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# Advances in Equine Nutrition

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J.D. Pagan

## COLIC

MIKE BEYER
*Hagyard-Davidson-McGee Veterinary Clinic, Lexington, Kentucky, USA*

Colic is any abdominal pain. The causes are numerous and many unidentified, but in general they are related to the anatomy of the horse's gastro-intestinal tract (GI) and the balance of bacterial flora in the gut. From a veterinarian's viewpoint the nutritionally-related causes lie in the disruption of the bacterial flora in the gut and its consequences. Two scenarios can lead to an abrupt change in the bacterial flora: first, feeding improper ratios of forage and grain; second, feeding tainted forage or grain (moldy hay or grain).

To better appreciate the mechanism of colic keep in mind the anatomy of the horse's GI tract; they are hindgut fermenters, more like a rabbit than a dog. The path of ingesta through the horse's GI tract is oropharynx, esophagus, stomach, small intestine, cecum, large colon, small colon, rectum. The cecum and large colon are large segments of the tract which allow for fermentation and absorption of nutrients after partial digestion in the stomach and small intestine. These large segments are prone to malfunctions in position, absorption, and motility, all of which can be nutritionally related.

The first category, an improper position, can sometimes potentially be attributed to diet. A change in diet which causes increased gas production can cause part of the intestinal tract to become buoyant as well as create abnormal motility which in turn leads to a change in position. Displacement or torsion is most common in the large colon. The large colon forms two horseshoe shaped loops (one on top of the other) on the floor of the abdomen with little tissue attachment and can thus almost be free-floating. Being displaced with gas buildup is one potential cause of large colon displacements, torsions, and nephrosplenic entrapments. Some simple displacements can be corrected without surgery whereas other lesions including torsions must be corrected surgically.

The second category, malfunctions of motility, can be due to the feed consistency and content. We will often see impactions or increased transit time due to lack of moisture or too much coarse material. Referrals in our practice show a 30-50:1 ratio of impactions in confined horses versus farm horses. Common sites of impaction include the pelvic flexure of the large colon, the transverse colon, and the cecum. All three sites are areas of abrupt change in the diameter of the colon.

The third category of causes of colic are those due to abnormality in absorption. If, for example, a horse is suddenly fed a large amount of grain, the time the ingesta

spends in the stomach and small intestine is significantly decreased because of the decrease in the amount of fiber in the diet. The hindgut, the cecum, and colon receive a large volume of highly digestible foodstuff, bacteria rapidly break the ingesta down, and the pH of the hindgut decreases. The bacteria produce a large volume of gas and can in turn have a large increase in numbers and subsequent die-off in bacteria leading to endotoxin release and mucosal damage. Mucosal damage reduces absorption and less fluid is absorbed. In this scenario both a faster transit time through the gut and a decrease in the amount of absorption can lead to spasmodic gut activity, colic, and even diarrhea.

Treatment of the cases of colic associated with diarrhea consists of compensating the horse for its fluid losses, treating potential secondary complications, and attempting to reverse the process which led to the diarrhea. Fluid replacement is accomplished through aggressive IV fluid therapy.

Secondary complications include laminitis, endotoxemia, and peritonitis if the bowel is compromised to the point of leaking.

Once the horse is maintained through the acute phase of diarrhea, we attempt to improve absorption by repopulating the gut with normal bacterial flora and secondarily by slowing or altering the transit time of ingesta. The tactic for slowing the transit time consists of feeding a ration high in dry matter and fiber in an attempt to pull some of the excess fluid in the gut into a foodstuff that is not readily broken down and stays in the hindgut for a longer period of time. One such foodstuff that we recommend is beet pulp, especially in chronic diarrheas. Any feed that is high in fiber, low moisture, and relatively absorptive or hygroscopic will meet this need.

