Tutorial Article

Recommendations for the diagnosis and treatment of equine gastric ulcer syndrome (EGUS)

THE EQUINE GASTRIC ULCER COUNCIL

Introduction

This article was derived from a document produced to provide a consensus of current opinion on treating the equine patient with gastrointestinal ulcers. The contents were compiled and ratified in 1998 by the Equine Gastric Ulcer Council, whose membership includes clinical specialists with a particular interest in this area of disease management. The following veterinarians authored this manuscript: F. Andrews, W. Bernard, D. Byars, N. Cohen, T. Divers, C. MacAllister, A. McGladdery, A. Merritt, M. Murray, J. Orsini, J. Snyder and N. Vatistas.

Gastrointestinal ulcers – definition

A gastrointestinal ulcer is defined as an alteration of the gastrointestinal mucosa that destroys cellular elements, resulting in a defect that could extend to the level of the lamina propria. Less severe disruptions are referred to as erosions and frequently are precursors to clinical ulcers (Table 1). Ulcers:

- Vary in severity
- Are induced by multiple aetiologies
- Have diverse distribution patterns (although they predominate in the squamous mucosa adjacent to the margo plicatus*)
- Are treated with a variety of therapeutic medications.

Diagnosis

Clinical signs

The diagnosis of equine gastric ulcer syndrome (EGUS) is confounded by the fact that there is not a well defined (pathognomonic) set of associated clinical signs. Furthermore, there is some inconsistency in the relationship between the presence of ulcer lesions, their severity and the manifestation of clinical signs. The following clinical signs are considered compatible with the presence of ulcers.

Foals

In neonates and some older foals, the following signs have been associated with ulcers:

- Intermittent colic
- Frequent dorsal recumbency

- Intermittent nursing (interruption of feeding, presumably due to discomfort)
- Diarrhoea and/or history of diarrhoea
- Poor appetite
- Bruxism* (grinding of teeth)
- Ptyalism (salivation).*

*These extreme signs are often associated with gastric outflow dysfunction as a result of duodenal ulceration.

Older individuals

Signs include:

- Poor appetite/failure to completely consume a meal
- Dullness
- Attitude change
- Decrease in performance/reluctance to train
- Poor body condition/rough hair coat/weight loss
- Low-grade colic or excessive recumbency.

The presumptive diagnosis of EGUS is based typically on nonspecific clinical signs and response to therapy.

Clinical pathology

There have been numerous attempts to find biochemical correlates to indicate the presence of gastrointestinal ulcers but currently, there are no known laboratory markers for EGUS. Attempts to evaluate faecal material for the presence of occult blood have been unreliable in the horse.

Endoscopic evaluation

Endoscopy (Fig 1) is currently the only reliable method for confirming a diagnosis of EGUS. Specifications for endoscopic equipment are related to the type of practice, caseload, whether the equipment will reside within a clinic or be in ambulatory care, equipment costs, and the interests of the practice owners. Endoscopic equipment can be grouped into 2 categories: fibreoptic and video.

Flexible endoscopes are available from several manufacturers in various lengths and diameters. For mature equine gastric endoscopy, a minimum working length of 200 cm is required. However, a 280–300 cm long...
endoscope is required to perform duodenoscopy in mature individuals. A working length of 110 cm with an outer diameter of 10 mm (human gastroscope) is sufficient to reach the stomach of foals up to age 30–40 days. 

**Fibreoptic endoscopic equipment** uses glass-fibre bundles to transmit light to the area to be viewed and transmit this image to an eyepiece. The image is magnified by a lens system within the eyepiece. This is important in alimentary endoscopy, since 150 W lamps used in most portable light sources provide poor illumination of a horse's stomach. More powerful light sources are available (up to 300 W) but become larger and less portable as the lamp intensity increases. The quality of the fibreoptic instrument is determined largely by its image resolution, which is related directly to the number of optical fibres. 

