Nutritional Support of Horses with Colic

AMELIA MUNSTERMAN
Auburn University, Auburn, Alabama

INTRODUCTION

Nutritional support of the critically ill patient is no longer seen as an adjunct therapy. Recent studies in humans support the fact that early and adequate nutritional support can reduce complications, shorten the duration and severity of the disease, and improve patient outcomes. However, it is important to note that even in human medicine recommendations for nutritional support are limited by the heterogeneity of the patient populations, their illnesses, and statistical power. In the veterinary literature, these limitations are even more restrictive. This paper offers basic guidelines for nutritional support of adult horses with colic based on a review of available literature and expert opinion.

The Association Between Nutrition and Colic

The horse was designed to be a continuously grazing animal, with hindgut fermentation supporting the digestion of high-fiber, low-carbohydrate feeds. Modern horse management either neglects this fact or is unable to provide a lifestyle for the horse that its digestive system was evolutionarily refined for. Intermittent feedings, large boluses of cereal-based feeds, and stall confinement are the norm for most modern horses. While the horse is able to adapt to some extent, it is not unexpected that the adaptations that have allowed horses to live among us periodically fail. Nutrition is often implicated as the cause of gastrointestinal pain; however, the multifactorial nature of the problem, including types of feed, quality of feedstuffs, and variations in feeding practices make it difficult to pinpoint epidemiologically the true role of diet in colic.

In veterinary medicine, the search for the cause and effect of a condition is often hampered by financial limitations, small group sizes, and limited record-keeping. It is important, however, to take an unbiased and critical view of all the information provided, since the knowledge we gain will be used to directly influence the treatments provided. Evidence-based medicine is the practice of integrating unbiased research and clinical expertise, and applies a grading scheme for published literature. It acknowledges that all evidence is not created equal and requires careful consideration when applying that research to the clinical patient. In this paper, only studies with a level of evidence of grade 2 or higher were included to provide statistical evidence linking colic and nutrition. When assessing the evidence, the veterinary practitioner is cautioned to keep in mind the strengths and weaknesses of the published literature in order to make decisions based only on the most valuable information available. The first step in linking feeds to colic is to analyze the evidence related to the most common feeds provided to horses, including grasses, dried forages, and concentrates.

Feeds and Colic: Pastures

The horse was evolutionarily designed to eat grasses. The large colon, specifically, developed to ferment these grasses into the short-chain fatty acids (acetate, propionate, and butyrate) for energy (Bergman, 1990). While grasses have the capability of storing large quantities of carbohydrates as starches and fructans, which could upset the delicate microbial populations, continuous grazing should allow for the bacteria to respond to any changes in carbohydrate content gradually (Crowell-Davis et al., 1985; Boyd et al., 1988; Longland and Byrd, 2006; Durham, 2009). However,

access to pastures with high levels of fermentable carbohydrates has been implicated as the cause of colic and laminitis, and may relate to the apparent seasonality of colic events (Cohen et al., 1999; Longland et al., 1999; Hoffman et al., 2001). While grazing on pasture has been regarded as protective against colic for the horse, its effect may be tempered by other factors including water supply, weather, rate of feed intake, stocking density, quality of pasture, and supplements provided.

Pasture access was found to reduce the likelihood of colic in a case-control observational study of 364 horses, which noted a threefold increase in the risk of colic for horses with no pasture turnout or that had a recent reduction in paddock size or time at pasture (95% CI 1.4-6.6, P=0.007) (Hudson et al., 2001). A separate case-control study in Britain noted that stall confinement tended to increase the risk of colic (OR=9.30, 95% CI 1.68–51.40, P=0.011) with stabling for 24 hours per day associated with the greatest risk for colic (OR=35.2) (Hillyer et al., 2002). Lack of grazing activities may also predispose to specific types of colic, including enterolithiasis, which was noted to occur 2.8 to 4.0 times more frequently in horses that spent less than 50% of their time outdoors (Cohen et al., 2000; Hassel et al., 2008). Access to pasture did not reduce the risk of colic in a report by Reeves and coworkers (1996), but if water was not available in the paddock, it more than doubled the risk of colic (OR=2.2, 95% CI 1.2-4.3). Despite this published evidence, it is difficult to separate any beneficial effect of grazing activities from the patterns and timing of ingestion, the horse's activity levels, and the effects of exercise on intestinal motility.

Feeds and Colic: Dried Forages

Hay often provides a large portion of the modern horse's diet due to the confines of space and resources. While dried forages provide some consistency to the horse's diet, there is still variability that can occur between batches due to changes in source, the type of hay fed, and even the preservation process that produced the hay. Poor-quality forages consisting of high concentrations of hemicellulose, cellulose, and lignin increase the risk of impaction-related colic (Cohen and Peloso; 1996; White and Dabareiner; 1997; Hudson et al., 2001; Little and Blikslager; 2002). Feeding hay from round bales may also increase the risk by 2.5 times (95% CI 1.1-5.6, P=0.028), likely due to the methods of preservation, exposure to the elements before and during feeding, and the unchecked quantities available to the horse (Hudson et al., 2001).

Abrupt changes in the type of hay or a batch of hay has been noted in the literature to be a common cause of colic. In a case-control study of horses experiencing colic in Texas, it was noted that while a change in feed within the previous two weeks was significantly associated with colic (OR=5.0, 95% CI 2.6-9.7, P<0.001), a change of hay increased the risk even further (OR = 9.8, 95% 1.2-81.5, P=0.035) (Cohen et al., 1999). This was confirmed in an additional study in 2001 (OR=4.9 95% 2.1-11.4, P<0.001) by Hudson et al. On the Eastern seaboard, a change in diet was again linked to colic, specifically with a change in hay, resulting in a 2.1-times increase in colic incidence (95% CI 1.2-3.8, P=0.01) (Tinker et al., 1997).

The specific type of hay also may be an important factor in the risk of colic. Coastal Bermuda hay has been implicated in one cohort study as a cause of colic (OR=1.65, 95% CI 1.01-2.7, P=0.045) (Cohen and Peloso, 1996), and has been suggested by numerous retrospective case series to result in impactions of the ileum (Embertson et al., 1985; Pugh and Thompson, 1992; Hanson et al., 1998). Coastal Bermuda hay was confirmed as a risk factor for ileal impactions in a retrospective case-control study by Little and Blikslager (2002), who found that horses fed coastal Bermuda had a 4.4-times higher risk of this type of colic (95% CI 2.1-9.1) versus noncolic controls, a 5.7-times higher risk (95% CI 2.4-13.6) versus medical colic, and a 2.7 times higher risk (95%

CI 1.2-6.5) versus surgical colic. However, this study also noted that feeding this type of hay alone did not increase the risk of colic in general, and diluting it with other hays did not reduce the risk of ileal impactions.

Alfalfa hay has been associated with an alkalinizing effect on the colon ingesta, resulting in alterations in colonic microflora and a reduction in volatile fatty acid production, which provides an environment suited to the formation of enteroliths (Hintz et al., 1988). In one study, the odds of enterolithiasis were increased if the diet was comprised of more than 50% alfalfa (OR=4.2, 95% CI 1.3-12.9, P=0.01) (Cohen et al., 2000). Two additional studies confirmed this finding, supporting the restriction of alfalfa hay from the diet of horses at risk (Hassel et al., 2004, 2008). One possible explanation is that the high magnesium content of the alfalfa contributes to the alkalinizing effects of the hay on the colon contents (Hassel et al., 2004, 2009a). In addition, the high protein content may decrease magnesium absorption and increase ammonia available to precipitate formation of enteroliths (Murray et al., 1992). Conversely, grass hays may be useful for prevention of enteroliths and were noted to have protective effect if greater than 50% of the diet (Hassel et al., 2008).

Feeds and Colic: Concentrates

Carbohydrate-rich feeds, including grains, are the most commonly implicated dietary cause of colic in the horse, likely due to the well-documented influences of this substrate on the flora of the equine gastrointestinal tract (Shirazi-Beechey, 2008; Durham, 2009). While the carbohydrate source is much more abundant in grains, there is also a significant difference in the type of carbohydrate present in these feeds versus forages. In general, carbohydrates can be divided into two groups: rapidly hydrolysable carbohydrates (starches, hexoses, disaccharides, and some oligosaccharides), which are primarily degraded in the small intestine, and fermentable carbohydrates degraded by bacterial populations to the short-chain fatty acids (acetate, propionate, and butyrate) in the large intestine and cecum (Hoffman et al., 2001). Grains contain larger quantities of rapidly hydrolysable carbohydrates.

Hydrolysable carbohydrates presenting to the small intestine are normally degraded by pancreatic alpha amylase to oligosaccharides, which are further digested by the brush-border enzymes to glucose for absorption (Roberts, 1974; Shirazi-Beechey, 1995; Dyer et al., 2002). While glucose transport by the enterocytes can be improved by adaptation to high-starch feeds, monosaccharides produced by amylase are required to stimulate this adaptive process (Kienzle et al., 1994). However, the overall activity of amylase in the horse is low compared to other species (Lorenzo-Figueras et al., 2007) and highly variable between horses (Roberts, 1974; Kienzle et al., 1994). The lack of sufficient amylase enzyme has been proposed as the rate-limiting factor in accommodation of carbohydrate digestion in the horse (Richards, 2004).

Research has supported this conclusion by documenting that starch intakes exceeding 0.4% of body weight in one meal can allow hydrolysable carbohydrates to present to the lumen of the hindgut of the horse (Potter et al., 1992). In the cecum and colon, they are fermented to lactic acid by saccharolytic bacterial species, reducing colon pH and therefore the survival of the bacteria required for fiber fermentation (*Clostridiaceae, Fibrobacter,* and *Spirochaetaceae*) (Goodson et al., 1988; Julliand et al., 2001; Medina et al., 2002). The overall changes in the microbial environment can alter fermentable carbohydrate digestion (Drogoul et al., 2001), reduce the production of volatile fatty acids (Hintz et al., 1971; de Fombelle et al., 2001; Julliand et al., 2001; Medina et al., 2002), dehydrate the ingesta (Clarke et al., 1990b; Lopes et al., 2004), increase transit times (Lopes et al., 2004), and result in gas distention of the large intestine (Shirazi-Beechey, 1995), all

predisposing the horse to colic.

