The stomach of the horse may undergo pathological changes of its wall associated with gastric ulcer disease, rupture, chronic impaction or neoplasia.

**Gastric Ulceration in the Adult**

Gastric ulceration syndrome (GUS), a complicated and multi-factorial problem, is a common disorder of the stomach of mature horses. Horses that are turned out to pasture and are not involved in work have normal stomachs as do most horses used for light work or pleasure (Murray 1994). In recent years the widespread nature of this disorder has gained increased recognition and studies have shown that racing horses and horses in race training have a high prevalence of gastric ulcers with estimates ranging from 66 per cent (Hammond et al. 1986) to over 90 per cent (Murray 1994). Horses used for pleasure have a lower prevalence (37 per cent) and severity of ulcers (Murray et al. 1989). It has been suggested that the prevalence and severity of gastric ulcers increase with the duration of race training. Prevalence in a randomly selected group of normal thoroughbred horses in race training was 28 per cent at the start of the study and 63 per cent with ongoing racing and training (Orsini and Pipers 1997). GUS is due to an imbalance between mucosal aggressive factors (hydrochloric acid, pepsin, bile acids and organic acids) and mucosal protective factors (bicarbonate and mucus). The equine stomach is lined dorsally by a stratified squamous epithelium and ventrally by a glandular epithelium. These epithelia have different functions and different susceptibilities to peptic injury. The squamous portion of the stomach appears to serve as a reservoir for ingesta and has no secretory or absorptive function. Because its mucosa has no surface barrier to hydrochloric acid, its protection from peptic injury depends on limited exposure to acidic gastric secretions. In contrast the glandular mucosa has more protective factors and may have different causes for ulceration. It secretes hydrochloric acid and pepsin as well as some water and electrolytes, and a variety of endocrine mediators are produced within this mucosa. The gastric glandular mucosa has evolved elaborate mechanisms to protect itself from peptic injury including a mucus/bicarbonate barrier that prevents back diffusion of hydrochloric acid, mucosal blood flow, cellular restitution and growth factors that promote mucosal healing. Prostaglandin E2 promotes secretion of the mucus/bicarbonate layer and enhances mucosal blood flow, mucus and bicarbonate production. Consequently, inhibiting prostaglandin synthesis decreases mucosal blood flow and mucus and bicarbonate secretion and increases gastric acid secretion by the glandular mucosa. Prostaglandins may provide additional protection by helping to maintain the integrity of the glandular and non-glandular mucosa by stimulation of surface-protective phospholipids, enhancement of mucosal repair and prevention of cell swelling by stimulation of sodium transport. The stress of training and confinement in adult horses may lead to the exogenous release of corticosteroids which can inhibit prostaglandin synthesis. This decrease in prostaglandin synthesis may lead to breakdown of mucosal protective factors. The equine stomach secretes hydrochloric acid continuously even when the horse is not eating. Gastric acidity is least when the horse is eating because eating stimulates secretion of bicarbonate-rich saliva that can neutralise some gastric acid, and roughage absorbs gastric secretions so that they do not contact the mucosal surface. Once a horse stops eating, gastric acidity can rapidly increase, with pH falling below 2.0 and acidity remaining high while the horse does not eat.

The gastric mucosa is damaged by excessive exposure to hydrochloric acid and the proteolytic enzyme pepsin. Lesions in the gastric squamous mucosa form within 24 - 48 hours if horses are prevented from eating because the gastric hydrochloric acid comes into contact with the mucosa surface which has no inherent protection from HCl-induced injury. The non-glandular mucosa has no mucous layer and responds to acid irritation by increasing the thickness of its keratin layer, which provides only minimal protection from acid and pepsin. Stomach pH has been strongly implicated in gastric ulceration in adult horses. In one study the squamous epithelium had a lower pH than the glandular mucosa, with the lowest readings near the margo plicatus, where most ulcers naturally occur. Approximately 50 per cent of the horses with moderate to severe...
Ulceration demonstrates a significantly lower stomach pH than horses with mild or no ulceration.