**Videoendoscopic systems** use glass-fibre bundles to transmit light, but use a charge-coupled device (CCD) chip to transmit the image. Generation of the light source (300 W) and processing of the CCD-generated electronic signal occurs in the endoscope's processor.

**Assessment of severity**

Assigning a score or grade to a gastric lesion in order to characterise its severity facilitates the selection of treatment modalities and helps clinicians determine the anticipated duration of treatment required for lesion healing. The EGUS Council has adopted a lesion grading system that:

- Is simple and straightforward
- Can be applied to the squamous and glandular mucosal linings of the equine stomach
- Can be used by individual practitioners and researchers
- Is similar to other grading systems used to characterise clinical severity of other body, systems (e.g. lameness, neurological disorders, heart murmurs).

**Lesion grading system**

*Note that no mention of bleeding is made in assigning lesion grades, because bleeding does not determine lesion severity. Small superficial erosions (Fig 3) may bleed, whereas deep ulcers may not have active haemorrhage at the time of endoscopic examination.*

**Current concepts of therapeutic options**

Before describing the medications currently used to treat equine gastric ulcers (Fig 20), it is useful to review the treatment options for peptic ulcers in man. For more information on gastric physiology (such as mechanisms of acid production), please refer to physiology.

**Gastrointestinal ulceration** remains a considerable health problem in man worldwide. In 1910 the phrase 'no acid, no ulcer' was coined (Schwartz), which led to strategies designed to suppress acid production. In the 1940s, researchers discovered that inhibiting the transmission of vagal impulses significantly benefited patients suffering from ulcerative disease. The 1970s heralded the era of blocking acid production at the cellular level through the use of histamine receptor antagonists (H2 blockers). The most recently developed anti-ulcer medications are the so-called proton-pump inhibitors, which inactivate the intracellular H+K+ATPase pump. 

Proton-pump inhibitors have demonstrated enhanced acid suppression and relief for patients. Finally, the discovery of *H. pylori* and its major role in the course of human ulcer disease, has significantly influenced the development of
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E = oesophagus; LC = lesser curvature; GC = greater curvature; D = duodenum

**Fig 1:** Schematic representation of the endoscopic path as it courses through the stomach towards the intestines.

**Fig 2:** Endoscopic image of a normal stomach. Junction of glandular region (GL) and nonglandular region (SQ) referred to as margo plicatus (MP). Note: Gastric contents (GC).

**Fig 3:** Numerous erosions in the squamous mucosa extend from the margo plicatus (MP) into the fundus.

**Fig 4:** Ulcer grade 0. Normal-appearing squamous mucosa along the lesser curvature of the stomach. The margo plicatus (MP) is the junction of the squamous and glandular mucosa.

**Fig 5:** Ulcer grade 1. The squamous mucosa is characterised by yellow appearing hyperkeratosis, which is a response to excessive acidity.

**Fig 6:** Ulcer grade 2. There are small superficial erosions (black arrow) in the squamous mucosa adjacent to the margo plicatus.
Table 3: Cell types in gastric glands

<table>
<thead>
<tr>
<th>Cell type</th>
<th>Substance released</th>
</tr>
</thead>
<tbody>
<tr>
<td>Parietal cell</td>
<td>Hydrochloric acid (HCl)</td>
</tr>
<tr>
<td>Chief cell</td>
<td>Pepsinogen (converted to the proteolytic enzyme pepsin)</td>
</tr>
<tr>
<td>D cell</td>
<td>Somatostatin (inhibits gastrin release)</td>
</tr>
<tr>
<td>Mast cell</td>
<td>Histamine</td>
</tr>
<tr>
<td>Enterochromaffin-like cell (ECL)</td>
<td>Histamine, Serotonin</td>
</tr>
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Antacids

Antacids, such as magnesium/aluminum hydroxide have been used but must be administered frequently (up to 6 to 12 times/day). The therapeutic or prophylactic efficacy has not been critically tested in horses. Furthermore, the number of treatments required would probably be impractical to administer and potentially stressful.

