

Large Animal VETERINARY Rounds®

MARCH 2007
Volume 7, Issue 3

AS PRESENTED IN THE ROUNDS OF THE DEPARTMENT OF LARGE ANIMAL CLINICAL SCIENCES
OF THE WESTERN COLLEGE OF VETERINARY MEDICINE, UNIVERSITY OF SASKATCHEWAN

Equine Gastric Ulcer Syndrome

By Fernando J. Marqués, DVM, Diplomate ACVIM

Equine gastric ulcer syndrome (EGUS) is a broad term used to describe a wide array of clinical symptoms, including erosions and ulcers in the distal esophagus, the glandular and nonglandular portions of the stomach, and the proximal duodenum.^{1,2} There is a high prevalence of EGUS in horses used in many competitive activities, such as racing, dressage, show jumping, endurance, and western performance.^{3,4} In the adult horse, classic complaints associated with EGUS are low-grade colic, poor body condition, and decreased performance. Foals with gastric ulceration usually demonstrate inappetence, bruxism, sialorrhoea, colic, diarrhea, and a tendency to lie in dorsal recumbency.^{1,2,5} This issue of *Large Animal Veterinary Rounds* discusses the prevalence, pathogenesis, and treatment of gastric ulcer syndrome in the horse.

Prevalence

Gastric ulceration affects a large number of horses and foals; the overall prevalence ranges from 25% to 50% in foals up to 2-months-old and between 80% to 90% for race horses in training.^{1,6} One study, based on endoscopic evidence, found that >50% of apparently normal horses had gastric lesions.⁷ The majority of gastric lesions in both foals and adult horses are reported in the squamous mucosa adjacent to the margo plicatus.^{1,6,7} More epidemiological studies are necessary to determine risk factors and to establish the real prevalence of EGUS in Western Canada.

Pathophysiology

One of the most striking features of the equine stomach is its small size in relation to the animal itself and to the amount of feed consumed daily. Normally, food consumption behaviour in adult horses grazing at pasture or in nursing foals is on a rather continuous basis.

The inner aspect of the equine stomach has two main regions, a large nonglandular region occupying the fundus and part of the body, and a glandular region that includes cardiac, proper gastric, and pyloric glandular zones. A stepped edge, the margo plicatus, divides the nonglandular and the glandular regions (Figure 1).⁸ There are specific inciting and protective factors involved in erosion and ulcer formation that differ between the areas of the gastroduodenal tract. An imbalance between the inciting and protective factors of the gastroduodenal mucosa can result in erosion or ulcer formation (Table 1).^{9,10}

Hydrochloric acid represents the primary intrinsic factor promoting mucosal damage, but bile acids and pepsin may also contribute to ulcer formation. On the other hand, along the gastroduodenal tract, protective intrinsic factors against ulcer formation include adequate mucosal blood flow, the mucus-bicarbonate layer, mucosal prostaglandin E₂, epidermal growth factor, and gastroduodenal motility.^{1,2,9,10} Ulceration or erosion of any portion of the stomach or proximal duodenum exposed to gastric acid is referred to as "peptic ulcer disease" (PUD).

The stratified squamous epithelial mucosa of the dorsal portion of the equine stomach has minimal intrinsic resistance to peptic injury and excess acid exposure is the predominant mechanism responsible for squamous mucosal erosion and ulceration. Intercellular secretion of bicarbonate ions



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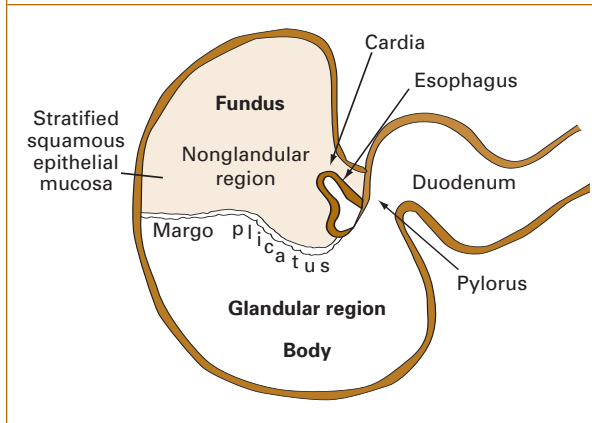
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The editorial content of *Large Animal Veterinary Rounds* is determined solely by the Department of Large Animal Clinical Sciences, Western College of Veterinary Medicine



The Canadian Veterinary Medical Association recognizes the educational value of this publication and provides support to the WCVM for its distribution.