Lesions in the gastric glandular portion of the stomach occur when there is impairment of mucosal resistance, permitting exposure of the mucosa to hydrochloric acid and pepsin. This can occur with illness, or from administration of excessive NSAIDs and possibly intense exercise. In one study, during intense treadmill exercise, blood flow in the gastric antrum was reduced by a greater proportion than in any other abdominal organ. Factors that impair mucosal resistance in adult horses are poorly understood but studies in laboratory animals have implicated reperfusion injury as a cause of impaired mucosal resistance and ulceration.

*Helicobacter pylori* is considered to be the primary cause of peptic ulcers in humans but this organism has not been found in the horse. However, in a recent study, Helicobacter-specific DNA was isolated from the glandular and squamous epithelia of seven horse stomachs including two horses with squamous erosions and one with glandular epithelial erosions (Scott et al. 2001). This study suggests that *Helicobacter* spp. may be involved in the aetiology of GUS in horses and merits further study.

**Epidemiology**

Horses of all breeds and uses can develop gastric ulcers. The prevalence of the lesions is influenced by the management and use of the horse. Horses at pasture or undergoing only light exercise have normal stomachs or only very mild erosions. In contrast, horses confined to stalls or trained intensively have a high prevalence, up to 90 per cent of gastric lesions. A recent study (Lorenzo-Figueras et al. 2002) suggests that increase of intra-abdominal pressure during intense exercise causes gastric compression, pushing acidic contents up into the proximal, squamous-lined region. Increased time of acid exposure, related directly to daily duration of exercise, may explain why squamous lesions tend to develop or worsen when horses are in continuous training.

The stressful effect of strenuous exercise may contribute to gastric ulceration by delaying gastric emptying and/or increasing gastric secretion.

Although the prevalence of gastric lesions is greatest in horses managed intensively, clinical problems associated with gastric ulcers occur in horses used for many activities. Management is probably a significant factor because the type of food and eating behaviour can influence gastric ulceration. Restricting access to roughage and feeding a large amount of concentrate, by reducing the amount of time a horse consumes roughage, promotes increased gastric acidity. High roughage diets tend to stimulate production of bicarbonate-rich saliva which may buffer gastric acid. Because performance horses consume diets high in fermentable carbohydrates, the volatile fatty acids by-products generated by resident bacteria can contribute to acid injury and ulceration in the non-glandular mucosa. In addition, feeding concentrates stimulates a greater postprandial serum gastrin response than feeding roughage and gastrin is a potent stimulus to hydrochloric acid secretion.

**Clinical Signs and Significance**

The clinical significance of gastric ulceration in adult horses is not well defined. The correlation between clinical signs and severity of ulceration appears to be quite variable (Murray et al. 1989). Horses may have severe ulceration and few, if any, clinical signs or mild ulceration and severe clinical signs. The signs associated with gastric ulceration include mild to severe colic, poor appetite, poor condition, attitude changes and poor racing performance. Many of these clinical signs are subjective and some, such as poor performance and attitude changes, may be difficult to document or attribute specifically to gastric ulceration (Orsini 2000). However, when clinical signs resolve or racing performance improves with treatment and resolution of ulcers, this is compelling evidence that ulcers may have significant adverse effects in some individuals, even if this does not hold for every case.