Mucosal protectants

Sucralfate, a complex salt of sucrose and aluminum hydroxide, has been used successfully to treat gastric and duodenal ulcers in man. Its main protective action is through adherence to the ulcerated surface. The aluminum and magnesium hydroxide buffering agents may have a prostaglandin-stimulating action as a heavy metal effect. The mucosal protective functions stimulated by prostaglandin E are discussed below. Results of some recent studies of sucralfate in horses indicate questionable efficacy in the treatment of squamous ulcers.

Prostaglandin analogues

When used in man, prostaglandin E\textsubscript{1} analogues are thought to act by inhibiting gastric acid secretion and enhancing mucosal protection. They have been shown to increase bicarbonate and mucus secretion in man and dogs. Published experimental data demonstrate that PGE\textsubscript{2} analogues protect the gastric mucosa from NSAID-induced ulceration. Side effects (diarrhoea, abdominal distention/bloating) reported in man may also apply to the horse. Anecdotal reports vary with regard to safety.

Gastric prokinetics

Compounds with recognised gastric prokinetic potential, such as bethanechol, metoclopramide, erythromycin, and cisapride, may be useful adjuncts to antacid therapy in the horse in cases in which there is no serious physical obstruction to gastric outflow. With the exception of metoclopramide, each of these agents has been shown to increase gastric emptying rate in controlled experiments in mature horses. Currently, however, only bethanechol has been used as an adjunct treatment of equine gastric ulcer disease and while it appears to have been helpful in some cases, no controlled therapeutic trials have been reported.

Acid suppression

This concept follows from the still widely held belief, 'no acid, no ulcer' (Fig 19). The current treatment of choice is to suppress acid production from the parietal cells. These cells release acid when any one of 3 distinct receptors (histamine, acetylcholine, gastrin) is stimulated. Currently, the most popular treatment involves an histamine receptor antagonist (H\textsubscript{2} blocker). The proton-pump inhibitors constitute a new treatment approach that blocks acid production within the cell. This modality is receptor-independent and therefore more effective.

Histamine receptor (H\textsubscript{2}) antagonists

The 4 most popular H\textsubscript{2} antagonists licensed in the United States for man are cimetidine, ranitidine, famotidine and nizatidine. Cimetidine has been shown to affect hepatic disposition of some drugs.

Most of these compounds have been tested in the horse; cimetidine and ranitidine have been studied most extensively (Table 2). Ranitidine at a dose of 6.6 mg/kg bwt q. 8 h has been demonstrated to maintain a pH level >4 when horses are allowed free access to hay.

Proton-pump inhibitors

This class of compounds is the newest addition to the antisecretory group. Unlike H\textsubscript{2}-receptor antagonists, proton-pump inhibitors block the enzyme (pump) responsible for a hydrogen-potassium exchange. Blocking this enzyme exchange retards the final step in parietal cell acid production. Omeprazole, one of 2 compounds in this class currently licensed in the United States for use in man, is now licensed for use in horses. It has demonstrated an excellent efficacy and safety profile. In double-blind, placebo-controlled field trials, omeprazole...
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Fig 7: Ulcer grade 2. There is generalised hyperkeratosis (yellow) of the squamous mucosa.

Fig 8: Ulcer grade 3. There are numerous erosions (arrows) in the squamous mucosa, extending from the margo plicatus (MP) into the fundus.

Fig 9: Ulcer grade 3. There are 2 medium to large ulcers (arrows) in the squamous mucosa adjacent to the margo plicatus (MP). Note: The smaller mucosal defects which extend into the fundus.

Fig 10: Ulcer grade 4. Deep, acute, bleeding ulceration, in the squamous mucosa along the greater curvature of the stomach.

Fig 11: There is a band of ulceration adjacent to the margo plicatus along the entire greater curvature, extending to the lesser curvature. This pattern of ulceration is consistent with a gastric emptying disorder.