Figure 1: Schematic view of the inner aspect of the equine stomach



and tight intercellular junctions, constitute the primary barriers to hydrochloric acid in the distal esophagus and dorsal nonglandular region of the equine stomach.^{1,2,11}

In the horse, hydrochloric acid is continuously secreted by parietal cells in the gastric glands via hydrogen-potassium adenosine triphosphatase (ATPase) pumps located on the luminal side. Gastric acid secretion is stimulated by gastrin secreted from G cells, by histamine secreted from enterochromaffin-like cells, and by acetylcholine via the vagus nerve (Figure 2).¹² The measured pH of the equine gastric content varies in relation to eating and nursing behaviour, with a pH of less than 2 in fasted horses and more than 6 in fed horses. Prolonged periods of acid exposure predispose horses and foals to mucosal erosion and ulceration, thus feeding practices and management can influence gastric pH (acidity) and squamous mucosal damage.^{1,2,12}

The gastric glandular epithelium is extremely metabolically active and is the location of gastrin-, histamine-, pepsin-, and hydrochloric acid-secreting cells. Protective mechanisms against mucosal ulceration in this region of the stomach include the mucus-bicarbonate layer, prostaglandins, nitric oxide, growth factors, mucosal blood flow, and cellular restitution. Mucosal blood flow is likely the most important factor protecting the gastric mucosa against ulcer formation. Nitric oxide, prostaglandin E, and cellular restitution represent key factors in blood flow regulation and thus, may play an important role in mucosal protection.^{2,13} In the gastric mucosa, bicarbonate ions are secreted into a thin mucus layer that has hydrophobic characteristics and protects against peptic injury. The pathophysiology of equine gastric glandular epithelium ulceration is not completely understood. The most common location for ulcerations and erosions in the gastric glandular region is the antrum and adjacent to the pylorus. In experimental models, nonsteroidal anti-inflammatory drugs (NSAID) can induce gastric ulcers, but this is not a common cause of gastric ulcers in most horses.^{2,14} In contrast to humans, gastric ulcers due to *Helicobacter pylori* infec-

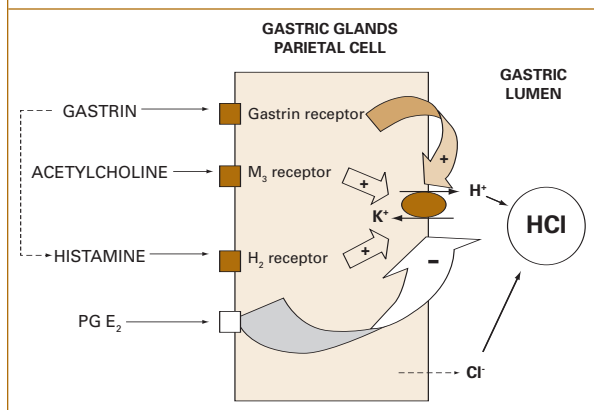
Table 1: EGUS: Inciting and protective factors involved in the gastroduodenal mucosal environment

Intrinsic protective factors	Intrinsic inciting factors
• Adequate mucosal blood flow	• Hydrochloric acid
• Mucus-bicarbonate layer	• Acetylcholine
• Mucosal prostaglandin E ₂	• Bile acids
• Tight intercellular junctions	• Pepsin
• Epidermal growth factor	
• Gastroduodenal motility	

tion could not be definitely identified in horses or foals with EGUS.^{1,2}

The high prevalence of gastric ulcers in neonatal foals suggests that gastric ulcer formation, particularly affecting the squamous epithelium, could be related to normal maturation and normal gastric development occurring during the first days of life. At birth, the equine gastric squamous epithelium is thin and not highly keratinized, and the measured stomach pH is less acidic than when measured at an older age. The equine squamous mucosa becomes hyperplastic and parakeratotic within days, correlating with a decrease in gastric pH and concurring with observations in other species. It is important for clinicians to be familiar with the differences in the normal appearance of squamous gastric mucosa in neonatal foals when performing endoscopic examinations. Desquamation of the squamous epithelium can be seen by endoscopy during the first 4-weeks of life in foals.⁵ Increasing gastric acidity temporarily parallels the normal process of squamous epithelial proliferation with minimal acidity during the first days of life and with marked acidity measured by 7 to 14 days of age. Gastric pH can also be very low in neonatal foals, particu-

Figure 2: Main factors regulating gastric acid secretion.



