

# The Equine Stomach

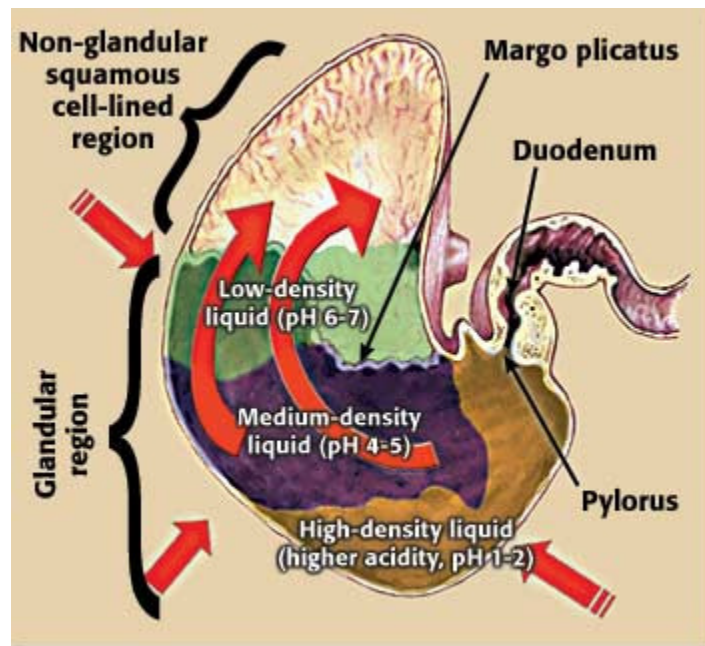
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The Milne Lecture, named for AAEP past president and distinguished life member Frank J. Milne, each year honors a researcher for his lifetime of work in a specific field. This year the presenter was Alfred Merritt, DVM, MS, of the University of Florida, who spoke on "The Equine Stomach: A Personal Experience (1963-2003)." Merritt's presentation highlighted advances made in the understanding of how the equine stomach functions and its diseases. He emphasized information on equine gastric ulcer syndrome (EGUS).

## Basic Anatomy

Merritt described the various components of mucosal anatomy (the mucous membranes of the stomach), which include the squamous (non-glandular), fundic glandular, cardiac glandular, and the pyloric glandular regions. He noted that the fundic glandular region is where acid is made. Merritt pointed out on a photo how the glandular region is redder than the squamous region, and the margo plicatus is the line between the non-glandular and the glandular regions. The pylorus is the opening from the stomach into the small intestine, with the duodenum being the first part of the small intestine. The pyloric sphincter is open most of the time.



DR. ROBIN PETERSON ILLUSTRATION

**The upper area of the stomach is the non-glandular squamous cell-lined area; the lower area is the glandular portion. The margo plicatus divides them. The pylorus is the opening from the stomach into the small intestine, with the duodenum the first part of the small intestine. The red arrows show the proposed way by which exercise in the horse, during which abdominal muscles compress the stomach more than they do at rest, causes a change in pH (acidity) in the upper portion of the stomach. This increased acidity can lead to gastric ulcers in the upper, non-glandular region of the stomach.**

The normal equine stomach is rarely empty. The contents separate into different density levels, with each layer having a different pH value. The more dense and acidic layer is at the bottom of the stomach.

## **Digestive Activity**

Merritt discussed the mechanisms of digestion in the equine stomach and control of gastric acid secretion, demonstrating that the stomach acts as its own pH meter. One of the chemicals that induces acid secretion in the horse is histamine. Histamine-2 (H<sub>2</sub>) receptor antagonists suppress gastric acid. An example of an H<sub>2</sub>-receptor antagonist is ranitidine, which is typically used to control acid in horses with gastric ulcers. Merritt said it takes less histamine to induce maximal acid secretion in the horse than in humans and other domestic species. "This is of particular interest to GI pharmacologists and might, in part, explain why horses require such large doses of H<sub>2</sub>-receptor antagonists, relative to other species, to effectively inhibit gastric secretion," he said.

"There now exist a number of reports concerning the anti-secretory efficacy of the proton pump inhibitor (PPI) omeprazole (an acid suppressant) on both basal and pentagastrin-induced acid secretion in the horse," he said. "As with H<sub>2</sub>-receptor antagonists, the acceptable effective dose of omeprazole for the horse is considerably higher than that required in other species, especially when given orally."

Prostaglandin-E<sub>2</sub> (PGE<sub>2</sub>) stimulates mechanisms that help protect mucosal surfaces in the stomach. It also inhibits acid production. Corn oil has been found to increase PGE<sub>2</sub> and reduce gastric acid.

It is known that continuous NSAID therapy for the treatment of chronic musculoskeletal problems can cause ulcers in the glandular mucosa. Due to protective effects of PGE<sub>2</sub> on mucosa, Merritt recommends corn oil supplementation for horses on continuous NSAID therapy.