The interplay of increased transit time through the gut and mucosal damage especially to portions of the hindgut explains why the most significant complication of diarrhea is laminitis. With compromised GI mucosa, the toxins in the gut and endotoxins released from the breakdown of gram negative bacteria cell walls can readily enter the systemic circulation leading to toxemia and endotoxemia. Endotoxic horses suffer from numerous pathological responses to the systemic insult.

In diarrhea cases treatment is directed towards ameliorating some of the effects of endotoxin and consequently indirectly addressing the potential for laminitis. Endotoxins in the systemic circulation lead to vasoconstriction especially in some of the smaller peripheral vascular beds, release of tumor necrosis factor, elaboration of clotting factors, and increased tendency to form microthrombi in small vessels. To oversimplify: because there is an insult to the body systemically blood supply is preserved to the vital, major organs and sacrificed to the peripheral, less essential vascular beds. One of these beds is that of the laminae of the hoof wall.

Endotoxemia is treated through fluid therapy, use of nonsteroidal anti-inflammatory drugs, use of rheologic drugs to decrease blood viscosity, and use of peripheral vasodilators to preserve blood supply to the peripheral vascular beds. The most commonly used drugs at our clinic are flunixin meglumine, pentoxifylline, and occasionally aspirin to help decrease the likelihood of microthrombi.

The most common presentations of feed-related colic and diarrhea at this practice are middle-aged adult horses who have either had an abrupt change in diet, have recently been shipped (deprived of free access to water and have become impacted) or have recently been on a course of antibiotics which alter the gut flora. Correction lies in providing digestible fiber to the hindgut and repopulation of the gut with normal bacterial flora. We have little we can offer with feed and basically rely on natural repopulation of the gut. It is questionable whether any live bacterial cultures reach the hindgut and survive the acidity of the stomach, but we will sometimes use yogurt to attempt to replace bacteria in the gut.

Many colic disturbances may be able to be traced back to changes in GI motility or bacterial flora. The horse was designed to be a grazer continually ingesting small quantities of forage all of the time. In our attempts to feed the highest quality rations for maximum performance we cannot overlook the potential complications caused with different feeding regimes.

## Laminitis

Laminitis by definition is any inflammation of the laminae that interdigitate between the hoof wall and P-3. These laminae can become inflamed in any generalized systemic condition which can affect peripheral circulation such as endotoxemia, septicemia, severe dehydration, and cardiovascular shock. Experimentally induced laminitis models are those caused by carbohydrate overload and black walnut induced toxicity (Praase, 1990 and Galey, 1991). Other causes include mechanical stress, endocrine disease, steroids, and some predisposition as seen in some ponies (Byars, 1995).

By far the most common cases of laminitis we see in the clinic situation are those that occur secondarily to diarrhea, especially colitis which compromises the gut mucosa and allows for absorption of endotoxins from the GI tract. As these toxins are absorbed into the circulation, the blood supply to the foot changes and the small laminae are compromised through poor circulation and ischemia (or deprivation of oxygen) to their tissues. Once signs of laminitis are obvious, the pathological process of ischemia and vessel endothelial damage due to venoconstriction or microthrombosis is already in effect. In practice the most successful intervention we can offer is a prophylactic one aimed at preventing the onset of the process.

Any therapies directed towards abating the effects of inflammatory mediators and circulatory changes have a therapeutic potential. Primarily used are anti-inflammatories such as flunixin meglumine (Banamine) or phenylbutazone (Bute), vasodilators such as acepromazine for short term or isoxsuprine for long term therapy, anticoagulants such as heparin, and pentoxifylline which blocks the production of tumor necrosis factor and decreases the viscosity of the blood. In cases of diarrhea/colitis with laminitis as a sequela, the prevention of absorption of endotoxins from the gut may address the potential cause. Experimentally, however, sublethal doses of endotoxin have not been shown to induce laminitis.

Clinically the most successful treatment occurs very early in the disease process or even prophylactically. Medically we can effect decreased blood viscosity, vasodilation, and decreased systemic inflammation. Nutritionally we have to effect a decreased absorption of endotoxin from the GI tract, repopulation of the bacterial flora of the gut, and increased transit time of ingesta through the hindgut via increased fiber content and less acidic pH in the colon.