Gastrin, acetylcholine and histamine promote acid secretion via hydrogen-potassium ATPase pumps. Note that gastrin also stimulates histamine secretion via enterochromaffin-like cells. Prostaglandins of the E series inhibit acid secretion.

larly between nursing periods, with gastric pH dropping to less than 2 within minutes of nursing cessation.^{1,2,15} The combination of a thin squamous epithelium undergoing normal maturation and proliferation, and high acid output and acid exposure may make neonatal foals susceptible to gastric ulceration.

The higher prevalence of gastric ulcers in critically ill neonatal foals leads to the possibility that illness is an important factor in the pathogenesis of glandular mucosal lesions in neonatal foals. Stress is thought to positively influence the development of gastric ulceration in humans, and there is probably a link between illness and stress in foals; however, the exact mechanism of stress ulcer formation is not well understood. Illness is the only stress directly associated with a higher incidence of gastric ulcers in humans and horses. It is possible that other sources of stress such as intense training programs can indirectly play a role in EGUS.^{1,2}

The main protective mechanisms against erosion and ulceration in the esophageal squamous mucosa for several species are the tight intercellular junctions and bicarbonate secretion, although the latter has not been documented in horses. The main barrier against peptic injury is a glycoconjugate substance from cells present in the stratum spinosum, with a contribution of tight junctions in the stratum corneum. A normal salivary flow, a functioning lower esophageal sphincter, and salivary mucins contribute to peptic injury protection in human beings. In contrast, horses do not have a lower esophageal sphincter to act as a mechanical barrier.²

One interesting aspect is that most gastric ulcers in horses develop along the margo plicatus, particularly in the lesser curvature of the stomach that coincides with the normal gastric fill line just below the cardia.² Considering that duodenogastric reflux normally takes place in the horse,^{1,2} bile salts and pepsin may contribute to gastric ulcer formation as it does in many other species. In summary, risk factors for EGUS development include:

- intermittent feeding
- increased exercise intensity
- dietary factors such as high-concentrate, low roughage diets.

Clinical syndromes

Neonatal foals

Clinical signs classically associated with gastric ulcers in neonatal foals include bruxism, sialorrhea, inappetence, colic, diarrhea, and a tendency to lie in dorsal recumbency. Only a small number of foals with endoscopic evidence of EGUS show clinical signs, therefore, when clinical signs are observed, severe ulceration probably exists. Most gastric lesions in foals are found in the gastric squamous mucosa.^{6,10}

Physiologic stress associated with concurrent illness has been linked with gastric ulceration in neonatal foals. In this group of foals, gastric lesions are usually located in the

glandular epithelium in the body of the stomach. Only 4% to 9% of clinically normal foals have lesions in the gastric glandular mucosa when examined by endoscopy.^{1,2,5,6,10}

Sucklings and weanlings

The squamous mucosa adjacent to the margo plicatus along the greater curvature of the stomach is the most common location for gastric ulcers in foals less than 50-days-old. Similar lesions can be found in foals as young as 2-days of age. Histologically, these gastric lesions are characterized by neutrophilic inflammation and disruption of the epithelial layers of the mucosa.²

In older foals, squamous epithelial lesions are more prevalent along the margo plicatus in the lesser curvature of the stomach. When severe, these lesions can be associated with diarrhea, poor appetite, poor growth, and poor body condition.

