses are not associated with a prominent inflamma-

tory response, such as occurs with *H. pylori* infection in people.

### 3. Foals

#### A. Ulcer Syndromes

Gastric lesions occur in approximately 50% of foals in the first month of life.<sup>7</sup> Most lesions are erosions in the squamous epithelium adjacent to the margo plicatus (Fig. 1). These lesions heal spontaneously in most foals, but they can become more severe and lead to clinical problems. Young foals appear to be more susceptible to glandular mucosal erosion than older animals (Fig. 2), presumably because of immature gastric mucosal defenses. I found that as many as 40% of foals developed glandular mucosal erosions in the first 3 weeks of life. Most lesions healed spontaneously and no clinical signs were

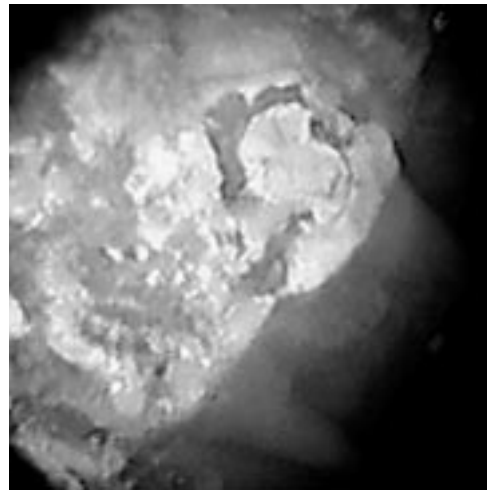


Fig. 1. Endoscopic view of gastric squamous epithelium in a 5-day-old normal foal; there is multifocal erosion of the gastric squamous mucosa with some flaking (desquamation) of the superficial epithelium.

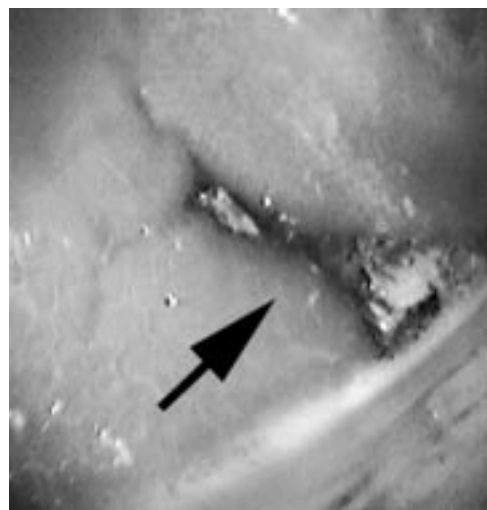


Fig. 2. Ulceration (arrow) in the gastric glandular mucosa of a 3-month-old foal that presented with depression and intermittent abdominal discomfort.

noted. After 2–3 months of age, lesion prevalence decreases, but lesions tend to be more severe and are more likely to be associated with clinical signs. Importantly, foals that are sick are more likely to have gastric ulcers than normal foals. I have confirmed this on several occasions, and I have found prophylactic acid suppressive treatment to be effective in these foals.

Duodenal lesions occur in foals of all ages, but typically the most severe clinical cases are seen in foals that are close to weaning age. The prevalence and cause of duodenal ulceration in foals have not been determined. From owners' accounts, it appears that the onset of disease can be rapid, with devastating consequences occurring within hours to a few days. Lesions occur primarily in the proximal duodenum and range from diffuse inflammation to focal, bleeding ulcers. Gastric ulceration frequently occurs secondary to duodenal ulceration, as a result of physiologic or anatomic obstruction to gastric emptying, and tends to be severe, often leading to gastroesophageal reflux and esophagitis.

Gastric and duodenal ulcers in young foals (<1 month old) may result in significant blood loss, resulting in anemia and hypoproteinemia. Perforation is a dramatic although infrequent sequela to gastric ulceration. In many cases perforation is not preceded by signs typical of gastric ulceration, and foals are found acutely depressed or dead. Most foals presented with perforation have significant peritonitis, which can have a tremendous fibrinous component. In such cases it is possible for peritoneal fluid cell count and protein to be normal, because of sequestration of cells and protein in fibrin clots within the omentum. Careful inspection of a Wright's or gram-stained slide for bacteria may confirm a perforated viscus. A small perforation along the greater curvature of the stomach or in the duodenal ampulla can be sealed by the greater omentum. Foals with perforated ulcers will be febrile and often will have signs of shock. In general, the sequelae to duodenal ulceration are more severe than gastric ulceration. These include severe gastric emptying dysfunction, duodenal perforation with peritonitis or adhesions, duodenal stricture with complete or partial obstruction, and ascending cholangitis and hepatitis.