In clinical patients, the association of concentrates with colic was variable, having a significant effect in one (OR=2.6, 95% CI 0.9-7.2, P=0.064) (Hudson et al., 2001) but none in a second study (Cohen et al., 1999). While feeding more than 2.7 kg of oats per day increased the risk in Hudson's study (OR=5.9, 95% CI 1.6-22, P=0.009), previous work had noted that any whole grain (oats, barley, etc.) was associated with an increased risk of colic if fed in amounts greater than 2.5 kg per meal (OR=4.8, 95% CI 1.4-16.6, P=0.01) (Tinker et al., 1997). Others have noted that only whole corn was a risk factor (Reeves et al., 1996). Pelleting the grain may increase the risk of colic based on some studies, but others were not as clear (Morris et al., 1986; Tinker et al., 1997; Little and Blikslager, 2002). However, changes in the concentrate fed were shown statistically to increase the risk of colic (OR=3.6, 95% CI 1.6-5.4, P<0.001) (Tinker et al., 1997). Current recommendations regarding concentrates advise to provide less that 0.2% of body weight per meal to prevent adverse effects on digestion (Hussein et al., 2004).

General Practices to Prevent Colic

Prevention of colic should be centered on providing a consistent diet, since a recent change in diet is the factor most commonly associated with colic in the horse (Cohen et al., 1995; Cohen and Peloso, 1996; Tinker et al., 1997; Cohen et al., 1999; Hudson et al., 2001; Mehdi and Mohammad, 2006). Variations in the type of feed (forages, concentrates, pasture turnout), the quantity provided, and the frequency that feed is offered to the horse all may play a role in altering the pH of the ingesta and the bacterial populations that are relied on for effective digestion (Tinker et al., 1997; Cohen et al., 1999; Hudson et al., 2001). Therefore, the horse should be provided at least 60% of its diet from forage sources at a minimum of 1-1.5% of body weight (Geor, 2007). Concentrates should be kept to a minimum and provided in three or more feedings per day to reduce acid production in the stomach and colon (Hussain et al., 2004; Geor, 2007). If concentrated energy sources are required, vegetable oil, strained sugar beet pulp, or soy hulls are good alternatives to starch-based feeds. Finally, any change in the horse's diet should occur over 7-10 days to allow for adjustment of the microbial populations as well as small intestinal enzymes and glucose transporters required for carbohydrate digestion (White, 2005).

Diets for Specific Diseases

Uncomplicated Colic

In the majority of colic cases, the specific diagnosis is not determined. Vagotonia (gas colic) and mild impaction are the top suspects. Physical examination in these cases is typically unremarkable, and advanced diagnostics are often unnecessary. Treatment including oral fluids, mineral oil, and nonsteroidal anti-inflammatory medications is usually successful, with a full recovery in most cases in 12-24 hours.

In cases of simple colic, feeding should be resumed as soon as normal borborygmi and fecal production are noted. Good-quality forages should be provided for the first 12-24 hours of refeeding in small amounts (0.5 kg or 1.1 lb) every 4-6 hours. If there are no further signs of discomfort, feedings can gradually increase over 24-48 hours to normal rations. Forages are the preferred diet in any horse, but alternatives to hay include complete pellets, alfalfa pellets, hay cubes, or grazing starting at 15- to 20-minute intervals. Mashes are often acceptable ways to increase water intake and palatability of the feeds. Grains should be avoided for 10-14 days to avoid additional microbial disruption. The clinician should make an effort to reintroduce the horse to feed using the type of feed it will

be fed after it recovers. If the feed that the horse is reintroduced to is different than its regular diet, changes should be made gradually over 7-10 days if the owner wishes to return to the original diet.

Most cases of uncomplicated colic do not recur. However, the diet, environment, and feeding schedules should be closely scrutinized to identify any predisposing factor that may have led to the colic incident. If a problem is identified, such as a change in feed supplier or recent travel, preventative measures can be recommended.

Equine Gastric Ulcer Syndrome

Equine gastric ulcer syndrome (EGUS) is a term used to encompass a variety of disease processes that result in ulceration of both the glandular and squamous portions of the stomach and signs of colic. Gastric ulceration results from an imbalance between ulcer-promoting factors (hydrochloric acid, bile acids, pepsin) and protective factors (mucus, bicarbonate, mucosal blood flow, prostaglandin E2, epidermal growth factor, gastroduodenal motility) (Sanchez, 2004). Dietary factors that may contribute to EGUS include high-concentrate diets, low-roughage diets, poor-quality fiber, meal feeding, and fasting (Vatistas et al., 1999; Merritt, 2003; Lester, 2004; Jonsson and Egenvall, 2006; Luthersson et al., 2009). Dietary recommendations for prevention of EGUS are centered on increasing the frequency of feedings, reducing the quantities of starches fed, and increasing the quality of forages provided.

Saliva and ingested feeds are the primary buffers for the acid produced in the stomach of the horse. Because of this, pasture turnout is often recommended as the best method to prevent and control gastric ulcers (Buchanon and Andrews, 2003; Reese and Andrews, 2009; Videla and Andrews, 2009). However, this may not completely eliminate ulcers, since horses naturally tend to eat less at certain times of the day. These foraging patterns have been suggested to be the cause of a circadian pattern to the pH of the proximal stomach in horses regardless of their housing (Husted et al., 2008). This may be the reason that horses have been diagnosed with gastric ulcers even when confined solely at pasture (le Jeune et al., 2009). While the horse's voluntary intake is not under our control, free-choice forages should still be provided, since intermittent feeding or feed withholding is the experimental model used to consistently produce gastric ulcers (Murray and Schuseer, 1993; Murray, 1994).

Stall confinement and meal feeding are separate factors that have been associated with altered feed intake and gastric ulceration (Murray and Eichorn, 1996). In one study, feeding meals greater than six hours apart increased the likelihood of nonglandular ulceration, suggesting that continuous access to forages may be critical to reducing this risk (Luthersson et al., 2009). This was supported by the results of a second study that found ulcers in 75% of horses fed twice daily and in 57.9% of horses fed three times daily (Feige et al., 2002). Based on these findings, continuous feeding of alfalfa or good-quality grass hay has been recommended for horses not on pasture to protect the nonglandular mucosa. The high protein and calcium content of alfalfa may provide better protection that regular grass hays alone, speculatively due to a direct buffering effect of the high protein content or through a reduction in the secretion of gastrin (Nadeau et al., 2000). This effect on pH was consistent, even when alfalfa was fed with concentrates such as oats, barley, or pelleted rations (Buchanon and Andrews, 2003; Lybbert et al., 2007). Based on these findings, alfalfa may be the forage of choice for horses at risk for, or diagnosed with, gastric ulcers (Videla and Andrews, 2009).

Grains should be kept at a minimum in horses with EGUS due to their known association with the formation of gastric ulcers (Vatistas et al., 1999). Soluble carbohydrates present in most cereals fed to horses are fermented readily in the stomach to volatile fatty acids. These fatty acids are able to

penetrate the cells of the mucosa, resulting in cell swelling and disruption (Nadeau et al., 2003a,b; Andrews et al., 2006).

Concentrate diets are also known to increase the release of gastrin, the hormone responsible for acid secretion (Smyth et al., 1988). While the acid can be disruptive alone, the effects of volatile fatty acids are exacerbated by a pH less than 4 (Nadeau et al., 2003a,b; Andrews et al., 2006). In addition, grains have been shown to delay the secretion of gastrin, increasing the likelihood of inappropriate acid secretion after ingesta has passed. As an empty stomach is more likely to expose the nonglandular mucosa to acid, injury and ulceration may be more likely when feeding grains (Sandin et al., 1998).

If needed, grains should only be fed in amounts less than 0.5 kg/100 kg body weight no more frequently than every 6 hours to reduce the volatile fatty acids to a level below the threshold for mucosal damage (Andrews et al., 2006). The grains selected should be low in starch, since a starch intake between 1-2 g/kg body weight per meal was associated with a 2.6-times increase in the likelihood of ulcers (95% CI 1.3–5.2; P=0.006) and an intake greater than 2 g/kg body weight per meal increased the likelihood by 3.2 times (95% CI 1.3-7.7; P=0.009) (Luthersson et al., 2009). Hay, preferably alfalfa, should be available at all times. Fat supplements such as vegetable oils may be used as a substitute for concentrates to provide energy. While lipids were unable to prevent nonglandular ulceration, they may be useful in the therapy and prevention of glandular ulceration by reducing gastric acid and increasing prostaglandin production (Cargile et al., 2004; Frank et al., 2006).

While horses with colic are at no higher risk for gastric ulcers than the general population, the likelihood that they will be withheld from feed due to their disease process may increase the chance of ulcer formation (Rabuffo et al., 2009). Horses with diseases that may require long periods without oral feeding (e.g., anterior enteritis) are particularly at risk (Dukti et al., 2006). This strengthens the argument that horses with colic should be fed as soon as their medical conditions allow, to reduce the risk of iatrogenic complications of feed withholding.

Duodenitis-Proximal Jejunitis

Duodenitis-proximal jejunitis (DPJ), also known as anterior enteritis, is an inflammatory disease resulting in hypersecretion in the proximal small intestine, gastrointestinal ileus, and copious nasogastric reflux. A full description of this disease process, diagnosis, and therapy can be found elsewhere (McConnico, 2004). While infectious organisms including *Salmonella spp., Clostridium perfringens*, and *Clostridium difficile* have been implicated, the exact cause is still unknown (Merritt et al., 1982; Griffiths et al., 1997; Edwards, 2000; Arroyo et al., 2006). A suspected link with aflatoxicosis or fusariotoxicosis has also been made, based on necropsy findings of lesions consistent with DPJ. Therefore, it is important to be highly vigilant for mold in concentrates and other feeds containing corn (Schumacher et al., 1994, 1995).

High-energy feeds such as concentrates were previously thought to increase the risk of developing this disease, due to high concentrations of hydrolysable carbohydrates that fermented in the stomach and possibly the small intestine (Huskamp, 1985). This link between grains and DPJ has been statistically supported by a modest odds ratio of 1.3-1.62 (P<0.001) in a case-control study performed in Texas (Cohen et al., 2005). In this study, horses with DPJ were fed a median of 4.1 kg concentrate daily versus 2.7 kg in the control group.

Horse with DPJ were significantly more likely to have had access to pasture grazing in this same study, with an odds ratio of 3.5-4.0 (P<0.0005) (Cohen et al., 2005). This connection to pasture grazing has caused comparisons to be made between DPJ and equine dysautonomia, another cause of ileus in the horse associated with grazing pastures. Speculated causes of dysautomonia

are similar to DPJ, including mycotoxins and *Clostridium* spp., supporting an infectious cause for both syndromes (Poxton et al., 1997; Robb et al., 1997; Hunter et al., 1999; McCarthy et al., 2004).