Fig 12: Ulcer grade 0. Comparison: normal glandular mucosa in the the stomach, with several prominent rugal folds (arrows).
Fig 13: Ulcer grade 1. Endoscopic view of the antrum. A rugal fold is thickened and the mucosa appears reddened.

Fig 14: Ulcer grade 1. Reddening and thickening of rugal folds in the antrum of the stomach.

Fig 15: Ulcer grade 2. Linear erosions in the glandular mucosa.

Fig 16: Ulcer grade 3. Several linear erosions and ulcers along rugal folds in the antrum.

Fig 17: Ulcer grade 3. Ulceration in the antrum.

Fig 18: Ulcer grade 4. Chronic ulceration at the pylorus, with fibrosis and restriction in diameter of the pylorus.
paste, administered at 4 mg/kg bwt was effective in eliminating or substantially reducing the severity of gastric ulcers. At this dose rate, pH >6 persisted for 24 h after the last treatment.

In human medicine, omeprazole has been approved for long term and prophylactic use in patients with erosive oesophagitis. This is meaningful, because ulcers in the squamous portion of the equine stomach are most prevalent (>90%) and resemble the human form of erosive oesophagitis.

**Other compounds**

Because omeprazole is a substituted benzimidazole, there has been interest in the potential value of the benzimidazole anthelmintic, fenbendazole, for treatment of gastric ulcer disease in the horse. At present there is insufficient scientific evidence to support the use of other benzimidazole compounds.

**Antibiotics**

While there is little doubt that H₂ blockers and proton-pump inhibitors suppress acid production and induce healing in humans, there is an alarming rate of recurrence (50% over 6 months and 95% over 2 years). Recent reports indicating that Helicobacter pylori plays a pivotal role in the disease process led to antibiotic therapies and to the use of antibiotics in combination with acid-suppression therapies.

When compared to acid suppression alone, these combination therapies demonstrate greater short-term efficacy and recurrence rates that were reduced by 80%. In addition to its superior acid-suppression properties, omeprazole has been shown to have a bacteriostatic effect on H. pylori.

Numerous strategies have been developed to combine antibiotics with antisecretory compounds to treat the human syndromes. Omeprazole and amoxicillin have been shown to be the most effective and produce few side effects.

Numerous attempts to identify, isolate, or culture *H. pylori* in the horse have been unsuccessful. It is now generally agreed that Helicobacter infection is not a factor in the equine gastric ulcer syndrome.

**Treatment of uncomplicated equine peptic ulcer disease**

Omeprazole has the advantages that:

- It is licensed for use in horses
- Its efficacy has been demonstrated in controlled clinical trials
- Its ease of administration and once-daily dosing promotes compliance with a recommended treatment strategy.

**Prevalence of gastric ulcers in foals and adults**

**Foals**

The syndrome of consequential gastric mucosal ulcerations was first reported in 1964 in a retrospective *post mortem* study. Within 20 years of the first published study, the syndrome has been recognised as a well-established clinical problem. In one study at the University of Florida, a 25% prevalence and numerous forms of the disease were identified. No breed or sex predilection was found and more disease was detected at earlier ages (1–4 months).

Endoscopy provided additional insights into prevalence. In another study conducted in Virginia and Maryland, 94/183 (51%) of foals examined had endoscopic evidence of predominantly squamous gastric ulceration. An endoscopic study in the UK resulted in similar findings, which contrasted sharply with a nonendoscopic survey of UK practitioners supervising more than 6000 foals in which a prevalence of less than 1% was reported. This discrepancy emphasizes the occult nature of the disease, which provides few clues to its existence until the advanced stages.

Distribution of lesions includes both nonglandular and glandular portions of the stomach, duodenum, oesophagus, and (less frequently) the pylorus. Colonic ulcers have also been reported but are not considered to involve the same mechanisms that produce EGUS pathology.