**Diagnosis of Gastric Ulceration**

Gastric ulceration in mature horses may be suspected from clinical signs but a diagnosis can only be made by endoscopy. Most gastric lesions occur in the squamous mucosa usually adjacent to the margo plicatus, along the right side or the lesser curvature. Lesions may develop in the glandular mucosa mostly in the antrum. A 2 or 3 m endoscope is required to reach the squamous portion of the stomach. The horses must have food withheld for 6 - 8 hours prior to endoscopy to allow time for the stomach to empty and permit visualisation of the mucosa. A means of flushing residual food material and mucus from the mucosa is helpful. A number of scoring systems have been used in clinical studies to quantify the severity of ulcers. However, much debate exists regarding these scoring systems and their ability to accurately predict the severity or depth of gastric ulcers. In a study to evaluate the ability of two endoscopic non-glandular gastric ulcer scoring systems to predict
ulcer severity by ulcer depth on histopathological examination (Andrews 2002), endoscopy was found to be accurate in observing non-glandular ulcers but glandular gastric ulcers may be present but not identified. The severity of gastric ulcers in this study was underestimated on endoscopy examination when compared to the same ulcers scored at necropsy and on histopathological examination.

**Prevention and Treatment of Gastric Ulcers**

Pasture turnout is the most natural and least ulcerogenic environment. Horses that must be confined to a stall should have continuous access to hay and should eat it. The practical requirements of race training may make these recommendations difficult or impossible to achieve. The primary principle of treating gastroduodenal ulcers in horses is to reduce gastric acidity: This provides symptomatic relief and creates an environment that is conducive to ulcer healing. Although natural processes that promote healing are initiated within hours of peptic injury and lead to healing of individual ulcers without treatment, in an acidic environment new ulcers can form. Therefore in a horse that has clinical signs referable to gastric ulceration, treatment is recommended.

Treatments that reduce gastric acidity include antacids, histamine type-2 receptor antagonists (H2 antagonists) and the proton pump inhibitors. Antacids such as magnesium oxide and aluminium hydroxide neutralise existing gastric acid but only for a brief period and must be given in large volumes every 2 - 4 hours.

Horses with documented ulcers respond well to treatment with histamine type-2 receptor (H2) antagonists such as ranitidine and cimetidine but there is wide variability among horses in acid suppression achieved with these drugs. This is probably largely related to varying bio-availability of the drug in individual horses but other unknown factors may contribute. This variability is most profound at low doses.

A newer and more effective treatment approach is inhibition of the proton pump which is the final pathway in gastric acid secretion. The use of an acid inhibitor such as Omeprazole achieves maximum control of acid production regardless of the stimulus to the parietal cell. One important advantage of proton pump inhibitors is their ability to inhibit acid production for 24 hours with once a day dosing. Merritt et al. (2000) investigated the percentage to time over a 24 hour post treatment period that the gastric pH was > 4.0 (the accepted ulcerogenic cut-off point in human medicine) after treatment with 3 generic preparations of Omeprazole. They concluded that Omeprazole at 40 mg/kg per os q.d. can effectively maintain intragastric pH at an accepted anti-ulcerogenic level for at least 12 hours post-administration. Omeprazole now available in a paste formulation for horses (Gastroguard), given orally at 4.0 mg/kg once daily has been shown to be highly effective in promoting gastric ulcer healing in horses. In several trials ulcer healing in Omeprazole-treated horses was substantially superior to controls. Importantly, in one set of trials, ulcer healing occurred in more than 77 per cent of horses that remained in race training and this has not been noted in horses treated with H2 antagonists.

The duration of treatment required for ulcers will vary depending on the severity of lesions and the management of the horse. Gastric erosions will heal more quickly. Deep ulcers may require weeks to heal because granulation of the ulcer bed followed by epithelial contraction is necessary for complete healing.