Medical therapy for DPJ involves maintaining hydration with IV fluids, supporting oncotic pressure with plasma or colloids, providing anti-inflammatory medications, and prokinetics. Surgery should be considered if obstructive diseases of the small intestine cannot be ruled out or if medical management is unsuccessful (Johnston and Morris, 1987; Freeman, 2000). If surgery is elected, decompression of the small intestine into the cecum or an incomplete bypass may be performed (Huskamp, 1985, White et al., 1987; Gillis et al., 1994; Edwards, 2000). Both medical and surgical treatments have a good chance for full recovery if aggressive treatment is pursued and the disease is allowed to run its course (Edwards, 2000; Underwood et al., 2008).

Horses with DPJ are usually unable to take in enteral feed due to the severity of the ileus, resulting in significant volumes of gastric reflux. In addition, protein loss is often severe due to loss of the intestinal mucosal barrier and protein catabolism from prolonged cachexia. Based on the body condition at the time of diagnosis, parenteral nutrition should be considered for any horse identified as malnourished (low body condition score), horses with increased demands (pregnancy, lactation), and horses with DPJ that continues to reflux for more than 48 hours. Parenteral nutrition should continue until the horse is voluntarily eating at least 60% of its resting energy requirements.

Enteral feeding can begin once progressive motility is confirmed by a lack of net gastric reflux and ultrasonographic confirmation of small intestinal contractions. Water is initially provided in small amounts (1-2 liters every 2-4 hours) to confirm forward motility. If water is accepted without complication, hay, softened complete feeds, or alfalfa pellets can be introduced slowly, starting at 25% of the daily ration divided into meals every 2-4 hours. The horse should be reintroduced to full feed within 2-3 days. Vital parameters, appetite, attitude, and repeated ultrasonographic examination will assess the progression of feedings during reintroduction. Concentrates or fresh grasses with a high concentration of fermentable carbohydrates should be avoided. After recovery, grains should be avoided for the first two weeks, and if required by the owner, should be limited to meals less than 0.5 kg/100 kg body weight and no more than four feedings per day. Protein-rich forages, including alfalfa, or vegetable oils may be required to regain weight lost during the disease.

Small Intestinal Strangulation

Small intestinal strangulations are caused by obstruction of the blood supply. Strangulating lesions can result from a number of conditions including mesenteric lipomas, mesenteric rents, intussusceptions, and epiploic foramen entrapment, but all are directly unrelated to dietary factors. Clinical signs of strangulation include severe pain unresponsive to analgesia, small intestinal distention on rectal examination and ultrasound, peritoneal fluid changes, and variable amounts of reflux depending on the duration and location of the obstruction. Diagnosis of the cause of the strangulating lesion is confirmed during an exploratory laparotomy, when resection may be performed if the bowel is deemed nonviable.

The small intestine is extremely sensitive to the manipulation required by surgery as well as distention caused by obstruction, and these factors may result in postoperative ileus. If gastrointestinal stasis occurs after surgery, enteral feeding is not possible; therefore, parenteral feeding is often required to maintain body condition and provide supportive nutrition for recovery. The timing for initiating early parenteral nutrition is case-dependant due to preexisting conditions, concurrent diseases, body condition on presentation, and the financial considerations of the owner. Despite evidence of improved nutritional status of horses post-surgery that are provided parenteral nutrition,

including lower total bilirubin and serum triglycerides, it is still unclear as to any benefits of providing parenteral nutrition to surgical colic cases, with the downside of an increase in cost to the client (Lopes and White, 2002; Durham et al., 2004). Based on the lack of clear evidence, most horses are simply held off feed until ileus subsides.

In an effort to prevent post-operative ileus, horses with small intestinal resections or enterotomies should be provided oral feed as soon as possible after surgery. Common practice has been to begin oral supplementation only when borborygmi are present and defecation has occurred, but human medicine is now recommending providing enteral feed even earlier (McClave et al., 2009). Veterinary medicine is changing as well in that regard. Freeman and coworkers advocate reintroducing feed for any horse without signs of colic or reflux on a set schedule post-surgery, rather than relying on defecation or intestinal sounds to guide reintroductions (Freeman et al., 2000; Freeman and Schaeffer, 2010). On this plan, horses are first offered water in small amounts at 12-18 hours after surgery, followed by alfalfa hay in handfuls at 18-24 hours, which increases slowly every 2-4 hours over the next 24 hours. The goal would be to reintroduce the horse to at least 75% of full feed over the next 1-2 days. Softened complete feed, alfalfa pellets, or grass offered as small meals would be viable alternatives to hay for horses in which anastomosis is questionable or mucosal edema is suspected to be severe. Vital parameters, attitude, packed cell volume, serum total solids, and progressive motility noted by lack of reflux are used to determine if feeding is progressing satisfactorily. Small, controlled feedings are thought to increase motility and reduce cost and complications including gastric ulceration.

Short bowel syndrome is defined as a malabsorptive disorder caused by strangulations with resections that result in a loss of more than 60% of the small intestine (Tate et al., 1983). However, the length of small intestine that is necessary for adequate absorption may be variable, based on reports of individual cases where horses have survived with less (12.3-15 feet) (Vachon and Fischer, 1995; Freeman et al., 2000). The small intestine is the primary site for absorption of carbohydrates, protein, and minerals, and the ileum is specifically the site for fat-soluble vitamin absorption. However, the large intestine appears to be able to compensate post-operatively for the lack of absorption of proteins, carbohydrates, and B vitamins. In addition, the small intestine is capable of a compensatory adaptive hyperplasia that increases the efficiency of digestion by increasing the length, diameter, and surface area of the remaining intestine (Chaves et al., 1987; Quigley and Thompson, 1993; O'Connor et al., 1999).

Clinical findings of short bowel syndrome include weight loss, hepatic damage, a capricious appetite, and diarrhea (Tate et al., 1983; Freeman et al., 2000). If recognized after surgery, the goal would be to optimize large bowel fermentation with a highly digestible fiber source such as alfalfa, beet pulp, or soybean hulls to support volatile fatty acid production (Freeman and Schaeffer, 2010). If the ileum or greater than 75% of the small intestine has been resected, fat-soluble vitamins may need to be added to the diet (Tate et al., 1983). Supplementation with rice bran or corn oil may be required to provide additional energy to maintain body weight but should be avoided in cases with ileal resection (Tate et al., 1983; Geor, 2001). Complete pelleted feeds provided in small frequent meals have also been successful in supporting horses with resections of up to 70% of the distal small intestine (Lewis, 1995). However, grains should be avoided if an extensive resection has been performed, due to the inability to adequately process starches (Dyer et al., 2002).

Ileal Impaction (Small Intestinal Obstruction)

Ileal impactions are most commonly noted in mature horses in the southeastern United States.

This obstruction is linked to the excessive nonfermentable fiber and fine stem character of poor-quality coastal Bermuda hay, which may be related to factors such as maturity at harvesting, soil composition, curing, and storage (Little and Blikslager, 2002; Lee et al., 2010). An alternative risk factor commonly cited for ileal impactions is infestation with *Anoplocephala perfoliata*, which localizes at the ileocecal junction and may result in obstruction of the valve due to excessive inflammation (Proudman and Edwards, 1998; Little and Blikslager, 2002).

Clinical signs of ileal impaction include moderate to severe signs of colic, depending on the duration and severity of the obstruction (Parks et al., 1989; Hanson et al., 1998; Little and Blikslager, 2002). Early in the course of the disease, the impaction may be felt on transrectal palpation as a firm tubular structure entering the cecum on the right side of the abdomen (Hanson et al., 1996). As the obstruction progresses and becomes complete, the small intestine proximal to the ileum fills with ingesta, and multiple loops may be palpable per rectum and noted on ultrasound examination. Ileus results, and gastric reflux may be obtained (Parks et al., 1989; Hanson et al., 1998).

Medical therapy is currently recommended, using intravenous fluids, gastric decompression, sedation, and analgesics (Hanson et al., 1996). Serial abdominocenteses are used to monitor the progress of medical therapy, and if protein and white blood cell counts increase with no response to medical therapy, surgery is recommended. At surgery, the impaction is reduced into the cecum using external massage, often with the assistance of direct infusion with balanced electrolyte solutions or carboxymethylcellulose (Parks et al., 1989; Hanson et al., 1998). In some cases, an enterotomy may be useful to reduce the trauma caused by this type of external manipulation. Post-operative ileus and adhesions are a risk for any surgical procedure used to relieve an ileal impaction.

In most horses, ileal impactions will resolve with medical therapy in 12 hours, so supportive care in the form of parenteral nutrition is rarely required (Hanson et al., 1996). In the acute phase of recovery, horses should be fed on a schedule similar to small intestinal strangulation whether they have been managed medically or surgically, in that feeding can resume gradually as soon as the impaction appears to have resolved. Clinically, resolution would be noted as a lack of significant reflux, good motility on ultrasound exam, and no palpable impaction or small intestinal distention. However, the ileum can become edematous or spasmodic due to inflammation from the impaction or surgical manipulation, making recurrence likely if feeding is too aggressive. Continued use of nonsteroidal medications during refeeding may reduce this risk, and some clinicians have recommended withholding hay until 48 hours after resolution of clinical signs (Hanson et al., 1996). Personal observations have noted that large intestinal impactions may often be a concurrent issue, and this problem should be addressed prior to refeeding.

Due to the fact that ileal impactions have been definitely linked to coastal Bermuda hay, it is recommended after recovery to avoid feeding this forage to horses that have developed this type of obstruction (Little and Blikslager, 2002). If alternative hay sources are not available, complete pelleted diets or pastures are viable alternatives to forage. If coastal Bermuda hay must be fed, it should be introduced gradually and not fed as free-choice round bales (Blikslager, 2005). High-quality, first-cutting hays are recommended, and water intake should be insured (Pugh and Thompson, 1992). In addition, feeding a pelleted ration or concentrates can increase gastrointestinal transit time and possibly reduce the risk of recurrence of impaction (Hintz and Loy, 1966).

Ascending (Large) Colon Impactions

Ascending colon impactions are the most frequent cause of colic in the horse and have been proposed to be caused by a number of external factors, including water intake, stress, feed quality, or

dietary changes that result in dehydration of the colon ingesta (White, 1990). Obstruction typically happens at points in the large colon where narrowing occurs, including the pelvic flexure and left ventral colon, as well as the sternal and diaphragmatic flexures (Dabareiner and White, 1995; White and Dabareiner, 1997). Clinical signs typically include mild to moderate colic, with a reduction in fecal output. Transrectal palpation may note firm, mucus-covered fecal balls, and if the impaction is palpable in the large colon, diagnosis can be confirmed. Therapy centers on administration of fluids, laxatives, and judicious use of analgesics and antispasmodics (Dabareiner and White 1995; Lopes et al., 1999; Hallowell, 2008). Feed should be withheld from the horse during treatment. Surgery is indicated if the impaction does not resolve in a timely manner, the abdomen becomes more distended, or pain becomes intractable. At surgery, the impaction is relieved by lavage through a pelvic flexure enterotomy.