#### B. Clinical Signs

Clinical signs vary from no signs to severe colic. Most young foals with gastric lesions have no clinical signs. Diarrhea was the most frequent clinical sign associated with gastric lesions,<sup>12</sup> and in many foals, treatment with an H<sub>2</sub> antagonist drug leads to resolution of diarrhea within 24 h. The classic clinical signs include bruxism, dorsal recumbence, salivation, interrupted nursing, and colic. These signs, though, are observed in the minority of foals with ulcers, and they usually are reflective of severe gastric lesions. Signs of salivation or esophageal reflux are indicative of gastric outlet obstruction or pseudo-obstruction, reflecting significant ulceration

associated with the pylorus or duodenum. Thus, observation of the classic signs associated with gastroduodenal ulceration in foals should alert the veterinarian to a serious ulcer condition that requires aggressive treatment.

#### 4. Yearlings and Adult Horses

##### A. Ulcer Syndromes

Normal yearlings not in work have no gastric lesions or only very mild erosions of the squamous mucosa. Adult horses of all breeds and uses develop erosion and ulceration in the gastric squamous epithelium (Fig. 3). Ulcers develop, to one degree or another, in all horses. However, in most cases, healing processes begin simultaneously with damage to the gastric mucosa, such that lesions heal quickly and do not progress to a clinical problem. Factors that appear to predispose to gastric lesions include eating behavior and feeding management, intensity of training, and concurrent illness. Horses in race training have a very high prevalence of gastric lesions (up to 90%), and the severity of lesions can worsen as training progresses.<sup>13</sup> Lesions in the glandular mucosa occur less frequently than in the squamous mucosa, although in one group of horses at a racetrack, 50% had glandular lesions (almost all healed spontaneously).<sup>13</sup> With the acquisition of a 3-m-long endoscope, I have found a relatively high incidence of lesions in and around the pylorus in horses presented with problems attributable to gastric ulceration. Horses with colic for reasons other than gastric ulceration have a high incidence of developing gastric ulceration.<sup>14</sup> Contributing factors include not eating and probably impaired gastric emptying in many cases. The administration of excessive doses of nonsteroidal anti-inflammatory drugs can cause gastric ulceration, but the routine administration of these drugs is not associated with gastric lesion development.

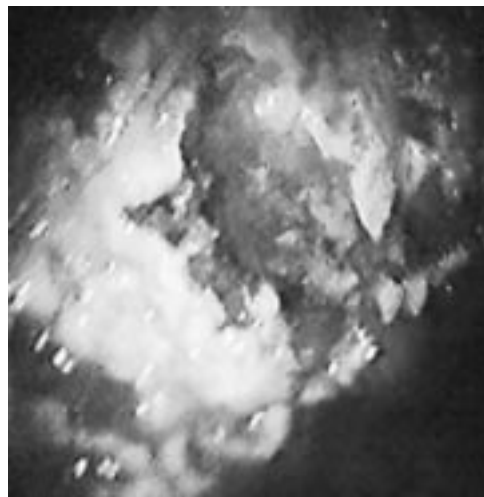


Fig. 3. Deep ulceration in the gastric squamous mucosa of a 4-year-old Thoroughbred race horse in training; no clinical problems were reported for this horse.

B. Clinical Signs

Clinical signs include poor appetite, poor condition, mild to severe colic, attitude changes, and poor racing performance. These signs, some of which are obscure or subjective, have been confirmed as being associated with gastric ulcers based on the results of endoscopic examinations and effective treatment with H<sub>2</sub> antagonists. Also, gastric lesions were significantly more prevalent and more severe in symptomatic horses compared with those in normal horses.<sup>15</sup> Bruxism and diarrhea are not features of gastric ulceration in adult horses, unlike in foals.

5. Diagnosis

Endoscopy is the best method of diagnosis. An endoscopic diagnosis of gastric ulceration allows the veterinarian to determine that ulcers are present, the location of the lesions, the severity of the lesions, and the response to therapy. Except in young foals, fecal occult blood is not a useful indicator of gastric lesions, nor are other laboratory findings. If characteristic clinical signs are present, response to treatment may be used to increase suspicion of gastroduodenal ulceration. Response to treatment usually is rapid (24–48 h for signs of colic and appetite). However, this rapid response can incorrectly lead an owner or trainer to assume that the ulcers have been healed.