Foals with duodenal ulceration usually present similar clinical signs as the ones associated with gastric ulceration, such as colic, bruxism, salivation, and diarrhea. Delayed gastric emptying and gastroesophageal reflux are common in this foal population, although the cause of duodenal ulceration in foals is unknown.^{1,2}

Yearlings and adult horses

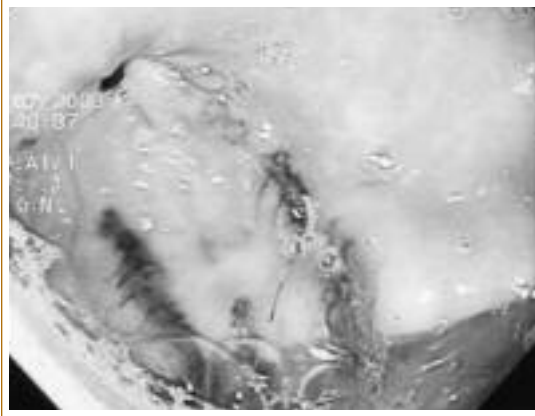
Clinical signs in older horses vary. Many adult horses with endoscopic evidence of EGUS may not demonstrate visible clinical signs or the signs may be so subtle or vague that they are unnoticed by the owner or trainer. Other horses may show more typical clinical signs such as low-grade discomfort, anorexia, and chronic or intermittent colic of varying degrees. Gastric ulcers are not usually a cause of diarrhea in the adult horse, although horses with diarrhea due to other causes may have concurrent EGUS. Attitude change, alterations in behaviour, and poor performance in the older horse are also signs that could cause a clinician to suspect EGUS.

Lesions are most commonly located in the gastric squamous mucosa, particularly along the margo plicatus. Deep gastric ulcers involving the squamous mucosa may bleed; however, bleeding ulcers in horses are not a cause of anemia or hypoproteinemia. Lesions in the pyloric region are also important to consider and, according to one study, the incidence of lesions in this area may be higher than previously reported.^{1,2}

Diagnosis

History, along with age-related clinical signs, can be suggestive of EGUS, although a definitive diagnosis requires endoscopic examination. In the adult horse, a 3-meter endoscope is necessary for performing a complete exam (ie, observation of the entire stomach, pylorus, and proximal duodenum). The Equine Gastric Ulcer Council recently published a consensus for classifying EGUS lesions; they propose a scoring system that ranges from Grade 0, an intact

Figure 3: Endoscopic view of the equine stomach showing small areas of superficial lesions



epithelium with no appearance of hyperemia or hyperkeratosis, to Grade 4, an epithelium with extensive lesions and areas of deep ulceration (Figure 3).^{1,2,17}

Sucrose is not absorbed through an intact gastrointestinal tract. A urine sucrose test and a serum sucrose test were recently investigated and both tests can identify horses with endoscopically visible gastric ulceration. Thus, they have the potential to be used as noninvasive methods for screening and monitoring horses with gastric ulcers.^{18,19}

Treatment

As in any other disease, to design a treatment plan, the pathophysiology of the disease should be examined and the most important and relevant pathophysiologic components targeted. Excess acid exposure is the predominant mechanism responsible for squamous mucosal erosion and ulceration; therefore, most anti-ulcer therapies aim to suppress or neutralize gastric acid. Therapeutic options for treating gastric ulcers in horses include:^{1,2,17}

- H₂ antagonists such as cimetidine, ranitidine, famotidine, etc.
- Proton pump blockers such as omeprazole
- Mucosal protectants such as sucralfate
- Antacids.

The mechanism of action with **H₂ antagonists** is the suppression of hydrochloric acid secretion through competitive inhibition of the parietal cell histamine receptor. The use of H₂ antagonists was proven to successfully raise the gastric pH and resolve gastric ulcers in foals and adult horses; however, there are large individual variations in the degree and duration of acid suppression by H₂ antagonists among horses.^{20,21}

Given the high incidence of gastric ulcers in critically ill neonatal foals, it is common practice to routinely and prophylactically administer anti-ulcer drugs

in this foal population. Sick neonatal foals demonstrate variable gastric pH responses to ranitidine, administered intravenously; some foals reveal a brief duration of action and, in other cases, there is no response. In addition, some critically ill foals have a more alkaline gastric pH and, with the possibility that gastric acidity plays an important role in preventing bacterial translocation in neonates, the use of prophylactic anti-ulcer therapy in neonatal foals remains controversial.^{1,2,22}