## **Motility**

The stomach can relax and expand to accommodate a meal; however, if the stomach does not relax, the volume of food that can be taken in is limited and there is a risk of rupture if a horse eats too much.

Merritt has measured the changes in the gastric wall as it relaxes and contracts. There is a distinct two-phase response to a meal. The first phase occurs while the horse eats and is called receptive relaxation. When the horse finishes eating, the second phase, called accommodation, occurs. This phase is more prolonged and probably is under the control of feedback from the small intestine.

Two factors affect emptying of the stomach's contents into the small intestine--increased tone of the proximal (upper) half, and peristaltic contractions (progressive wave-like motions produced by successive contractions of the muscle fibers of the lower stomach wall). Studies have shown that liquids move through the stomach more quickly than solids.

## **Diseases of the Equine Stomach**

**Impaction**--This occurs when dry contents don't move out of the stomach properly. The most common cause is grain overload. While colic from impaction of the gastrointestinal tract is a known problem, stomach impaction is rare.

There are two forms of gastric impaction--one has a partial gastric outflow obstruction with a physical cause, and one has no physical obstruction. A horse with a gastric impaction will present with colic of varying intensity, which might be recurrent, said Merritt.

**Tumors**--Merritt included inflammatory growths as well as abnormal growths (neoplasia) in his discussion. Neoplastic growths can include squamous cell carcinoma, which is found in the non-glandular squamous region of the stomach. If a horse older than five experiences weight loss, lack of appetite, anemia, and intermittent

drooling, he should have an endoscopic exam performed to look for squamous cell carcinoma. When masses have grown large enough or spread into the peritoneal cavity, they can sometimes be felt by rectal palpation, or neoplastic cells can be seen in peritoneal fluid. Necropsy can also indicate the presence of disease.

**Reflux--**Treatment of gastric reflux has historically included passing a tube into the stomach through the nose to drain gastric contents. When reflux is caused by a physical blockage or a functional obstruction due to a lack of motility in the small intestine, the fluid could come primarily from the pancreas. The horse normally produces a lot of pancreatic juices, which are watery and have little odor. If the fluid is reddish, cloudy, and has a fetid odor, the cause might be due to inflammation in the duodenum or jejunum (regions of the small intestine). If the fluid is in between these descriptions, the problem could be a small intestinal obstruction.

## **Equine Gastric Ulcer Syndrome**

Merritt said EGUS is mucosal erosion and ulceration within the esophagus, stomach, and/or upper duodenum. EGUS is classified based on a description of the horse, management conditions, medical status, and/or primary lesion site as determined by endoscopic exam. Lesions are described as primary squamous (non-glandular) lesions, primary glandular and/ or upper duodenal mucosal lesions, and primary lesions within the cardiac gland mucosa of highly stressed neonates.

**Primary Erosion and/or Ulceration of the Non-Glandular (Squamous) Mucosa--**These ulcers are primarily found with an endoscopic exam in adult horses in training or in some younger horses. Lesions are typically found near or along the length of the margo plicatus, and/or extending up into the non-glandular squamous mucosa.

"A large number of publications have appeared over the last 10 years indicating that up to 90% of horses in training, irrespective of breed and type of exercise, may have erosive and ulcerative lesions of the squamous mucosa of varying degrees of severity," said Merritt.

With these lesions, owners might notice a horse has a poor appetite, reluctance to train or decreased performance, poor body condition, poor hair coat, and/or low-grade colic, especially after a grain meal.

Lesions are believed to be caused by a change in, or excessive exposure to, some part of the acidic gastric contents, said Merritt. Treatment with acid-suppressing drugs has been found to help, even in horses still in training.

Another possible cause could include the production of volatile fatty acids (VFA), which are by-products of fermentation of feedstuffs with soluble carbohydrates. These types of feedstuffs are commonly fed to horses in training. Researchers have found that feeding alfalfa hay raises pH to less acidic levels, despite more VFA production, and alfalfa-fed animals have fewer squamous lesions than horses fed brome grass. This unexpected result is attributed to the buffering effect of constituent calcium salts and protein found in alfalfa.

Exercise has been linked to ulcers. Merritt explained that a horse at a gait faster than a walk will tense his abdominal muscles, which pushes gastric contents up into the squamous cell-lined region of the stomach, exposing it to corrosive acid. Lesions can resolve when a horse is taken out of training or is treated with acid-suppressing medications.

**Primary Glandular Disease--**This disease has been attributed to continuous NSAID therapy. NSAIDs are considered ulcerogenic since they affect the production of PGE2 as discussed previously. Merritt commented that if the recommended dosages of NSAIDs are followed, most horses will not have problems; however, some horses seem to be more sensitive to NSAIDs. Lesions of the glandular mucosa that are not related to NSAID therapy have also been seen in horses. The cause(s) of these lesions is unknown.

**Secondary Squamous Ulceration**--This type of ulceration is rarely seen in horses older than one year of age and is associated with gastroduodenal ulcer disease (GDUD), where the primary lesion is found in the duodenum. Clinical signs include watery diarrhea, refusal to nurse, mild colic, and/ or teeth grinding. On rare occasions, secondary gastric ulcers can perforate the non-glandular lining, which can be deadly.