For horses treated with medical therapy alone, feeding should not be delayed once the impaction has cleared. Signs of resolution include a lack of abnormalities on transrectal palpation, adequate fecal production, and normal gastrointestinal transit time as noted by the passage of mineral oil. Good-quality forages, ideally alfalfa hay, should initially be provided in small amounts (0.5 kg or 1.1 lbs) four to six times daily, with a gradual increase over 24-48 hours to normal rations (Geor, 2007). Free-choice hay may be preferable after successful reintroduction to feed, to reduce the risk of gastric ulcers. Alternatives to hay during the acute phase, depending on the patient and clinician preference, include complete pellets, alfalfa pellets, or grazing, starting at 15-minute intervals. The goal would be to have the horse on a full ration within 1-3 days after reintroduction of feed (1.5-2.5% of body weight in forages). Grains should be avoided for 10-14 days after colic has resolved to avoid further disruptions in the microbial populations by undigested starches. If the owner wishes to resume concentrate feedings, they should be reintroduced slowly, starting with less than 1 kg (2.2 pounds) per 500-kg horse twice a day, and increasing gradually if needed by 0.5-0.75 kg increments per day (Geor, 2007).

If the horse has undergone a celiotomy to relieve the impaction, the feeding schedule should be identical to that for a resolved medically treated impaction. Most horses can return to feed as soon as all anesthetic drugs have worn off, usually 3-6 hours after returning to the stall. Some clinicians prefer to withhold feed for 12-24 hours after performing an enterotomy. However, a secure closure and good surgical technique will not delay reintroduction of feed, and the lumen is large enough at the pelvic flexure that any distention by feed should not stress the suture line. In addition, an enterotomy with lavage has a disruptive effect on the large colon flora, which should be supported as soon as possible with a healthy substrate for fermentation (Hassel et al., 2009b). Grass hay has been associated with a reduced risk of post-operative diarrhea in horses after enterotomies. Therefore, grazing and grass hay are both recommended in the early post-operative period (Cohen and Honnas, 1996). It is important to remember hospital biosecurity when grazing horses after colic, due to the unavoidable alterations in gastrointestinal flora and the risk of salmonellosis. This is especially important post-celiotomy for large intestinal disease, which increases the risk of diarrhea twofold over other types of intestinal lesions ($P<0.006$) (Cohen and Honnas, 1996). An enterotomy was also found to increase the risk an additional 1.5 times ($P=0.042$).

For horses that are prone to impactions, prevention is centered on providing good-quality forage and encouraging adequate water intake. Late-season cuttings of hay, which are poor in fiber quality, should be avoided, as should forages known to be of poorer quality, including coastal Bermuda and straw. Green grass or alfalfa-based diets may provide a laxative effect, and complete pelleted feeds will improve overall digestibility of the diet for horses with dental disease. Alfalfa pellets, hay cubes,

or beet pulp may provide a highly digestible fiber source, with shorter fiber length to improve digestibility. While the value of regular dental prophylaxis is still under discussion, proper occlusion will aid in reducing fiber length and possibly reduce the risk of impactions. Salt (sodium or potassium chloride) may be added to the feed to encourage water intake, up to 8 tablespoons a day. Alternatively, electrolyte water, molasses-flavored water, or other flavored water may be added to provide interesting alternatives, alongside a plain water bucket.

Sand Impactions

Sand impactions are a specific type of intestinal obstruction resulting from the ingestion of sand typically obtained from the environment. Feeding on the ground or in sandy arenas has been specifically implicated as a cause. However, some horses, especially foals, may intentionally ingest sand, which is a vice known as pica. In addition, the naturally sandy soils in some regions (e.g., Florida, Georgia, and Arizona) provide ample opportunity during grazing for accidental ingestion, especially on short-cropped pastures (Colahan, 1987).

Clinical signs are often similar to feed impactions of the ascending colon (Specht and Colahan, 1988), but the irritating nature of the sand may result in debridement of the mucosal lining, diarrhea, and signs of endotoxemia due to bacterial translocation (Ragle et al., 1989a; Granot et al., 2008). Sand impactions can occur at multiple points throughout the digestive tract including the stomach, cecum, small intestine, ascending, and small colon. However, impactions of the dorsal ascending colon are most often noted in horses at surgery (Specht and Colahan, 1988; Ragel et al., 1989a; Granot et al., 2008). Peritonitis or rupture of the large intestine or stomach may occur due to large volumes of sand or long-standing impactions that damage the integrity of the intestinal wall.

Diagnosis of the presence of sand is often made based on a history of exposure and by identification of sand in the ingesta. Sand may be palpated per rectum as a “gritty” feeling in the rectum, or in the impaction if it is palpable. Fecal flotation may help to determine exposure to sand. It is performed by dissolving 3-4 fecal balls in water in a palpation sleeve, and identifying sand sediment in the fingers of the glove. However, fecal flotation poorly correlates to the presence of sand in the intestines, due to a high proportion of false negatives, and does not allow for the determination of the amount of sand present in the colon (Edens and Cargile, 1997).

The volume of sand present is better demonstrated by abdominal radiographs as a mineral opacity, but its sensitivity is low in larger horses, especially for impactions not present in the ascending colon. The accumulations can be graded 0 through 4, and a significant amount of sand is greater than 5 by 15 cm in length and pulls the large colon against the ventral body wall (Kendall et al., 2008). Ultrasound may also be helpful in identifying the presence of sand, but it cannot replace radiography for assessment of volume (Korolainen and Ruohoniemi, 2002). While auscultation may identify characteristic “waves on a beach” sounds caudal to the xyphoid in some horses with sand, it is not sensitive for sand obstructions, especially if colic is reducing large intestinal motility (Ragel et al., 1989b).

Treatment of colic due to sand impaction has centered on the use of fluid therapy, osmotic laxatives, and psyllium fiber at a dose of 0.5-1.0 g/kg (Ethell et al., 1993). Psyllium husks contain mucilage, which absorbs water and is believed to act as a laxative. Psyllium gels when mixed with water, so it must be administered quickly through a nasogastric tube. Alternatively, the psyllium can be mixed with 2 liters of mineral oil, which suspends the psyllium and allows for easier administration by nasogastric tube. The horse is administered 2-4 liters of water immediately after dosing with

the psyllium-oil mixture, to remove the psyllium from the oil suspension, and reform the gel in the intestinal tract (Blikslager and Jones, 2004).

According to the literature, treatment with psyllium has had a variable response, even when combined with additional laxatives or mineral oil (Ruohoniemi et al., 2001). One study noted no difference between treatment with psyllium (0.5 g/kg), wheat bran (1 g/kg), or a 1:1 mineral oil/water mixture (8 g/kg) compared with no treatment at all when sand was administered per nasogastric tube at a dose of 0.5 g/horse (Lieb, 1997). Doses of psyllium up to 1.0 g/kg were also ineffective in horses implanted with 10 g/kg of sand into the cecum (Hammock et al., 1998). Another study noted a significant improvement, with twice the amount of sand cleared using 0.5 kg psyllium with 2 liters of mineral oil versus mineral oil alone (Hotwagner and Iben, 2008). However, the total amount of sand cleared after 5 days of treatment was only 51%. In horses unresponsive to psyllium, additional treatment with magnesium sulfate (1 g/kg for 3 days) was effective in resolving radiographic signs of impaction (Ruohoniemi et al., 2001).

Horses that do not respond to medical therapy and analgesia are surgical candidates, and concurrent large colon displacements or volvulus are not uncommon findings at surgery (Specht and Colahan, 1988). Through a ventral midline celiotomy, the colon is exteriorized carefully, a pelvic flexure enterotomy performed, and the sand siphoned from the site of impaction, commonly the right dorsal colon (Ragel et al., 1989a; Granot et al., 2008). The gastrointestinal tract must be carefully examined for pressure necrosis, vascular compromise, and transmural damage due to sand. Short-term survival is good for both medical and surgical treatment of sand impactions, and long-term survival is excellent (Granot et al., 2008).

If the horse is treated for sand impaction with surgery and an enterotomy, the feeding schedule should be similar to that provided for surgical resolution of feed impactions. However, it is important to recognize that horses with diseases of the ascending colon treated by enterotomy and lavage are at a higher risk for developing diarrhea, and horses with sand colic are especially predisposed due to the irritating nature of the condition (Cohen and Honnas, 1996). Based on positive results in clinical cases, administration of di-tri-octahedral smectite (0.5 kg/500 kg BW in 4 L water, Q24h) for three days post-operatively may allow for a reduction in diarrhea (Hassel et al., 2009b). Di-tri-octahedral smectite may reduce damage to the colonic mucosa by neutralizing clostridial and *Bacteroides* toxins (Weese et al., 2003; Lawler et al., 2008; Traub-Dargatz, 2008). It may also have a prolonged protective effect through regulation of gastrointestinal flora and intestinal secretions (Albengres et al., 1985; Yao-Zong et al., 2004). This would be an important benefit for horses recovering from surgery, due to the disruption of intestinal flora and the mucous layer by an enterotomy.

It is unknown what volume of sand is significant in terms of causing clinical disease, and the specific volume may be different for each individual horse. It is reasonable to assume that any volume of sand may alter the integrity and function of the gastrointestinal tract due to its abrasive nature, and long-term exposure may be even more damaging (Bertone et al., 1988). While treatment can be provided for impactions, prevention is a better option. Often, removing the horse from the environment is not possible, but reducing the exposure to sand can be attempted. Horses should not be fed on the ground, and large tubs or rubber mats should be used to prevent feed from spilling into contaminated soil. Pastures should not be overgrazed, and should be rested and reseeded to encourage better growth. Psyllium, either as a generic powder or commercial pellets, may be used as a preventative if given at a dose of 0.25 kg once daily for one week every month. Horses with continuous exposure could be treated daily with psyllium, but there is some concern that gastrointestinal flora may acclimate to this new fiber source and digest the psyllium, reducing the effectiveness (Hammock et

al., 1998). The most effective method of clearing sand from the gastrointestinal system is to provide 2.5% of body weight per day of hay. The bulk alone is capable of removing almost 95% of ingested sand and was better in a controlled trial than psyllium, mineral oil, or wheat bran (Lieb, 1997).