Proton pump inhibitors, such as omeprazole, irreversibly bind to the hydrogen-potassium ATPase pump and block secretion of H⁺ at the parietal cell membrane. Omeprazole demonstrated good efficacy in healing NSAID-induced gastric ulcers as well as naturally occurring cases of EGUS. Further, omeprazole was effective in eliminating or reducing the severity of gastric ulcers in Thoroughbreds undergoing intensive training. Omeprazole can be safely used in either adult horses or foals; however, commercially available preparations are relatively expensive. Recently, the efficacy of compounded preparations has been questioned.²³

Sucralfate is effective in treating peptic ulcers in humans and it likely adheres to ulcerated mucosa, stimulating mucous secretion, prostaglandin E synthesis, and increasing concentrations of growth factor at the ulceration site. These are all factors involved in the gastric glandular mucosa and the efficacy of sucralfate in treating ulcers in the equine gastric squamous mucosa remains unknown. Sucralfate could be an effective therapy for stress-induced ulcers that occur in the glandular gastric mucosa of neonatal foals, but no clinical evidence supports this assumption.^{1,2,17}

Antacids can be used to reduce gastric acidity in horses, but their effects are relatively short lived (lasting for approximately 2 hours). Treating gastric ulcers in a standard adult horse with liquid antacid products requires the administration of large volumes of product several times a day.

Synthetic prostaglandin E₁ analogs (eg, misoprostol) are used in human medicine to treat gastric and duodenal ulcers. The proposed mechanism of action is inhibition of gastric acid secretion and mucosal cytoprotection. Adverse effects in humans include abdominal discomfort, diarrhea, and inappetence; therefore, misoprostol is not routinely used to treat gastric ulcers in horses.

Prokinetics should be considered and potentially included in the therapy plan when delayed gastric emptying without a physical obstruction is suspected, as well as in foals with duodenal disease and gastroesophageal reflux. Bethanechol, a cholinergic drug, increases the rate of gastric emptying in horses. Adverse effects include diarrhea, inappetence, salivation, and colic, but they seldom occur.^{1,2,17}

Summary

EGUS could be a cause of poor performance and colic in a wide range of equine athletes.²⁴ Clinical signs can be very vague and subtle. Based on previous studies, a large number of clinically normal horses not in training may also have EGUS. The negative impact of EGUS on this population of “normal” horses is not perceived until after the horse is diagnosed and treated.

EGUS affects horses of all ages and represents an important economic and clinical concern. More epidemiological and clinical studies are necessary to determine the real prevalence and importance of EGUS in Western Canada.

Dr. Fernando Marqués is an Associate Professor of Large Animal Internal Medicine in the Department of Large Animal Clinical Sciences at the Western College of Veterinary Medicine. His research interests include infectious diseases, sepsis and inflammation, the immune response to infection, and vaccine formulation.

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Abstracts of Interest

Gastric ulcer development in horses in a simulated show or training environment

McCLURE SR, CARITHERS DS, GROSS SJ, MURRAY MJ. AMES, IOWA

OBJECTIVE: To determine whether conditions representing activities that are typical in the recreational use of horses, including transport to and from show grounds, stall confinement in unfamiliar surroundings, and light exercise, are associated with increased incidence of gastric ulcers in horses.

DESIGN: Randomized controlled study.

ANIMALS: 20 client-owned horses.

PROCEDURE: Horses had no gastric ulcers as determined by endoscopic examination on study day -1. Ten control horses were maintained on-site with no changes in management variables. Ten horses were transported via trailer for 4 hours on day 0 to another site, placed in individual stalls, fed twice daily, and exercised twice daily for 3 days. On day 4, they were transported back to the original site via trailer for 4 hours. On day 5, endoscopic examinations were performed on all horses to assess gastric mucosa status.

RESULTS: Horses that were transported and housed off-site had a significantly higher incidence of hyperkeratosis and reddening of the gastric mucosa than control horses. Two control horses and 7 transported horses developed gastric ulcers by day 5. Ulcer scores of transported horses increased significantly from day -1, whereas ulcer scores in control horses did not change significantly from day -1.

CONCLUSIONS AND CLINICAL RELEVANCE: Activities that are typical in recreational use of horses were ulcerogenic, and ulcers in the gastric squamous mucosa can develop under these conditions within 6 days.