Once the laminae are affected to the point of partial separation between the hoof laminae and those of P-3, the prognosis of an athletically sound horse decreases (Stick in Kobluk, 1995).

In practice we rarely see laminitis in neonates, seldom see it in weanlings, and commonly see it in adults. Ponies are prone to laminitis as are heavily muscled stallions, obese horses and endotoxic adults. Nutrition can play a role both in initiating laminitis either through precipitating an abrupt change in bacterial flora and releasing an endotoxic shower or directly through a carbohydrate overload. It is also necessary to nutritionally manage the laminitic horse to reduce the numbers of endotoxin-producing bacteria and increase hindgut fermentation. The myriad of treatments we have to implement for laminitis indicate the limited success of any one treatment modality.

## Ulcers

Gastric ulcers were first recognized as a serious clinical entity by Rebhun in 1982. Since then, however, they have become widely recognized as a clinical problem in young adults as well due to the decrease in performance among racehorses.

Two regions of the stomach can be ulcerated. The nonglandular portion or the squamous epithelial portion is primarily ulcerated due to increased gastric acidity whereas ulceration of the mucosal glandular portion is due to a compromise in mucosal protection. Prostaglandin E is required for normal gastric mucus production. Non-steroidal anti-inflammatory drugs inhibit the production of prostaglandin E and hence the maintenance of the protective mucosal barrier of the glandular portion of the stomach.

In racehorses in training usually 2-5 years of age we see ulceration of the non-glandular portion of the stomach more frequently. This portion is also the most readily visible portion with the endoscope. The causes for gastric ulceration are numerous. In the young racehorse it is thought to be related to increased stress and feeding regimes. Most often these horses are being fed large quantities of grain with reduced quantities of hay and in 2 or 3 large feedings per day. Studies have shown that horses fed grain increase gastric acid production in the stomach as compared to those fed hay (Murray, 1994).

Adults with gastric ulcers usually present as having mild colic signs sometimes associated with eating, unthriftiness, and poor performance. Murray suggests that the activity of eating small amounts, often, decreases the acidity of the stomach.

Conversely acidity is greatest in horses that are held off of feed altogether through increased gastrin production. Therefore feeding a large quantity of grain 2-3x/day may exacerbate the tendency of horses in training to have hyperacidic stomachs and thus leads to the increased incidence among this group of horses. Again the diet best suited to prevention is one which has more forage than grain.

Our treatment for this group of horses consists of altering the feeding regime as much as is practical and instituting anti-ulcer medication with sucralfate and H2 antagonists. The H2 antagonists are cimetidine (Tagamet), ranitidine (Zantac), and famotidine (Pepcid). We commonly use ranitidine or cimetidine in the adult. An effective but expensive drug currently available is omeprazole (Prilosec). As a proton pump inhibitor it is especially effective against both squamous and mucosal ulceration.

Foals are prone to gastric ulceration, yet it is generally considered stress-induced hyperacidity that leads to both gastric and duodenal ulceration in foals. Foals affected are often not weaned and whose diet consists primarily of the mare's milk. It is therefore questionable whether diet is a factor. There is, however, a positive association of Rotavirus and other causes of diarrhea with the incidence of gastric ulcers in foals.

Foals are also treated with H2 antagonists (usually ranitidine) and sucralfate if ulcers are thought to already be present. Clinically affected foals can show signs including bruxism, excess salivation, colic after nursing, dorsal recumbency, diarrhea, weight loss, and pot-bellied appearance.

In general clinically significant ulceration has decreased in foals in this area probably as a result of widespread anti-ulcer use on farms. Ulceration in adults, however, is a more frequently diagnosed problem often as a reason for poor performance in training. Feeding regimes and perhaps feeds that would reduce the acidity of the stomach may be helpful in decreasing the incidence among young adults.

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