Enteroliths and Fecaliths

Enteroliths are intestinal concretions formed by the deposition of minerals, primarily magnesium, nitrogen, and ammonium phosphate salts, around a nidus of foreign material (Blue and Wittkopp, 1981; Murray et al., 1992; Hassel et al., 2001). Concretions may also form around fibrous undigested feed material, which are classified as fecaliths. These mineralized masses are believed to develop in the ampulla of the right dorsal colon and may cause partial or complete obstruction as they are propelled distally into the descending colon. Signs of abdominal discomfort may abate if the enterolith is able to dislodge proximally back into the right dorsal colon and these horses often have a history of recurrent and intermittent colic.

Any foreign material can serve as the center for calculus formation, including sand, pebbles, rope, cloth, or hair. Current research into enterolithiasis points to a multifactorial cause. However, the end result of all factors investigated is an intestinal environment that promotes the deposition of mineral, due to a basic pH and the presence of minerals including magnesium, nitrogen, phosphorus, calcium, sulfur, sodium, and potassium (Hintz et al., 1988, 1989; Hassel et al., 2004, 2009a).

Genetics have been identified as a cause of enteroliths in that members of certain breeds are predisposed, including Arabians, Morgans, American Saddlebreds, American Miniatures, and donkeys (Hassel et al., 1999, 2004; Hassel 2002; Cohen et al., 2000). There is also an increased predilection for the disease among siblings (Hassel et al., 1999). This may be due to a genetic predisposition for a more alkaline large intestinal content, which has been found in horses with enterolithiasis versus horses without the disease (Hassel et al., 2004, 2009a).

Diet also plays a role in that specific feeds have been found to provide an intestinal environment that favors enterolith formation. Alfalfa is rich in minerals, as well as protein, which can provide a source of ammonium nitrates (Lloyd et al., 1987). Feeding greater than 50% alfalfa hay results in higher calcium, magnesium, phosphorus, and sulfur, and a more alkaline pH in the colon contents (Hassel et al., 2009a), and has been statistically linked to enterolith formation (OR=4.74; 95% CI 1.44-15.63) (Hassel et al., 2008). While it has never been definitively linked to the disease, wheat bran is also higher in protein, magnesium, and phosphate than most grains, and is suspected to contribute to the problem (Cohen et al., 2000; Hassel et al., 2004, 2008). The environment can also be a source for minerals, from both the soil and the water provided. It is clear that horses in California are predisposed; however, enteroliths have been diagnosed in a number of other states and countries (Hassel et al., 1999).

An additional contributing factor is the management of the stable. Stall confinement for more than half the day, low-fiber diets, and feeding intermittent meals have been linked to enterolith formation (Cohen et al., 2000; Hassel et al., 2004, 2008). Inactivity may affect the motility of the ampulla of the right dorsal colon, encouraging mineral deposition in horses that are stalled (Hassel, 2002; Hassel et al., 2004). Grazing may provide both exercise and some dilution of the minerals ingested, along with production of short-chain fatty acids for fiber fermentation, potentially reducing colon pH and the chance of crystallization (Stevens, 1978; Hassel et al., 2004).

Diagnosis of enteroliths can be made at surgery or necropsy or by radiographs of the abdomen of suspected horses. Palpation may identify the concretion in the small colon, but often it is too far cranial to allow for access per rectum. In some cases, the owners may have reported seeing small

enteroliths passed in the feces, confirming the diagnosis. Horses typically present with a history of mild, recurrent colic with partial obstruction, but if complete, clinical signs will be similar to other forms of large intestinal obstruction. Advanced imaging may allow for diagnosis and early detection before complete obstruction occurs, but radiographs are less sensitive for enteroliths in the small colon and in horses with significant gas distention (Yarborough et al., 1994). Definitive treatment is surgical to allow for removal through a large colon or descending colon enterotomy. However, prognosis may be improved if the surgery is elective, before obstruction and pressure necrosis occurs.

Because genetics cannot be controlled, the practitioner should focus on prevention through dietary changes in high-risk horses, including those with a previous history of enteroliths, horses boarding at facilities with a high incidence of the disease, and those related to horses diagnosed with enterolithiasis. Horses of specific breeds, including Arabians, American Miniatures, and ponies, should also be included in the high-risk category if they have additional risk factors that would predisposed them to the problem (environment, diet) or have a history of chronic or recurrent colic.

Prevention should be centered on avoiding feeds that increase the risk of enterolith formation, including those that are high in magnesium and phosphorus. Alfalfa hay should be limited to less than 50% of the diet, and hay grown in California should be avoided (Hassel et al., 2008). Grass or oat hays appear to be protective, possibly due to lower protein and magnesium levels, and higher fiber content for the production of short-chain fatty acids (Hassel et al., 2008). High-protein diets also should be avoided due to the ammonium content, unless required. Minerals may be removed from the water source using a water filter, but this is an expensive proposition, and in most cases the mineral content of the feed far outweighs any contribution from the water sources (Lloyd et al., 1987).

Protective treatments include those that are able to acidify the colon contents. This is one instance where feeds high in soluble carbohydrates may be indicated to acidify the colon contents. Calculi are formed in basic environments, and if the colon is acidified by starch-digesting bacteria, there is a chance that stone formation could slow or stop. Similarly, there have been reports of in vivo dissolution of enteroliths using vinegar to reduce the colon pH (Hintz et al., 1988; Murray et al., 1992). While unproven as a preventative or therapy, there have been additional reports that administration may reduce the risk of recurrence (Hassel et al., 1999). Approximately 1-2 cups (8-16 fluid ounces) per day of apple cider vinegar is recommended (Hintz et al., 1989; Hintz, 2000).

Environmental factors are more difficult to control. While eliminating the source of a nidus for calculi formation is impossible, reducing the risk for ingestion of foreign items is still recommended (Rose and Rose, 1987). Regular treatment with psyllium (0.25-0.5 kg, once daily for one week a month) should be provided in areas endemic with sandy environments. Horses should not be fed on the ground, and feed should be placed in tubs or on rubber mats to reduce the ingestion of foreign bodies. Finally, based on evidence of a protective effect, pasture turnout should be provided daily.

Ascending Colon Displacement

The ascending colon is freely movable within the abdomen, since the right dorsal and transverse colons are the only points of attachment to the body wall for this organ. Displacement of the ascending colon may occur secondary to other disorders such as fecal impactions or as a primary problem due to management practices, including feeding large amount of concentrates as infrequent meals (Morris et al., 1986). Large concentrate boluses allow fermentable carbohydrates to reach the ascending colon, increasing the rate of fermentation and volatile fatty acid production (Argenzio, 1975; Clarke et al., 1990a). In addition, normal motility patterns may be disrupted by these large meals, increasing gas and fluid accumulation from altered fermentation (Ruckebusch,

1981; Clarke et al., 1990b). However, an underlying motility disorder may be the ultimate cause in some types of displacements (Smith and Mair, 2010).

Displacements are commonly grouped into right dorsal and left dorsal (or nephrosplenic) displacements, as well as pelvic flexure retroversion, or nonstrangulating volvulus at the ceco-colic junction (Hackett, 2002; Rakestraw and Hardy, 2006; Hardy, 2008). The exact position of the colon in the abdomen may vary; therefore, diagnosis may be simply large colon displacement. A left dorsal displacement is entrapment of the ascending colon over the nephrosplenic ligament. It is diagnosed on transrectal palpation by tracing a band of the left ventral colon into the dorsal left quadrant between the kidney and base of the spleen. Diagnosis is supported by the inability to locate the left kidney on ultrasound examination through the left flank (Santschi et al., 1993). Right dorsal displacement is movement of the colon into any number of configurations in the abdomen and is commonly diagnosed by a tight band running between the cecum and right body wall. In some cases, the pelvic flexure is not palpable, and the caudal abdomen feels empty due to retroflexion of the colon towards the diaphragm.

Clinical signs of displacement include mild to moderate abdominal pain with concurrent abdominal distention. Fecal production is also reduced. Obstruction of the lumen may result from the displacement of the colon itself or secondary to an impaction that may be a concurrent problem. The distention that occurs can obstruct venous and lymphatic outflow resulting in congestion and edema of the tissues; however, tissue damage is minor and resolves quickly once the displacement is corrected.

Medical management can be attempted using fluid therapy, analgesics, and hand-walking to stimulate motility (Huskamp, 1987). In cases with left dorsal displacement, phenylephrine and jogging may be attempted to displace the colon ventral if the large colon is not severely distended with gas or an impaction (Baird et al., 1991; van Harreveld et al., 1999). Trocarization may assist in medical therapy by reducing the amount of gas distention in the abdomen. Alternatively, the horse can be rolled under anesthesia to attempt to displace the colon ventrally (Boening and von Saldern, 1986; Kalsbeek, 1989).

Horses with significant distention per rectum, unrelenting pain, or an abnormal abdominocentesis should be taken to surgery immediately. A ventral midline celiotomy will allow for correction of most displacements. Alternatively, a standing flank approach may be performed to relieve left dorsal displacements; however, a full exploratory is not possible through this approach. A pelvic flexure enterotomy may be elected for relief of significant impactions.

Horses recovering from displacements are returned to feed in a similar manner to horses with ascending colon impactions once the displacement has resolved. If medical treatment was pursued, confirmation that the colon has returned to its normal position can be made by transrectal palpation, presence of adequate fecal production, and a lack of signs of colic for at least 12 hours. Good-quality forage is recommended when returning to feed, and the long fibers provided by most hays are preferred to increase the bulk of ingesta within the colon. It is believed that maintaining adequate fill of the ascending colon with ingesta will reduce the risk that gas distention will promote the migration to an abnormal position in the abdomen (Rakestraw and Hardy, 2006). If surgery and an enterotomy were performed to remove impacted feed, it is recommended to leave some ingesta in the lumen, again to provide some weight to the colon. The remaining feed should also promote normal fermentation to reduce the risk of diarrhea (Cohen and Honnas, 1996). Concentrates should be avoided for at least 10-14 days, or eliminated from the diet, to reduce the risk of soluble carbohydrates reaching the large intestine and resulting in abnormal gas production.