Overall, the currently accepted prevalence estimates of gastric ulceration is 30–50%, increasing sharply in foals demonstrating signs of disease, especially gastrointestinal signs.

**Mature horses**

Gastric ulceration has been observed at necropsy for decades, but was previously judged as an 'incidental inconsequential finding.' Frequently, during *post mortem* examinations, pathologists would observe gastric ulcerations, predominantly in the squamous mucosa near the *margo plicatus*. These lesions were not considered significant or were apparently not accompanied by clinical signs of gastrointestinal distress or both.

A report from Hong Kong, where land constraints have necessitated the routine sacrifice of large numbers of racehorses at the end of each racing season, led to heightened awareness of ulcers as a disease entity in mature horses. It was noted at necropsy that approximately 70% of horses were affected with squamous ulcerations. Additionally, horses in active training had both a higher prevalence and enhanced severity of ulcer lesions than did horses not in training. Lesions were independent of age or medications used.

With the introduction (first report in 1985) of fibreoptic and videendoscopic technology the ability to examine the equine stomach was significantly enhanced. A second
published report (from the USA) provided estimates similar to those identified at necropsy. Approximately 90% of horses with signs of gastrointestinal distress had squamous ulcerations, whereas 50% of horses without clinical signs also had ulcerative changes. Using an established grading system, horses at higher activity levels and with signs of gastrointestinal distress had both increased frequency and increased severity of ulcer lesions.

### Anatomy

The equine gastric mucosa can be divided into 2 distinct types, glandular and nonglandular (squamous). Equine gastric ulcers occur in the squamous portion of the stomach in 75-80% of cases.

The squamous epithelium, which covers approximately one third of the stomach wall (Fig 21) and the oesophagus, is a relatively simple structure that consists of a tightly bound cornified superficial layer of cells that serves as a protective barrier. The squamous portion of the stomach lining has no absorptive or secretory function, leaving it more vulnerable to peptic injury.

The more complex glandular portion of the stomach contains mucus-secreting cells and gastric glands (Fig 22), all of which respond to various stimuli and provide secretions that have a specific function. The gastric glands contain six predominant cell types (Table 3).

### Physiology

In some mammals, contact with of food awakes nervous stimuli to the stomach. Ingestion of food distends the stomach, which in turn stimulates glandular secretions and nervous stimuli. Properties of ingested food (such as protein content and the acidity of the food) also stimulate the release of supplemental glandular secretions.

An element of gastric physiology of particular interest is the production of hydrochloric acid (HCl), secreted by the parietal cells found in the fundic portion of the glandular mucosa. It has long been believed that HCl concentration, as expressed by the pH value, is the major determinant of gastric ulcerogenesis.

The parietal cell secretes H+ via H+/K+ ATPase proton pump (Fig 19) located on its apical membrane. The K+ used by this proton pump and the Cl− that combines with the H+ are secreted through the same apical membrane via ion-specific channels. The resultant HCl moves up through the gastric gland and into the gastric lumen, lowering the pH of the gastric contents.

Acid and the enzyme pepsin exert peptic influences on digestion. The stomach has a number of protective mechanisms that prevent acid and peptic activity from causing inappropriate mucosal destruction. Understanding of the physiological gastrointestinal mechanisms that protect the gastric mucosa from damage by cytotoxic elements (such as acids, bile salts, pepsin, and digestive enzymes) facilitates the following discussion of the pathophysiological gastrointestinal mechanisms of the ulcer disease syndrome.

### Glandular mucosal defence mechanisms

Two important substances involved in gastric glandular mucosal protection are epidermal growth factors (EGFs) and prostaglandin E2 (PGE2). Epidermal growth factors are found in salivary gland secretions and promote DNA synthesis and proliferation of gastric mucosal cells. They also play a role in prostaglandin synthesis. The PGE2 promotes numerous protective functions within the gastric mucosa which include:

- Suppression of HCl secretion
- Mucus secretion
- Bicarbonate secretion
- Epithelial restitution mechanisms (maintain tight junctions)
- Adequate mucosal blood supply.

