



Down on the Farm

Gastric Ulcers in Horses: A Widespread But Manageable Disease

by DR. JOE D. PAGAN

Every equine practitioner appreciates the delicate nature of the equine gut. Problems related to the small intestine and large intestine are well understood and routinely treated. What may be surprising to many is how often the stomach is affected. Specifically, the incidence of gastric ulcers is extremely high, particularly in performance horses.

Many studies since the mid 1980's have documented that gastric ulcers are commonplace in racehorses. An early postmortem study in Hong Kong (Hammond et al, 1986^o of 195 Thoroughbred racehorses showed that 80 percent of the horses in active training had ulcers. The incidence of ulcers in horses retired from racing for one month or longer was 52 percent. Murray, et. al., 1989, examined the stomachs of 187 horses ranging in age from one to 24 years. Eighty-seven horses had clinical problems, including recurrent colic, poor body condition or chronic diarrhea. One hundred horses had no clinical signs of gastrointestinal problems. Ninety-two percent of the horses with clinical problems had gastric ulcers. Surprisingly, 52 percent of the horses displaying no clinical signs also had lesions. Racehorses in training had a higher incidence of ulcers (89 percent) than non-racers (59 percent).

More recently, two studies evaluated the incidence of gastric ulcers in California racehorses. In one postmortem study (Johnson, et. al., 1994) of 169 horses in training, 88 percent of Thoroughbred horses in training had ulcers. A gastroendoscopic study of 202 Thoroughbred horses in training showed that 81 percent had ulcers (Vatistas, et. al., 1994).

Each of these studies revealed that 80 to 90 percent of racehorses in training have gastric ulcers. The vast majority of these lesions occur in the region of the stomach above the margo plicatus, with very few lesions in the glandular por-

tion (Figure 1). The upper half of the horse's stomach consists of squamous epithelial cells that are very similar to the tissue found in the esophagus. Ulcers in this part of the stomach are more similar to esophagitis (heartburn) in humans than the ulcers that occur in the glandular region of the human stomach. Research has shown that equine gastric ulcers are not caused by *Helicobacter pylori* bacteria which are a common cause of ulcers in humans.

Gastric ulcers are not caused by *Helicobacter pylori* bacteria which are a common cause of ulcers in humans.

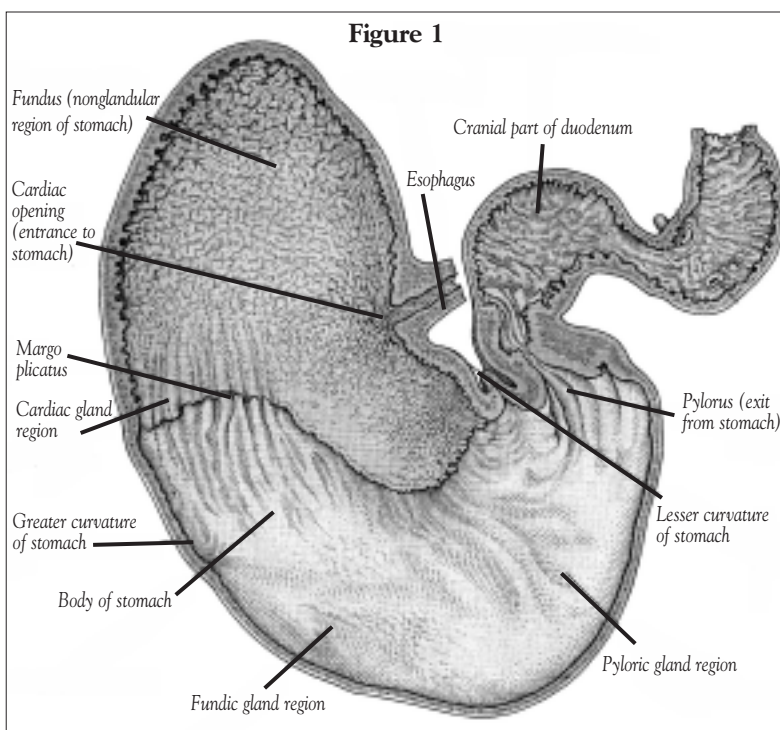
Gastric Acid is a Major Cause

Dr. M. J. Murray of the Marion duPont Scott Equine Medical Center in Leesburg, Virginia, has proposed that the major cause of gastric ulcers in horses is prolonged exposure of the squamous mucosa to gastric acid. Unlike the glandular portion of the stomach, the squamous mucosa does not have a mucous layer and does not secrete bicarbonate onto its luminal surface.

The only protection that this portion of the stomach has from gastric acid and pepsin comes from saliva production. If adequate saliva is not produced to buffer the gastric acid and coat the squamous epithelium, then gastric irritation occurs and lesions may develop.