J Am Vet Med Assoc 2005;227(5):775-777.

Sucrose concentration in blood: a new method for assessment of gastric permeability in horses with gastric ulceration.

HEWETSON M, COHEN ND, LOVE S, BUDDINGTON RK, HOLMES W, INNOCENT GT, ROUSSEL AJ. GLASGOW, SCOTLAND, UK.

A urine sucrose test has recently been reported to be a reliable method of detecting gastric ulcers in horses; however, technical difficulties associated with urine collection have limited the practical value of the test. The objective of this pilot study was to determine whether gastric sucrose permeability, as evaluated by serum sucrose concentration, could be used to detect gastric mucosal injury in horses. Twelve adult horses with naturally acquired gastric ulceration were studied. After a 20-hour nonfeeding period, each horse was dosed with 250 g of sucrose via nasogastric intubation. Blood samples were collected at 0, 15, 30, 45, 60, and 90 minutes, and horses underwent gastroscopy 4 hours later. The severity

of gastric ulceration in each horse was defined by means of a 4-point ulcer-scoring system, and the relationship with serum sucrose concentration was analyzed by means of a linear mixed-effects model. Serum sucrose concentration was measured by liquid chromatography operating in tandem with electrospray mass spectrometry. After nasogastric administration of table sugar, horses with moderate to severe gastric ulceration had significant increase in serum sucrose concentration at 30, 45, 60, and 90 minutes, relative to earlier times ($P < .05$). Peak sucrose concentration was observed at 45 minutes, and was correlated with ulcer severity (Spearman's rank correlation coefficient = 0.898, $P < .05$). These data indicate that determination of sucrose concentration in equine serum may be a useful test for identifying horses with endoscopically visible gastric ulceration and has potential use as a noninvasive method for screening and monitoring horses engaged in racing training and other performance-related disciplines.

J Vet Intern Med 2006;20(2):388-94.

Prevalence of gastric squamous ulceration in horses with abdominal pain

DUKTI SA, PERKINS S, MURPHY J, BARR B, BOSTON R, SOUTHWOOD LL, BERNARD W. LEXINGTON, KENTUCKY

REASONS FOR PERFORMING STUDY: Prevalence of gastric ulcerations differs widely according to breed and circumstances of management. Further study of the biological variables involved is required in order to identify more exactly the reasons for the reported range.

OBJECTIVES: The objectives of this present study, which do not appear to have been addressed previously in the literature, were 1) the prevalence of gastric ulceration in horses with abdominal pain. 2) difference in prevalence in horses responding to medical therapy and those requiring surgical intervention. 3) whether gastric ulceration is associated with any particular gastrointestinal tract lesion.

METHODS: Horses were included in the study if gastroscopy was performed within 24 h of presentation. The presence and grade of gastric ulceration was recorded together with the medical records. Data were analysed categorically using a Fisher's exact test or Chi-squared test.

RESULTS: One hundred horses met the selection criteria. Forty-nine percent (49/100) of horses had gastric ulceration, 63% (63/100) responded to medical therapy and 37% (37/100) to surgical intervention, and prevalence was higher in the former (59%) than the latter (32%). Horses with duodenitis-proximal jejunitis (DPJ) had a trend towards higher prevalence of gastric ulceration compared to those with other GI lesions. Sixty-eight percent (13/19) of horses diagnosed with DPJ, 32% (8/25) with a large colon impaction and 14% (1/7) with large colon volvulus had gastric ulceration.

CONCLUSIONS: The presence of gastric ulceration in all horses with abdominal pain was moderate. Horses responding to medical therapy had a higher prevalence of gastric ulceration compared to horses requiring surgery and there was a trend towards higher prevalence in cases of DPJ compared to other GI lesions.

There was not a statistically significant difference in gastric ulceration detected between specific lesions, including large colon impactions and large colon volvulus.

POTENTIAL RELEVANCE: The clinical relevance of ulceration is still unclear and further studies are required to differentiate between incidental and clinically important gastric ulceration.

Equine Vet J 2006;38(4):347-349.

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Dr. Marqués has stated that he has no disclosures to announce in association with the contents of this issue.

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This publication is made possible by an educational grant from

Schering-Plough Canada Inc.

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