6. Treatment

The primary objective in the treatment of gastric ulcers in foals and horses is to reduce or neutralize acid secretion so that the gastric mucosal epithelium can heal. Once ulcers form, there are changes in the tissue that promote healing. Suppressing acidity creates an environment within the stomach that is permissive for ulcer healing. Gastric acid secretion can be largely attenuated by use of histamine receptor type 2 (H<sub>2</sub>) antagonists. Treatment with H<sub>2</sub> antagonists has been successful in resolving the gastric lesions and in resolving the presenting problem.<sup>16</sup> Cimetidine and ranitidine<sup>a</sup> are the most frequently used, and both inhibit gastric acid secretion in equids.

Many dosages of H<sub>2</sub> antagonists have been recommended and used in practice. Because these drugs are expensive, there is pressure to use as little as possible. When deciding on a dose to use, one must recognize that as the dose of an acid-suppressive agent is lowered the percent of patients that will respond poorly or not at all increases. There is tremendous individual variability in the degree and duration of suppression of gastric acidity by H<sub>2</sub> antagonists between horses,<sup>5</sup> presumably as a result of differences in drug absorption and first-pass hepatic metabolism. In Fig. 4, two horses were administered 6.6 mg ranitidine per kg body weight per nasogastric intubation, and pH measurements were made on aspirated gastric fluid at 15-min intervals, 60 min before and 45–360 min after administration of ranitidine. Horse B had complete suppression of

gastric acidity for 360 min, whereas Horse A had intermittent, brief periods of increased gastric fluid pH. I have found that ranitidine 6.6 mg/kg PO q 8 h provides adequate suppression of acidity in the greatest percentage of horses. This dosage schedule resulted in a median 24-h gastric pH of 4.6 in horses with free access to hay, compared with a pH of 3.1 in horses with free access to hay but not given ranitidine. Effective dosages for cimetidine have not been critically evaluated, but very favorable pricing relative to ranitidine has prompted me to use cimetidine with greater frequency. I typically recommend 20 mg/kg PO q 8 h.

Formulations for the intravenous administration of H<sub>2</sub> antagonists are available but can be expensive. Ranitidine should be given intravenously at 1.5 mg/kg three times daily and cimetidine should be given at 6.6 mg/kg three to four times daily.

Recently, the H<sub>2</sub> antagonist drugs have become available as over-the-counter medications, and this has been accompanied by aggressive marketing by the pharmaceutical companies. Also, cimetidine has become available as a generic product, and consequently its cost has decreased considerably. For instance, at the time of manuscript preparation, in a 500-kg horse the daily cost of oral generic cimetidine (20 mg/kg q 8 h) from a national chain pharmacy cost \$21, and my cost for injectable generic cimetidine (6.6 mg/kg q 8 h) was \$40. Ranitidine in the prescription form (300-mg tablets) given orally at 6.6 mg/kg q 8 h costs approximately \$90/day in a 500-kg horse. By comparison, the daily cost in the same horse of the over-the-counter form of ranitidine<sup>b</sup> (75 mg/tablet) was \$36! With these and other developments that will improve the availability and cost of drugs that effectively reduce gastric acidity in horses, further research and critical examination of treatment effects of these medications will be required to ensure that rational treatment recommendations keep pace with the marketplace.

Other H<sub>2</sub> antagonists, famotidine<sup>c</sup> and nizatidine,<sup>d</sup> are on the market for use in humans, but effective dosages in the equine have not been established at

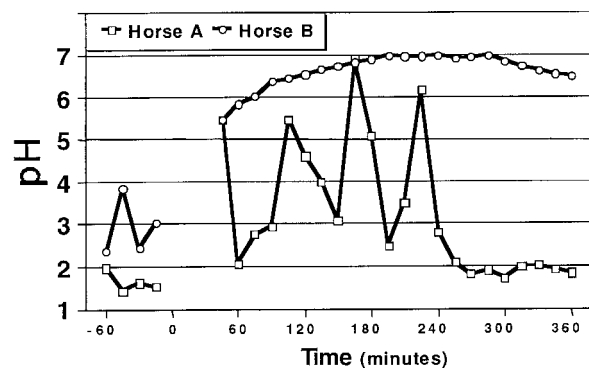


Fig. 4. Graph of gastric fluid pH from two horses illustrating the marked interhorse variability in response to oral H<sub>2</sub> receptor antagonists. (Courtesy of W.B. Saunders, *Current Therapy in Equine Medicine*, 1997;191–196.)