The high incidence of ulcers seen in performance horses is a man-made problem resulting from the way that we feed and manage these horses, since ulcers are extremely rare in horses maintained solely on pasture. Horses evolved as wandering grazers with digestive tracts designed for continual consumption of forage. Meals of grain or extended periods of fasting lead to excess gastric acid output without adequate saliva production.

Horses secrete acid continually whether they are fed or not. The pH of gastric fluid in horses that were not fed for several hours has consistently been measured to be 2.0 or less (Murray, 1992). Horses who received free choice timothy hay for 24 hours had mean gastric pH values that were



significantly higher than fasted horses—3.1 in fed horses versus 1.5 in fasted horses (Murray and Schusser, 1989). Higher pH values in hay-fed horses should be expected since forage consumption stimulates saliva production. Meyer, et. al., 1985, measured the amount of saliva produced when horses ate either hay, pasture or a grain feed. When fed hay and fresh grass, the horse produced 400 to 480 grams of saliva per 100 grams of dry matter consumed. When a grain-based feed was offered, the horses produced only about half (206 grams/100 grams dry matter) as much saliva.

Grains and pelleted concentrates also increase the production of gastrin, a hormone that stimulates gastric acid production (Smyth et al, 1988). Hay fed alone affects gastrin production to a lesser degree. Therefore, horses who are fasted or who are fed high grain diets are more likely to produce more gastric acid with less saliva than horses offered free choice forage without grain.

It is easy to see why racehorses have such a high incidence of ulcers. Most horses in training are confined for most of the day and fed large grain meals. Often, racehorses are fasted for an extended period before exercise, allowing gastric acid to accumulate in the stomach. Intense exercise further increases the production of gastric acid so that the squamous mucosa of the stomach gets thoroughly bathed in acid during work.

Treatment and Prevention of Ulcers

Treating ulcers involves either inhibiting gastric acid secretion or neutralizing the acid produced. There are three classes of drugs that can be used to inhibit gastric acid secretion:

1) Histamine type-two antagonists (H₂ antagonists). H₂ antagonists act by competing with histamine for histamine type-two receptor sites on the parietal cell and therefore blocking histamine-stimulated gastric acid secretion. The two most popular H₂ antagonists used in horses are cimetidine (Tagamet) and ranitidine (Zantac);

2) H⁺/K⁺ATPase inhibitors. Direct inhibition of the proton pump can be achieved by substituted benzimidazoles. The only proton pump inhibitor licensed for horses in the US is omeprazole (GastroGard); and

3) Prostaglandin analogues.

This article was provided by Kentucky Equine Research, Inc., 3910 Delaney Ferry Road, Versailles, KY 40383, (859) 873-1988 or www.ker.com

References

- Hammond, C. J. Mason, D.K. and Watkins, K. L. (1986), Gastric ulceration in mature Thoroughbred horses. *Equine Vet J.* 18, 284-287.
- Johnson, W., Carlson, G. P., Vatistas, N., Snyder, J. R., Lloyd, K., and Koobs, J. (1994), Investigation of the number and location of gastric ulcerations in horses in race training submitted to the California racehorse postmortem program. 40th AAEP Convention Proceedings, 123-124.
- Meyer, H., Coenen, M. and Gurer, C. (1985), Investigations of saliva production and chewing in horses fed various feeds. *Proceedings of 9th ENPS, East Lansing, MI.*, 38-41.
- Murray M.J. and Schusser, G. (1989), Application of gastric pH-metry in horses: measurement of 24 hour gastric pH in horses fed, fasted, and treated with ranitidine. *J. Vet. Intern. Med.* 6, 133.
- Murray, M. J., Grodinsky, C., Anderson, C. W., Radue, P. F. and Schmidt, G. R. (1989), Gastric ulcers in horses: A comparison of endoscopic findings in horses with and without clinical signs. *Equine Vet J. Suppl* 7, 68-72.
- Murray, M. J. (1992), Aetiopathogenesis and treatment of peptic ulcer in the horse: a comparative review. *Equine Vet J. Suppl.* 13, 63-74.
- Smyth, G. B., Young, D. W. and Hammond, L. S. (1988), Effects of diet and feeding on post-prandial serum gastrin and insulin concentrations in adult horses. *Equine Vet. J. Suppl.* 7, 56-59.
- Vatistas, N. J., Snyder, J. R., Carlson, G., Johnson, B., Arthur, R. M., Thurmond, M., and Lloyd, K. C. K. (1994) Epidemiological study of gastric ulceration in the Thoroughbred race horse: 202 horses 1992-1993. 40th AAEP Convention Proceedings, 125-126.

An alternative to suppression of acid production is to neutralize stomach acid and protect the squamous mucosa from exposure to acid. The natural buffering mechanism in the horse is saliva production and, indeed, the most effective way to treat ulcers is simply to turn the animal out on pasture. In situations where this is not possible, administration of antacids may be a useful adjunct to acid suppression therapy in horses.