**Gastric Rupture**

Gastric rupture occurs as a sequel to severe and prolonged gastric distension with ingesta, fluid or gas or a combination of all three. The adult equine stomach can hold up to 25 l (even 40 l in the largest horses) when maximally distended. Gastric rupture can occur from simple excessive distention but may be predisposed to .... by compromised integrity of its wall because of decreased blood flow. In some cases it has appeared that rupture occurred as the result of an infarction of the stomach wall in the absence of substantial distension. Gastric ulceration rarely leads to perforation in adult horses in contrast to foals. In three retrospective studies on equine colic prevalences of gastric rupture of 5 per cent (Tennant, Wheat and Meagher 1972), 8 per cent (Coffman 1970) and 3 per cent (Edwards 1997) were reported and prevalences of 1.6 and 3 per cent respectively in three series of necropsies by Baker and Ellis (1981), Delli Quadri (1972) and Todhunter, Hollis & Roth (1980). In contrast with spontaneous gastric rupture in humans which occur on the lesser curvature usually near the cardia (Dale 1980), most ruptures in horses are located on a line parallel with the greater curvature and about 5 cm medial to it. Gastric rupture is usually a biphasic process. The seromuscular tear is generally longer than the mucosal tear indicating that the seromuscularis weakens first. In most cases rupture of the mucosa quickly follows but may be delayed possibly for several days. Successful repair of seromuscular tears has been reported by Boening and Von Plocki (1982) and Steenhaus, Vlaminck and Gastbuys (1986). Much of the ingesta remains in the stomach if the breach in the mucosa is small or is trapped in by the greater omentum but the fluid content of the stomach is rapidly disseminated throughout the abdomen.
resulting in a fulminating peritonitis which is rapidly fatal.
Characteristically a horse with gastric rupture is severely depressed and stands immobile because of severe parietal pain.
Rigid "boarding" of the abdominal musculature is further evidence of intense peritoneal inflammation. The heart rate is in excess of 100 per minute and the pulse is weak and difficult to palpate. The horse sweats profusely but its extremities are cold. Respirations are rapid and shallow, and the mucus membranes cyanotic. The horse succumbs rapidly to overwhelming shock.

**Chronic Gastric Impaction**
Stomach impaction is rare and comprised only 0.3 per cent of cases reviewed in the Bolshoi Study (White and Lessard 1986). In its acute form it is usually characterised by severe abdominal pain but some horses may present with intermittent mild to moderate pain over several days. The cause of the impaction is often not known. However it may be the result of an intrinsic disturbance of stomach function such as atony or defective secretion. Ingestion of feedstuffs which have a tendency to swell in the stomach, particularly if they are not masticated properly, may be implicated. Dental problems, inadequate water supply and greedy eating may be contributory factors.

Successful evacuation of the stomach, be it by medical or surgical means, usually resolves the problem provided any predisposing causes are identified and corrected.

In contrast, chronic impaction of the stomach which appears to develop slowly over several weeks or even months has a very poor prognosis (Edwards 1997, Huskamp, Scheidemann and Schusser 2000). Diagnosis is difficult in the early stages. Despite the extreme gastric filling, affected horses show very little or no evidence of overt abdominal pain. They continue to eat and defaecate but tend to lose weight. Salivation and bruxism may occur. Although the presence on gastric endoscopy of fibrous ingesta, which does not appear to be reduced after 24 hours starvation, is suspicious, the true extent of the impaction cannot be determined by this means. However, the grossly enlarged and very firm stomach with time may be palpated on rectal examination together with caudal and medial deviation of the spleen. The size of the stomach may be gauged more accurately by radiographic and ultrasonographic examination. A lateral radiograph of the cranial abdomen showed the stomach extending caudally from the diaphragm to the level of the 13th or 14th intercostal space in several cases. The sonographic appearance is of a markedly enlarged gastric echo extending over six or more intercostal spaces on the left side of the abdomen. A marked increase in the thickness of the wall of the stomach may also be imaged. Surgical evacuation of the gastric contents has been attempted. With the horse in dorsal recumbency, the impacted stomach can be felt extending back to midway between the xiphisternum and the umbilicus and is therefore readily accessible via a midline celiotomy. As much as 30 - 40 kg have been removed via a gastrotomy but postoperatively normal gastric motility was not restored and impaction recurred (Edwards 1997). Reduction of stomach volume by partial resection of its flaccid wall was similarly unsuccessful (Huskamp et al 2000). At post mortem examination there is marked transmural hypertrophy of the non-glandular region of the stomach particularly near the cardia, and large chronic ulcers along the margo plicatus. The contents of the body and fundic area are foetid and fermenting, and clearly have been retained for several weeks or longer. The most recently ingested food passes directly from the cardia to the pylorus.