An endoscopic exam must include examination of the duodenum for diagnosis. The duodenum might show swelling, along with a yellowish covering of pus and lesions. Lesions might also be found in the squamous mucosa.

With anti-ulcer medication, most foals can be back to normal within a week. Those which do not recover might begin drooling, grind their teeth, have periodic bouts of colic (especially after nursing), and have notable weight loss. These signs could indicate severe inflammation of the duodenum with fibrous tissue, which blocks gastric emptying and often causes severe secondary gastric squamous (and sometimes esophageal) ulceration.

**Stress-Related Disease**--This form of ulcers is perhaps related to a disruption of circulation in the mucosa of the stomach, or possibly as a result of a disruption in PGE2 production. This problem is most often seen in foals suffering from a severe illness or trauma, and due to a variety of problems that can result, antacid drugs are often routinely given to very sick foals. These lesions are mainly found in the glandular mucosa next to the margo plicatus in the cardiac gland region. Perforation and death are possible.

### **Anti-Ulcer Treatment**

Treatment for ulcers can include medicating with an aluminum/magnesium hydroxide preparation to buffer gastric acid.

"Clinical experience has indicated that administration of 240 mL of an extra-strength oral antacid product twice a day has been effective in treating squamous ulcer disease in some patients," said Merritt. "A few horses will actually eat flavored products mixed in with grain. There is also a feed additive for horses, which is marketed with the claim of having effective acid-buffering properties that can eliminate the signs that we associate with squamous ulcer disease. To date, however, there are no intra-gastric pH data available showing that this feed additive meets these claims."

Another treatment involves histamine-2 receptor antagonists, such as cimetidine and ranitidine. Merritt said any of these types of products on the market should work as long as an effective dose is given.

The third type of anti-ulcer treatment involves administration of a proton-pump blocking agent, such as omeprazole. The only FDA-approved product is an oral paste called GastroGard. Daily dosing with GastroGard at 4 mg/kg once a day has been shown to reduce the severity and occurrence of primary squamous lesions in horses in training.

Although many companies have tried compounding omeprazole, experimental studies done at the University of Florida and clinical studies done at the University of California, Davis, and the University of Pennsylvania, show that the compounded drugs are not as effective.

Polysulfated sugar (sucralfate) is a compound available as an ulcer-coating agent. "So far, clinical trials of its efficacy in treatment of experimental EGUS have not been very convincing," Merritt said.

### **The Future of Research**

"In my opinion, there are currently two major challenges on the table concerning EGUS," he said. "The first challenge is the determination of the specific cause of gastroduodenal ulcer disease syndrome. The second major challenge regarding EGUS is the development of nutritional and other management strategies to reduce the incidence of training-related squamous ulcer disease." (See article [#4959](#) online.)

## Preventing Gastric Ulcers

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"Gastric ulcers are a significant problem," said Gary W. White, DVM, of Salisaw Equine Clinic in Oklahoma. "Omeprazole (Gastrogard) is FDA-approved for the treatment of gastric ulcers in horses and foals greater than four weeks old at 4 mg/kg/ day," he said, adding that reports had suggested that doses lower than 2 mg/kg/day (a dose proven to prevent recurrence of ulcers) might be effective in preventing development of gastric ulcers.

White and 10 veterinarians from private and university equine hospitals in the United States and Canada investigated whether a dose of 1 mg/kg/day would prevent occurrence and recurrence of gastric ulcers in racehorses. A total of 285 horses (including Thoroughbreds, Quarter Horses, and Standardbreds) were used to confirm the dose required to stave off ulcer occurrence and prevent recurrence. "During the study period, these horses were maintained in active race training and were allowed to race, so they were maintained under strong ulcerogenic conditions," said White.

Veterinarians compared the effects of orally administered omeprazole paste at 0.5, 1.0, or 2.0 mg/kg/day and a sham-dose (an empty tube used in the control group) for 28 days. They found the 1.0 mg dose of omeprazole was more effective in preventing recurrence of ulcers than either the sham or 0.5 mg doses. In the prevention of the occurrence of ulcers, the 1.0 mg dose was more effective than the sham dose, and the effect did not differ significantly from the 2.0 mg dose.

Researchers found gastric ulcers developed in 86% and 82% of horses receiving the sham dose in both trials. White said, "This shows the studies were designed so the horses were under intense ulcerative pressure."

Only 18% of horses receiving the 1.0 mg dose got ulcers, showing that the dose provided 82% prevention of occurrence. Ulcers recurred in only 19% of cases, showing 81% prevention of recurrence.

He summarized, "Omeprazole can be administered at an oral dose of 1 mg/kg/day. Ulcer prevention was demonstrated under ulcerogenic conditions, and we found that higher doses and loading doses don't seem to enhance the prevention or recurrence

*By Sarah L. Evers and Stephanie L. Church*