**Table 1. Comparative Prices for Acid-Suppressive Drugs or Antacids Recommended for Treatment of a 500-kg Horse with Gastric Ulcers<sup>a</sup>**

Drug (size)	Dosage	Approximate Daily Cost (\$)
Cimetidine, generic (800 mg)	20 mg/kg q 8 h	21 <sup>b</sup>
Ranitidine (75 mg)	6.6 mg/kg q 8 h	36 <sup>c</sup>
Omeprazole (20 mg)	1.0 mg/kg q 24 h	84 <sup>b</sup>
Maalox TC (12 oz. bottle)	240 ml (8 oz.) q 4 h	32 <sup>b</sup>

<sup>a</sup>Prices will vary by region and pharmacy. Depending on the severity and location of ulcers in a given horse, I may recommend greater dosages or frequency of administration than listed here. Note that in a given horse, the acid-suppressive effects of these drugs at the dosages given may, in fact, be dissimilar.

<sup>b</sup>Prescription price from a national chain store pharmacy.

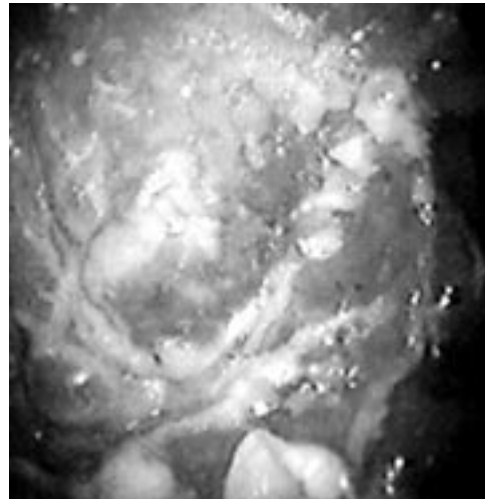
<sup>c</sup>Over-the-counter product.

this time. From limited experience, it would appear that the effect on gastric acidity from the oral administration of famotidine 3.3 mg/kg is similar to that with ranitidine 6.6 mg/kg.

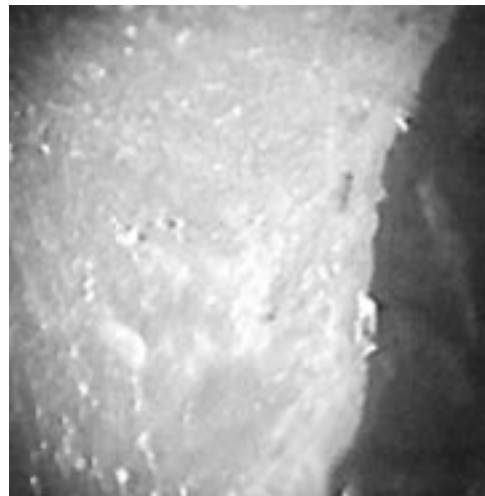
H<sub>2</sub> antagonist therapy should continue for 14–21 days to ensure complete healing. In most cases, 3 weeks of treatment is required to achieve complete healing. Eighty to ninety percent of adult horses treated with ranitidine 6.6 mg/kg q 8 h for 3 weeks had complete healing of gastric ulcers, whereas at 2 weeks complete healing occurred in only 15–40%.<sup>16</sup> It has become apparent from treating horses in training for racing with H<sub>2</sub> antagonists that if the horse is kept in training while being treated, clinical signs may improve but the lesions do not. Thus, for healing of the ulcers to be achieved, treatment with an H<sub>2</sub> antagonist should be accompanied by refraining from training.

Omeprazole<sup>e</sup> is a potent inhibitor of gastric acidity in horses.<sup>17,18</sup> The drug blocks gastric acid secretion by irreversibly inhibiting the parietal cell hydrogen ion pump,<sup>19</sup> and depending on the dose used, once-daily treatment is feasible. Studies in horses have shown that 0.7 mg/kg effectively inhibited gastric acidity, although 1.4 mg/kg was superior.<sup>17,18</sup> Administration of omeprazole 1.5 mg/kg, once daily by nasogastric tube, promoted the rapid restoration of normal gastric squamous mucosa in a vehicle-controlled study of Thoroughbred race horses with moderate to severe gastric ulceration (Fig. 5).<sup>20</sup> Omeprazole currently is marketed only as a drug for humans and is supplied as enteric coated granules. This is required because the drug is destroyed in an acidic environment, but this makes the drug difficult to administer to horses. I prefer to administer omeprazole granules by nasogastric tube. Because omeprazole in its human formulation is more expensive than H<sub>2</sub> antagonists and is more difficult to administer, I currently reserve its use for patients with severe gastroduodenal ulceration.