**Mucus**, secreted by specialised mucous neck cells, is a viscous, hydrophobic glycoproteinaceous gel that adheres to the mucosa and resists acid and pepsin contact. The gel also acts as a lubricant that minimises mechanical damage by gastric contents.

**Bicarbonate** secretion by gastric mucosal cells is triggered by a response to luminal acid concentrations, mechanical irritation, and by release of endogenous prostaglandins. Bicarbonate trapped in the mucous barrier adhering to the stomach wall forms a pH gradient that allows a physiological pH at the mucosal surface and a pH similar to that of stomach acid at the luminal surface.

**Prostaglandins** have an important role in gastric mucosal protection, although their precise mechanism is vague. Prostaglandins inhibit acid secretion, promote mucosal blood flow (vasodilate), increase mucus and bicarbonate secretions, and support mucosal cell repair. Treatment with nonsteroidal anti-inflammatory drugs (NSAIDs), corticosteroids, or the administration of prostaglandin antibodies results in ulcer formation in various species, including horses. This effect can be blunted by administering exogenous prostaglandins.

**Epithelial cell restitution** is another important mechanism in the maintenance of gastric mucosal integrity. Epithelial cells act as a protective covering, counter shear forces that induce damage, and provide rapid restoration of damaged protective barriers. In the case of the gastric mucosa, epithelial injury induces a migration of adjacent cells to replace damaged cells. This occurs within minutes without need of new cell proliferation. Shear forces, induced by mixing of ingested material raking against the mucosal wall, cause cell damage that is normally countered by the process of epithelial restoration.

An abundant mucosal blood supply is required to provide the mucosa with the oxygen and nutrients necessary to produce the mucus-bicarbonate layer and to support the rapid turnover of epithelial cells. An adequate blood supply is also required to remove acid that has diffused through the mucous layer to the mucosa. Alterations in blood flow (i.e., shock, microvascular thrombosis) are highly correlated with mucosal erosions and gastric ulcers in man.
**Epidermal growth factors** are found in salivary gland secretions and promote DNA synthesis and proliferation of gastric mucosal cells. They also play a role in prostaglandin synthesis and inhibit the parietal gland secretions of hydrochloric acid.

**Squamous mucosal defence mechanisms**

Many discussions surrounding equine squamous disease are extrapolated from human oesophageal disease and data from other species. In these species, the initial response of squamous tissues exposed to acid is to thicken. **Cell-to-cell junctions** are closely adhered (‘tight junctions’), which ensures a weak acid barrier. **Buffering** also is a component of the squamous tissue defence scheme, but unlike the glandular portion, there is no external mucus-bicarbonate layer. Rather, squamous tissues buffer internally (within the cell) and use leucotrienes for defence. By contrast, glandular tissue relies on prostaglandins for adequate mucosal protection.

**In summary**, the squamous tissues of the stomach have a limited number of defence mechanisms that centre around acid repulsion and intracellular buffering. Once the acid penetrates these defences, it builds up within the cell layers and necrosis (cell death) leads to ulcer formation.

**Gastric acidity and secretion**

The patterns and magnitude of basal and stimulated acid secretion vary among species. **The horse secretes acid in a continuously variable pattern**, such that acid secretion occurs in the absence of ingestion of feed. On a bodyweight basis, compared with man, the horse has a greater basal acid output, but a similar stimulated acid output.

The mean pH of gastric fluid in horses withheld from feed for several hours has consistently been found to be 2.0 or less. However, periodic fluctuations, in which the pH reaches 6–7 for 5 to 15 min, are commonly seen.