Prevention of large colon displacements is centered on providing adequate quantities of good-quality fiber and ensuring adequate water intake. Concentrates should be eliminated from the diet or fed in only in small quantities (less than 0.5 kg/100 kg/body weight no less than every 6 hours). Due to the fact that ascending colon impactions are often a concurrent problem, strategies recommended for prevention of impactions can be used for displacements as well. While the recurrence rate for nephrosplenic entrapment is low (between 7.5% and 8.1%), surgical ablation of the nephrosplenic space can reduce this risk (Baird et al., 1991; Hardy et al., 2000; Farstvedt and Hendrickson, 2005). Recent literature has noted an increased risk of recurrent colic after diagnosis and surgical treatment of right dorsal displacements, with a recurrence rate of 41.9% versus only 8.3% for left dorsal displacement in that study (Smith and Mair, 2010). Retroflexion of the pelvic flexure and nonstrangulating volvulus also had higher rates of recurrent colic. However, the recurrence rate for a second right dorsal displacement was still similar to nephrosplenic displacement, about 7%. It is speculated that the cause of right dorsal displacement may be an underlying motility disorder versus increased gas production proposed to cause left dorsal displacement (Hardy, 2008). This may be the cause of the increased risk of recurrence, which could undermine other preventative strategies. Based on these statistics, colpopexy may be recommended in cases with repeated displacements (Hance and Embertson, 1992).

Ascending Colon Volvulus (Large Colon Torsion)

Ascending colon volvulus is produced by the twisting of the colon on its mesenteric axis, typically in a counterclockwise direction at the level of the attachment of the right and left dorsal colons to the abdominal wall (Snyder et al., 1989). The cause of volvulus is still debated, but loss of abdominal fill after parturition or recent diet changes have been blamed (Rakestraw and Hardy, 2006). If the colon twists greater than 270 degrees on its axis, both the vasculature and lumen can be occluded, resulting in strangulation of blood flow and significant abdominal hypertension due to fluid and gas distention within the lumen. After occlusion of the vasculature occurs, the colon has been shown to suffer irreversible damage within 3-4 hours, and expedient therapy is essential to improve survival (Snyder et al., 1988). Examination of the horse is typically abbreviated due to the severity of the disease process. Clinical findings include pain refractory to analgesics and a distended large intestine on rectal exam.

Treatment of volvulus is detorsion of the large colon through a ventral midline celiotomy. After derotating the colon, viability is assessed by evaluating the colon for a return of a pink color to the serosal surface, active mucosal bleeding at a pelvic flexure enterotomy, the degree of mucosal edema and hemorrhage, arterial pulses in the colonic arteries noted by palpation or Doppler, muscular motility, and fluorescein uptake. The gold standard for assessment of viability is histopathology, but results are often only available after the horse has recovered from surgery or euthanized (Van Hoogmoed et al., 2000). If the colon is deemed nonviable, it can be resected up to the level of the cecocolic fold, but damage often extends farther than can be adequately removed (Hughes and Slone, 1997). In these cases, questionable bowel may remain in the abdomen, even with a resection, increasing the likelihood of dehiscence and fatal peritonitis.

Post-operative feeding of horses with volvulus is dependent on the degree of damage sustained by the colon. In horses treated quickly, with minimal mucosal edema and mural hemorrhage, feeding can begin in the early post-operative period (between 6-12 hours after surgery). Usually a pelvic flexure enterotomy is performed, and some surgeons prefer to wait at least 12-24 hours before testing the suture line with feed. If damage to the colon is minimal at surgery, the horse can be started

on good-quality forage at a rate of 0.5 kg every 4-6 hours. Alternatives to hay include complete rations or alfalfa pellets at the same rate, or grazing for 15-20 minutes at a time. The diet should be slowly increased over the next 1-3 days until the full ration is reached (2-2.5% of body weight in hay). Grains should be avoided due to the disruption of normal flora from both the ischemic event and lavage through the pelvic flexure enterotomy (Hassel et al., 2009b).

In horses with large colon resection, or if the colon remains in situ but has sustained extensive damage due to ischemia, it is recommended to postpone oral nutrition until 12-48 hours after surgery to reduce the risk of obstruction or failure of the anastomosis. This is especially true for horses with a questionable suture line due to the inability to completely resect the involved colon. In these horses with compromised intestine, endotoxemia, anorexia, and disruption of both fermentation and absorption may persist, and parenteral nutrition may be recommended. Once the horse is reintroduced to feed based on vital parameters and appetite, a highly digestible diet is required due to the loss of surface area of the ascending colon.

The colon is the site for protein and cellulose digestion and phosphorus absorption, so adjustments should be made in the post-operative period to ensure the diet contains at least 12% protein, 0.4% phosphorus, and a quality fiber (<28%)(Ralston et al., 1986; Bertone et al., 1989a). Pelleted diets, including alfalfa pellets or complete rations, would seem ideal due to the reduction in transit time after resections but were found to have reduced cellulose digestibility (Bertone et al., 1989a, 1989b). If they are included in the diet, pelleted feeds often must be fed at a rate above maintenance to prevent weight loss (Bertone et al., 1989b). While grass hay is recommended to reduce the risk of post-operative diarrhea, it does not have the protein content to maintain body weight in horses with large colon resections or severe colon damage. Alfalfa hay is preferred, and horses should receive at least 2% of body weight to maintain condition (Bertone, 1989). Despite reduced absorption, additional phosphorus supplements are not needed to maintain blood levels (Bertone et al., 1989b). However, adequate water intake should be ensured after resection, up to 8 liters more than normal horses, due to increased loss of water in the feces (Bertone et al., 1989b). Over time, the digestive tract appears to adapt by hypertrophy and special diets are not required (Ralston et al., 1986; Driscoll et al., 2008; Ellis et al., 2008). In some horses, supplementing with oil or soybean meal may improve and maintain weight gain in the early post-operative period after a volvulus (Driscoll et al., 2008).

Prevention of large colon volvulus is centered on surgical techniques. Recurrence rates vary in the literature between 5% and 50%, so large colon resections or colpopexy of the left ventral colon may be chosen to reduce this risk (Harrison, 1988; Markel, 1989; Hughes and Slone, 1997). Dietary changes should be gradual, and a forage-based diet is recommended to reduce the amount of carbohydrates entering the colon. However, occupational risk as a broodmare is a factor that often will not be eliminated.

Cecal Impactions

Impaction is the most common disorder resulting in colic related to the cecum (Dart et al., 1997; Edwards, 2002). If the impaction is the primary problem, the cause is often multifactorial, associated with poor-quality roughage, poor dentition, and reduced water intake (Campbell et al., 1984; Collatos and Romano, 1993). Alternatively, cecal impactions can be secondary to treatment for a separate disorder, including orthopedic diseases or ophthalmic disorders. Secondary impactions have been related to painful conditions, nonsteroidal anti-inflammatories (or lack thereof), anesthetic drugs, atropine, and stall rest (Edwards, 1992, 2002; Lester et al., 1992; Collatos and

Romano, 1993; Dart et al., 1997; Dabereiner and White, 1997; Little et al., 2001).

Cecal impactions are further classified by the physical character of the impaction (Dabereiner and White, 1997). Type one impactions are noted to consist of firm, dry ingesta and are often comparable to ascending colon impactions in that they are often attributed to a mechanical obstruction. Type two impactions are characterized as an idiopathic stasis with ingesta having a normal to fluid-like consistency. These horses often have an edematous or thickened cecal wall with peritonitis and evidence of endotoxemia. However, there is often significant overlap between classifications (Rakestraw and Hardy, 2006).

Clinical signs are subtle and often only noted as depression, anorexia, and reduced fecal output. Cecal impactions can rupture without any outward signs of discomfort, and therefore fecal production of hospitalized horses should be monitored carefully (Campbell et al., 1984; Dart et al., 1997; Plummer et al., 2007). Diagnosis is by transrectal palpation, which will note either a feed- or fluid-filled cecum in the caudal right abdomen with taut cecal bands. Cecal impactions can be treated medically with IV and/or oral fluids, and analgesics and feed should be withheld (Campbell et al., 1984; Collatos and Romano, 1993; Dart et al., 1997; White and Dabareiner, 1997; Huskamp and Scheidemann, 2000). However, surgical management may be indicated if the cecum is grossly distended, medical management is not progressing, or a type two stasis is suspected (Plummer et al., 2007). Some surgeons recommend early surgical intervention to improve survival (Campbell et al., 1984). At surgery, typhlotomy and lavage may be performed to relieve the impaction (Campbell et al., 1984; Dabereiner and White, 1997; Roberts and Slone, 2000, Plummer et al., 2007; Smith and Mair, 2010). If impaction recurs, or if a type 2 impaction is diagnosed, a complete or incomplete ileocolostomy or jejunocolostomy may be advised with or without occlusion of the distal small intestine (Craig et al., 1987; Gerard et al., 1996; Plummer et al., 2007; Lores and Ortenburger, 2008; Quinteros et al., 2010; Smith et al., 2010).

Feeding after cecal impactions is complicated by a high rate of recurrence (Dart et al., 1997). Therefore, reintroduction to feed should be a slow and gradual process, especially when the inciting cause cannot be resolved (i.e., stall rest, pain) or when cecal dysfunction is suspected. Only water should be offered in small amounts for the first 12-24 hours after the impaction is determined to be resolved. Resolution is based on a normal rectal examination and adequate fecal output. Horses that tolerate oral fluids can be gradually reintroduced to feed. However, those that have had a bypass may require a slower reintroduction to feed, starting at 36-48 hours post-surgery, due to an association between refeeding and colic (Gerard et al., 1996). Hand-walking and limited hand-grazing (5-15 minutes) may be allowed early on to stimulate motility, as complete fasting can reduce cecal motility further (Ross et al., 1990). Once feed is introduced, low-residue feeds including complete pelleted diets and alfalfa pellets are often preferred to reduce bulk within the large intestine and cecum. Reintroduction to hay should be gradual, starting with handfuls, and alfalfa is preferred due to its laxative effects. Coastal hay should be avoided because of its association with intestinal impactions (Plummer et al., 2007).

Similar to ascending colon impactions, prevention is centered on providing good-quality forage and encouraging adequate water intake. Quality fiber should be provided, as first cutting hays, fresh grass, or beet pulp. Laxative diets, including alfalfa-based diets or green grass, may be preferred, especially if the horse cannot be removed from the influence of one of the predisposing causes of cecal impactions. If possible, exercise should be encouraged and pasture turnout provided to increase cecal motility (Sullins, 1990; Cohen et al., 1999). Finally, adequate anthelmintics, including praziquantal, should be administered due to the suspected association between tapeworms and

cecal impaction (Beroza et al., 1983; Campbell et al., 1984; Proudman et al., 1993; Roberts and Slone, 2000).