Neutralization of gastric HCl is achieved by the use of antacid compounds that typically contain salts, such as calcium carbonate, aluminum hydroxide, or magnesium hydroxide. The use of antacids in the treatment of gastric ulcers has been examined



(a)



(b)

**Fig. 5.** (a) Extensive ulceration in the squamous mucosa along the greater curvature of the stomach of a 4-year-old Thoroughbred racehorse. (b) Healed ulceration after 14 days of treatment with omeprazole 1.5 mg/kg q 24 h by nasogastric intubation.

in the equine to a limited degree. At a dose of 180 ml of an over-the-counter liquid antacid (7.2 g magnesium hydroxide/8.1 g aluminum hydroxide),<sup>f</sup> gastric pH was greater than 4.0 for only 30–45 min in one group of horses.<sup>21</sup> In another report, 250 ml of another product (15 g magnesium hydroxide/30 g aluminum hydroxide)<sup>g</sup> resulted in increased gastric pH for up to 2 h in horses.<sup>22</sup> Antacids must be given both in large volumes (≥240 ml) and very frequently (four to six times daily) to be effective even in alleviating clinical signs. Antacids may be particularly useful in horses that have evidence of excessive gastric acidity (moderate to severe hyperkeratosis of the gastric squamous mucosa), but no gastric ulceration. Mucosal (glandular) protective effects of aluminum-containing antacids, independently of their effect on gastric pH, have been recognized. These

effects may be mediated through prostaglandins, nitric oxide, and submucosal afferent neurons.<sup>23</sup>

Sucralfate<sup>h</sup> is a sulfated polysaccharide and is effective in the treatment of peptic ulcers in people. The mechanism of action likely involves adherence to ulcerated mucosa, stimulation of mucus secretion, binding of salivary epidermal growth factor, and enhanced PGE synthesis. These are all factors relevant to glandular mucosa, and it is doubtful that sucralfate is effective in treating ulcers in the equine gastric squamous mucosa. I have had horses develop squamous mucosal lesions while being treated with sucralfate for glandular ulcers. Sucralfate appears to be effective in the treatment of ulcers in the gastric glandular mucosa and duodenum in equids. When treatment is initiated without an endoscopic examination, sucralfate should be given as an adjunct to H<sub>2</sub> antagonist therapy, rather than the sole treatment. Effective dosages of sucralfate have not been determined in horses, although 10–20 mg/kg q 8 h has appeared to be effective in clinical cases.

Prokinetic drugs stimulate gastrointestinal motility and enhance gastric emptying. They are used in cases in which delayed gastric emptying is suspected or confirmed. I prefer bethanecol,<sup>i</sup> a cholinergic agonist. In horses, bethanecol was reported to enhance gastrointestinal motility while not increasing gastric acid output<sup>24</sup> and to increase gastric emptying in normal horses.<sup>25</sup> In cases of acute gastric atony, 0.025 mg/kg SQ q 4–6 h has been effective in promoting gastric motility and emptying. Oral maintenance dosages of 0.35 mg/kg three to four times daily are effective. Diarrhea has been observed infrequently with the higher dosages, but this resolved when the dosage was decreased. I have administered the drug chronically (months) to a few horses, with no apparent adverse effects.

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<sup>a</sup>Zantac, Glaxo, Inc., Research Triangle Park, NC 27709.  
<sup>b</sup>Zantac 75, Glaxo, Inc., Research Triangle Park, NC 27709.  
<sup>c</sup>Pepcid, Merck & Co., Inc., Rahway, NJ 07065.  
<sup>d</sup>Axid, Eli Lilly, Inc., Indianapolis, IN 27709.  
<sup>e</sup>Prilosec, Astra/Merck Group, Merck & Co., Inc., Rahway, NJ 07065.  
<sup>f</sup>Maalox, Rhone-Poulenc Rorer Pharmaceuticals, Collegeville, PA 19426-0851.  
<sup>g</sup>Maalox TC, Rhone-Poulenc Rorer Pharmaceuticals, Collegeville, PA 19426-0851.  
<sup>h</sup>Carafate, Marion Laboratories, Kansas City, MO 64137.  
<sup>i</sup>Urecholine, Merck & Co., Inc., West Point, PA 19466.