**Whereas acid output** reflects the magnitude of hydrochloric acid secretion, **gastric acidity** is determined by the pH of gastric secretions and/or contents. The pH of a solution is defined as the negative logarithm of hydrogen ion activity, with hydrogen ion activity determined by both the hydrogen ion concentration and the ionic strength of a solution. The greater the ionic strength, the lower the pH of a solution with a gastrointestinal concentration of acid.

The distinction between gastric acid output and pH is relevant, since administration of antisecretory medications may significantly decrease acid output but have minimal effect on gastric fluid pH in horses until the output becomes very small.

Few studies on the effect of feeding on gastric physiology have been performed in horses. Serum gastrin increase after feeding was more profound when grain was fed compared with feeding only hay. Horses with free access to hay had greater mean 24 h gastric pH than did horses withheld from feed for 24 h (3.4 ± 0.9 vs. 1.9 ± 0.5). These latter results support the continuous acid secretion of the equine stomach and the buffering capacity of feed and bicarbonate-rich salivary secretions that are stimulated by feeding. In foals, milk has been demonstrated to have a marked buffering effect.

There is a circadian pattern of basal gastric acid secretion in man, and gastric acidity is greatest during the late-night hours. A circadian pattern of gastric acidity has not been reported in horses.

Periods of prolonged gastric acidity (pH <2.0) were induced in horses using a protocol of intermittent feed deprivation, which resulted in severe ulceration of the gastric squamous epithelial mucosa. Concurrent administration of acid suppression therapy during feed deprivation substantially reduced the lesions in the gastric squamous epithelial mucosa.
Pathophysiology

The pathophysiology of ulcer formation in the horse is poorly understood. Since the exact mechanisms have not been identified, it is presumed that they are similar to other mammalian species, in which the causes are related to factors that undermine or overwhelm the protective defence mechanisms. Excessive acid or pepsin production is rarely identified as the cause of the ulcer.

Extrapolating from other species, causes of glandular ulcer formation would include:

- Bile reflux into the stomach
- Hypotension/shock (resulting in the diminution of mucosal blood flow)

- Increased sympathetic tone (leading to a decrease in mucosal blood flow)
- Severe disease states such as uraemia, infections, coagulopathies and conditions that result in the impairment of blood flow
- Neurological imbalance, resulting in impairment of gastric motility (results in the accumulation of acids)
- Micro-organisms (i.e., H. pylori, as yet not identified as a problem in the horse).

With regard to equine squamous ulcer disease, it is really more like the human GERD and the porcine gastroesophageal ulcer disease, where the pathophysiology is not yet well understood, except to know that acid is, again, important in the ulcerogenesis. Bile salts and SFCA have also been considered, but the
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Fig 21: Internal view of the stomach.

Fig 22: Mucus-secreting cells and gastric glands.

Publications and thoughts of opinion leaders regarding the subject of equine gastric ulcer syndrome; however, a great deal is yet to be discovered.

Highlights of this report include:

- EGUS is prevalent and represents a significant health problem.
- Risk factors for the development of EGUS include intensive exercise, transportation, serious illness, diet, management changes, and high doses of NSAID therapy.
- Endoscopy is the only definitive diagnostic method available at this time.
- Clinical signs, while nonspecific, include:
  - poor appetite/failure to completely consume a meal
  - dullness
  - attitude change
  - decrease in performance/reluctance to train
  - poor body condition/rough hair coat/weight loss; and,
  - low-grade colic or excessive recumbency.
- Therapeutic trials of anti-ulcer medication may be useful in horses who manifest clinical signs compatible with ulcers and do not have access to endoscopy.
- Numerous compounds are available for therapy; to date, the most rigorously tested anti-ulcer medications shown to be safe and effective are the proton-pump inhibitors.

For a complete list of references please send SAE to the Editorial Office.

Conclusions

This article has compiled a consensus of all the current evidence to date concerning this role is minuscule.

Drugs such as NSAIDs have been shown to induce gastric ulcers in man, canines, foals, and mature horses. They do so by inhibiting prostaglandin synthesis.