Cecocecal and Cecocolic Intussusception

Cecocecal intussusceptions result from the invagination of the cecal apex into the cecal body. The apex can continue on into the right ventral colon to become a cecocolic intussusception. Cecal intussusceptions may result from changes in diet, bacterial infections, parasites, and medications that alter motility or result in inflammation (Gaughan and Hackett, 1990; Edwards, 1992; Dart et al., 1997; Gaughan and von Harreveld, 2000; Boussauw et al., 2001). Clinical signs are nonspecific due to the variable degree of intussusception and obstruction of the flow of ingesta (Gaughan and Hackett, 1990; Dart et al., 1997; Martin et al., 1999). Horses may have a prolonged illness with mild to intermittent signs of colic, fever, or weight loss (Martin et al., 1999). Physical exam findings are nonspecific as well, but it is possible that the intussusception may be palpated on the right side of the abdomen or located on ultrasound examination (Gaughan and Hackett, 1990; Martin et al., 1999).

Treatment is surgical, involving manual reduction of the cecum from the intussusception followed by a typhlotomy, or amputation with the aid of a colotomy (Gaughan and Hackett, 1990; Martin et al., 1999; Hubert et al., 2000; Rakestraw and Hardy, 2006). Due to the often protracted nature of the disease, reduction may be impossible. A complete bypass is then performed using an ileocolostomy or jejunocolostomy with or without amputation of the cecum within the colon (Tyler, 1992; Ward and Fubini, 1994; Boussauw et al., 2001; Lores and Ortenburger, 2008).

Depending on the procedure performed, refeeding after intussusception can begin as soon as 24 hours after surgery (Rakestraw and Hardy, 2006). However, if a bypass procedure was elected, feeding should be delayed for 36-48 hours due to the association of refeeding with signs of colic (Gerard et al., 1996). Water should be offered initially, and if the horse tolerates liquids, feed can be offered in a manner similar to horses after a large colon impaction. Small quantities of good-quality hay are provided (0.5 kg or 1.1 pounds, 4-6 times daily) and slowly increased over 2-3 days until the horse is on a full ration. Alternatives to hay include pelleted complete diets or alfalfa. Grazing should be encouraged, starting at 15 minutes every 4 hours, due to its effect on cecal motility (Ross et al., 1990). Concentrates should be avoided for 10-14 days to prevent any negative effects on microbial populations.

Descending (Small) Colon Obstructions

Fecal impactions are the most common cause of obstruction of the small colon, alongside enteroliths in regions that are endemic (see previous section for specific information on enteroliths) (Dart et al., 1992; Edwards, 1997; Rhoads et al., 1999). Risk factors for fecal impaction are similar to those that result in impactions of other portions of the gastrointestinal tract. Poor-quality roughage, poor dentition, parasitic damage, inactivity, dehydration, and motility disorders have all been implicated as contributing factors (Ruggles and Ross, 1991; Dart et al., 1992; Rhoads et al., 1999a; Schumacher and Mair, 2002). It is important to note that horses with small colon impactions have an increased risk of salmonellosis, but it remains unknown whether the diarrhea that is a common complication during or after a small colon impaction is the cause or result of this infection (Ruggles and Ross, 1991, Rhoads et al., 1999; Frederico et al., 2006).

Clinical signs of descending colon obstruction may include reduced fecal output, mild to moderate abdominal pain, abdominal distention, small volume diarrhea or scant feces, fever, and straining to defecate (Ruggles and Ross, 1991; Rhoads et al., 1999). Diagnosis of obstruction is typically

made on palpation, with a tubular impaction or foreign body noted in the lumen of the descending colon. If the obstruction is more proximal, or in the transverse colon, only gas distention of the ascending colon and cecum will be noted on palpation. Radiographs may be helpful in visualizing enteroliths; however, they are not always visible on the films (Yarborough et al., 1994).

For feed impactions of the small colon, medical therapy includes IV and oral fluids, laxatives, and analgesics. Antibiotics or antiserum may be indicated if the horse is leukopenic or endotoxic. (Rhoads et al., 1999). Surgery is indicated if the horse is unresponsive to medical therapy or analgesics, abdominal distention increases, if the mass is believed to be a foreign body or enterolith, or if the abdominal fluid is abnormal. During a ventral midline celiotomy, a high enema can be administered to lavage the impaction with the assistance of extraluminal massage. An enterotomy can also be performed to remove the impaction directly if extraluminal massage does not work. If a foreign body or enterolith is present, it can be delivered, per rectum, with the aid of the enema. If it cannot be mobilized, an enterotomy in the taenia of the descending colon can be performed for removal, or in the pelvic flexure if the obstruction can be retropulsed into the colon (Taylor and Hillyer, 1979). In addition, a large colon enterotomy should be performed to remove the majority of fecal matter proximal to the obstruction to reduce the risk of reimpaction (Plummer, 2009).

If medical therapy resolves the small colon impaction, noted by adequate fecal output, feed can be reintroduced slowly in a manner similar to ascending colon impactions (see previous section). Water is typically allowed for the first 12-24 hours after resolution, followed by a slow reintroduction to feed. The main difference between refeeding an ascending and descending colon impaction is that pelleted diets are preferred initially to prevent reimpaction. Hay is generally reintroduced 3-4 days after resolution of the impaction (Frederico et al., 2006).

Horses treated surgically for descending colon obstruction are reintroduced to feed in a more conservative manner. This portion of the colon is more prone to stricture and dehiscence after enterotomies due to the size of the lumen, the high concentration of collagenase enzymes, and the mechanical stress of fecal balls passing through the site (Hawley et al., 1970; Stashak, 1982). It is also important to note that ileus may contribute to reimpaction, since time to first defecation is significantly delayed after surgery for descending colon impaction (30-40 hours) (Prange et al., 2010). Due to this concern, horses should be held off feed for 36-48 hours after surgery, and then feed should be slowly reintroduced starting at 0.5 kg every 6 hours and increasing gradually to a full ration over 2-4 days (Frederico et al., 2006; Prange et al., 2010). Low-residue feeds such as complete diets or alfalfa pellets are preferred for the first 10-14 days after surgery to prevent reimpaction, and wetting the feed into a slurry may be beneficial (Schumacher and Mair, 2002). This diet can be continued indefinitely, if luminal size is a concern. Laxatives and oral fluids may also be helpful to maintain soft feces in the immediate post-operative period, along with a laxative diet (alfalfa or fresh grass). In addition, magnesium sulfate can be administered in the feed, at a rate of 0.2 g/kg/day.

Small colon impactions can possibly be prevented by providing good-quality roughage and adequate fresh water. Dental care should be provided, and older horses with loss of an adequate grinding surface should be provided a complete, extruded feed, rather than hay. In horses with a suspected stricture, a pelleted diet should be fed as well. Prevention of enteroliths and fecoliths are described previously. Horses that obstruct with a foreign body often outgrow the need to eat inappropriate items, but the environment should be modified to reduce the risk of ingestion. Often a good-quality pasture will reduce the consumption of foreign material (Gay et al., 1979).

Descending (Small) Colon Strangulations

Strangulation of the descending colon most commonly occurs due to a mesenteric lipoma, but volvulus, herniation, and intussusception of the colon have also been reported (Kirker-Head and Stekel, 1988; Ross et al., 1988; Dart et al., 1992; Edwards, 1997; Rhoads and Parks, 1999). Clinical signs often include moderate to severe colic and abdominal distention, but signs of discomfort are often delayed. Rectal exam is similar to small colon obstruction, in that large colon distention is noted, as well as a lack of fecal balls. Sometimes the clinician will be able to palpate the obstruction of the lumen if it is far enough caudal. The abnormal rectal findings, progressive abdominal distention, and peritoneal fluid changes will indicate the need for surgery. Treatment is resection of the affected colon as well as a pelvic flexure enterotomy to lavage ingesta form the ascending colon (Bristol and Cullen, 1988; Hanson 1988a,b). Post-operative refeeding is similar to small colon obstructions treated surgically.

References

Albengres, E., S. Urien, and J.P. Tillement, et al. 1985. Interactions between smectite, a mucus stabilizer, and acid and basic drugs. Eur. J. Clin. Pharmacol. 28:601–605.

Andrews, F.M., B.R. Buchanan, S.H. Smith, et al. 2006. In vitro effects of hydrochloric acid and various concentrations of acetic, propionic, butyric, or valeric acids on bioelectric properties of equine gastric squamous mucosa. Am. J. Vet. Res. 67(11):1873-1882.

Argenzio, R.A. 1975. Functions of the equine large intestine and their interrelationship in disease. Cornell Vet. 65:03-330.

Arroyo, L.G., H.R Stampfli, and J.S. Weese. 2006. Potential role of *Clostridium difficile* as a cause of duodenitis-proximal jejunitis in horses. J. Med. Microbiol. 55:605-608.

Baird, A.N., N.D. Cohen, T.S. Taylor, et al. 1991. Renosplenic entrapment of the large colon in horses: 57 cases (1983–1988). J. Am. Vet. Med. Assoc. 198:1423-1426.

Bergman, E.N. 1990. Energy contributions of volatile fatty acids from the gastrointestinal tract in various species. Physiol. Rev. 70:567-590.

Beroza, G.A., W.P. Barclay, T.N. Phillips, et al. 1983. Cecal perforation and peritonitis associated with *Anaplocephala perfoliata* infection in three horses. J. Am. Vet. Med. Assoc. 183:804–806.

Bertone, A.L. 1989. Large colon resection. Vet. Clin. N. Am.-Equine 5(20):377-393.

Bertone, A.L., S.L. Ralston, and T.S. Stashak. 1989a. Fiber digestion and voluntary intake in horses after adaptation to extensive large colon resection. Am. J. Vet. Res. 50:1628–1632.

Bertone, A.L., P.J. Van Soest, and T.S. Stashak. 1989b. Digestion, fecal and blood variables associated with extensive large colon resection in the horse. Am. J. Vet. Res. 50:253–258.

Bertone, J.J., J.L. Traub-Dargatz, R.W. Wrigley, D.G. Bennett, and R.J. Williams. 1988. Diarrhoea associated with sand in the gastrointestinal tract of horses. J. Am. Vet. Med. Assoc. 193:1409-1412.

Blikslager, A.T., and S.L. Jones. 2004. Obstructive diseases of the gastrointestinal tract. In: Equine Internal Medicine. S.M. Reed, W.M. Bayly, and D.C. Sellon, ed. Saunders, St. Louis pp. 992-936.