**Gastric Neoplasia**
Squamous cell carcinoma (Tennant et al. 1982, Olsen 1992, McKenzie et al 1997), adenocarcinoma (Thoonan and Ide 1941), leiomyoma (Petit 1904, Ball 1906) and lymphosarcoma (Hayley and Sprakert 1983, MacKay et al. 1981) of the stomach have been reported in horses. Although squamous cell carcinoma is the most common gastric neoplasm in the horse (Meagher et al. 1974) only 3 per cent of carcinomas in horses are of gastric origin (Feldman 1932) which is in marked contrast to the incidence in man. Middle-aged and older horses are most susceptible. There is a reported 4:1 male:female ratio (Boulton 1987).

**Aetiopathogenesis**
The tumour usually originates in the squamous epithelium of the oesophagus or stomach, infiltrates the wall and projects like a cauliflower into the lumen. In humans there are dietary, genetic and environmental factors that may contribute to oesophageal cancer but because oesophageal and gastric cancers are so uncommon in horses, contributory factors are not known. The rate of growth and aggressiveness of alimentary squamous cell carcinoma in horses is variable. In some horses tumours remain localised within the stomach, whereas in others they may extend through the stomach wall and spread to adjacent liver and spleen or result in metastatic nodules in the abdominal and thoracic cavities (Freeman 1982).
Clinical Signs
Affected horses have a history of gradual weight loss, poor appetite and lethargy extending over a period of 2 - 6 weeks (Meagher et al. 1974). Abdominal pain is not usually a feature of gastric carcinoma but involvement at the cardia may result in dysphagia.

Ascites and ventral oedema may be a primary sign in a few horses so that despite weight loss the abdomen seems distended (Kraus 1932). Gastric scc has been associated with pseudoparathyroidism and hypercalcaemia (Meutens 1978). Anaemia (PCV 12 - 28 per cent) may develop as a result of haemorrhage into the stomach or depressed erythrogenesis with resulting pallor and increased heart rate. Faeces frequently test positive for occult blood. A fever to 40ºC may occur as a result of necrosis in the tumour and the respiratory rate be raised in response to metastatic masses in the thorax.

Many horses with scc have leucocytosis and hypofibrinogenaemia. Some will have hypoproteinaemia due to bowel inflammation and protein exudation whereas other cases will have hyperglobulinaemia.

Peritoneal fluid will vary from normal, if the tumour is confined within the stomach, to an exudate if it has spread. Neoplastic cells from a primary gastric scc occasionally will be observed in a sample of peritoneal fluid and will be large, poorly differentiated epithelial cells with a bluish ground-glass appearing cytoplasm (Wright’s stain).

Passage of a nasogastric tube may meet resistance if the tumour involves the stomach adjacent to the cardia. Neoplastic cells may be found in fluid recovered by gastric lavage. Endoscopy enables a positive diagnosis by direct visualisation and biopsy of the tumour (Brown et al. 1985, Tennant et al. 1982). Exploratory laparotomy or laparoscopy allows a more complete examination of the stomach and allows biopsy of the primary tumour mass or metastatic nodules. Percutaneous biopsy though the left seventh intercostal space is another option. Radiographs of the thorax may reveal fluid and a pneumogastrogram may be of value in delineating the intraluminal portion of the tumour (Freeman 1982). Ultrasonography from the left cranial abdomen may show thickening and abnormal echogenicity of the stomach wall or parenchyma of the liver or spleen if metastatic spread has occurred.

Prognosis
Successful treatment has not been reported in horses. By the time a diagnosis is made, the tumour has progressed beyond the point where any treatment is feasible and euthanasia is the only option.

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