Blikslager, A.T. 2005. Treatment of gastrointestinal obstruction–Stomach Impaction, ileal impaction, and cecal impaction. In: Proc. of the Am Assoc Equine Practitioners Focus Meeting, Quebec, Canada.

Blue, M.G., and R.W. Wittkopp. 1981. Clinical and structural features of equine enteroliths. J Am. Vet. Med. Assoc 179:79-82.

Boening, K., and F. von Saldern. 1986. Nonsurgical treatment of left dorsal displacement of the large colon of horses under general anesthesia. In: Proc. Equine Colic Research Symp 325.

Boussauw, B.H., R. Domingo, and H. Wilderjans, et al. 2001. Treatment of irreducible caecocolic intussusceptions in horses by jeujuno(ileo)-colostomy. Vet. Rec. 149:16-18.

Boyd, L.E., D.A. Carbonaro, and K.A. Houpt. 1988. The 24-hour time budget of Prezwalski horses. Appl. Anim. Behav. Sci. 21:5-17.

Buchanon, B.R., and F.M. Andrews. 2003. Treatment and prevention of equine gastric ulcer syndrome. Vet. Clin. N. Am.-Equine 19:575-59.

Campbell, M.L., P.C. Colahan, M.M. Brown. 1984. Cecal impaction in the horse. J. Am. Vet. Med. Assoc. 184:950–952.

Cargile, J.L., J.A. Burrow, I. Kim, et al. 2004. Effect of dietary corn oil supplementation on equine gastric fluid acid, sodium, and prostaglandin E2 content before and during pentagastrin infusion. J. Vet. Intern. Med. 18:545–549.

Chaves, M., M.W. Smith, and R.C.N. Williamson. 1987. Increased activity of digestive enzymes in ileal enterocytes adapting to proximal small bowel resection. Gut 28:981-987.

Clarke, L.L., R.A. Argenzio, and M.C. Roberts. 1990a. Effects of meal feeding on plasma volume and urinary electrolyte clearance in ponies. Am. J. Vet. Res. 51:571-576.

Clarke, L.L., M.C. Roberts, and R.A. Aregnzio. 1990b. Feeding and digestive problems in horses: Physiologic responses to a concentrated meal. Vet. Clin. N. Am.-Equine 433-450.

Cohen, N.D., P.L. Matejka, C.M. Honnas, et al. 1995. Case-control study of the association between various management factors and development of colic in horses. J. Am. Vet. Med. Assoc. 206(5):667-673.

Cohen, N.D., and C.M. Honnas. 1996. Risk factors associated with development of diarrhea in horses after celiotomy for colic: 190 cases (1990–1994). J. Am. Vet. Med. Assoc. 209:810–813.

Cohen, N.D., and J.G. Peloso. 1996. Risk factor for history of previous colic and for chronic, intermittent colic in a population of horses. J. Am. Vet. Med. Assoc. 208(1):697-703.

Cohen, N.D., P.G. Gibbs, and A.M. Woods. 1999. Dietary and other management factors associated with colic in horses. J. Am. Vet. Med. Assoc. 215(1):53-60.

Cohen, N.D., C.A. Vontur, and P.C. Rakestraw. 2000. Risk factors for enterolithiasis among horses in Texas. J. Am. Vet. Med. Assoc. 216:1787-1794.

Cohen, N.D., E. Murphy, et al. 2005. Is duodenitis-proximal jejunitis associated with high carbohydrate diets? In: Proc. Eight International Equine Colic Research Symp. Quebec City, Canada.

Colahan, P. 1987. Sand colic. In: Current Therapy in Equine Medicine II. N.E. Robinson, ed. W.B. Saunders, Philadelphia, pp. 55-58.

Collatos, C., and S. Romano. 1993. Cecal impaction in horses: Causes, diagnosis and medical treatment. Comp. Cont. Educ. Pract. 15:976–981.

Craig, D.R., R.L. Pankowski, B.D. Car, et al. 1987. Ileocolostomy: A technique for surgical management of equine cecal impactions. Vet. Surg. 16:451-455.

Crowell-Davis, S.L., K.A. Houpt, and J. Carnevale. 1985. Feeding and drinking behaviour of mares and foals with free access to pasture and water. J. Anim. Sci. 60:883–889.

Dabareiner, R.M., and N.A. White. 1995. Large colon impactions in horses: 147 cases (1985-1991). J. Am. Vet. Med. Assoc. 206:679-685.

Dabareiner, R., and N.A. White. 1997. Diseases and surgery of the cecum. Vet. Clin. N. Am.-Equine 13:303-315.

Dart, A.J., J.R. Snyder, J.R. Pascoe, et al. 1992. Abnormal conditions of the equine descending (small) colon: 102 cases (1979-1989). J. Am. Vet. Med. Assoc. 200:971-978.

Dart, A.J., D.R. Hodgson, J.R.E. Snyder. 1997. Caecal disease in equids. Aust. Vet. J. 75: 552-557.

de Fombelle, A., V. Julliand, C. Drogoul, et al. 2001. Feeding and microbial disorders in horses: 1. Effects of an abrupt incorporation of two levels of barley in a hay diet on microbial profile and activities. J. Equine Vet. Sci. 21:439-445.

Driscoll, N., P. Baia, A.T. Fischer, T. Brauer, A. Klohnen. 2008. Large colon resection and anastomosis in horses: 52 cases. Equine Vet. J. 40(4):342-347.

Drogoul, C., A. de Fombelle, and V. Julliand. 2001. Feeding and microbial disorders in horses 2: Effect of three hay:grain ratios on digesta passage rate and digestibility in ponies. J. Equine Vet. Sci. 26:487–491.

Dukti, S.A., S. Perkins, J. Murphy, et al. 2006. Prevalence of gastric squamous ulceration in horses with abdominal pain. Equine Vet. J. 38(4):347-349.

Durham, A.E., T.J. Phillips, J.P. Walmsley, and J.R. Newton. 2004. Nutritional and clinicopathological effects of post-operative parenteral nutrition following small intestinal resection and anastomosis in the mature horse. Equine Vet. J. 36(5):390-396.

Durham, A.E. 2009. The role of nutrition in colic. Vet. Clin. N. Am.-Equine 25:67-78.

Dyer, J., E. Fernandez-Castano Merediz; S.H. Salmon, et al. 2002. Molecular characterization of carbohydrate digestion and absorption in equine small intestine. Equine Vet. J. 34(4):349-358.

Edens, L.M., and J.L. Cargile. 1997. Medical management of colic. In: Current Therapy in Equine Medicine IV. N.E. Robinson, ed. W.B. Saunders, Philadelphia, pp. 182-191.

Edwards, G. 1992. The clinical presentation and diagnosis of caecal obstruction in the horse. Equine Vet. Educ. 4:237-240.

Edwards, G.B. 1997. Diseases and surgery of the small colon. Vet. Clin. N. Am.-Equine 13:359-375.

Edwards, G.B. 2000. Duodenitis-proximal jejunitis (anterior enteritis) as a surgical problem. Equine Vet Educ. 12:411-414.

Edwards, G.B. 2002. Caecal diseases that result in colic. In: Manual of Equine Gastroenterology. T. Mair, T. Divers, and N. Ducharme, ed. W.B. Saunders, London, pp. 267-278.

Ellis, C.M., T.M. Lynch, D.E. Slone, F.E. Hughes, and C.K. Clark. 2008. Survival and complication safer large colon resection and end-to end anastomosis for strangulating large colon volvulus in seventy three horses. Vet. Surg. 37:786-790.

Embertson, R.M., P.T. Colahan, M.P. Brown, et al. 1985. Ileal impaction in the horse. J. Am. Vet. Med. Assoc. 186(6):570-572.

Ethell, M.T., A.J. Dart, D.R. Hodgson, and R.J. Rose. 1993. Alimentary System. In: Manual of Equine Practice, R.J. Rose and D.R. Hodgson, ed. W.B. Saunders, Philadelphia, p. 319.

Farstvedt, E., and D. Hendrickson. 2005. Laparoscopic closure of the nephrosplenic space for prevention of recurrent nephrosplenic entrapment of the ascending colon. Vet. Surg. 34:642-645.

Feige, K., A. Furst, and M.W. Eser. 2002. Effects of housing, feeding, and use on equine health with emphasis on respiratory and gastrointestinal disease. Schweiz. Arch. Tierh. 144(7):348–355.

Frank, N., F.M. Andrews, S.B. Elliott, and J. Lew. 2006. Effects of dietary oils on the development of gastric ulcers in mares. Am. J. Vet. Res. 66:2006-2011.

Freeman, D.E. 2000. Duodenitis-proximal jejunitis. Equine Vet. Educ. 12:415-426.

Freeman, D.E., P. Hammock, B.T. Goetz, J.H. Foreman, D.J. Schaeffer, R.A. Richter, O. Inoue, and J.H. Magid. 2000. Short- and long-term survival and prevalence of postoperative ileus after small intestinal surgery in the horse. Equine Vet. J. Suppl. 32:42-51.

Freeman, D.E., and D.J. Schaeffer. 2010. Comparison of complications and long-term survival rates following hand-sewn versus stapled side-to-side jejunocecostomy in horses with colic. J. Am. Vet. Med. Assoc. 237:1060-1067.

Frederico, L.M., S.L. Jones, and A.T. Blikslager. 2006. Predisposing factors for small colon impaction in horses and outcome of medial and surgical treatment: 44 cases (1999-2004). J. Am. Vet. Med. Assoc. 229(10):1612-1616.

Gaughan, E.M., and R.P. Hackett. 1990. Cecocolic intussusceptions in horses: 11 cases (1979-1989). J. Am. Vet. Med. Assoc. 197:1373-1375.

Gaughan, E.M., and P. van Harreveld. 2000. Cecocecal and cecocolic intussusceptions in horses. Comp. Cont. Educ. Pract. 22:616-621.

Gay, C.C., V.C. Spiers, B.A. Christie, et al. 1979. Foreign body obstruction of the small colon in six horses. Equine Vet. J. 11:60-63.

Gerard, M.P., K.F. Bowman, A.T. Blikslager, et al. 1996. Jejunocolostomy or ileocolostomy for treatment of cecal impactions in horses: nine cases (1985-1995). J. Am. Vet. Med. Assoc. 209:1287-1290.

Gillis, J.P., T.S. Taylor, and M.J. Puckett. 1994. Gastrojejunostomy for management of acute proximal enteritis in a horse. J. Am. Vet. Med. Assoc. 204:633-635.

Geor, R.J. 2001. Nutritional support of the sick adult horse. In: Advances in Equine Nutrition II, J.D. Pagan and R.J. Geor, ed. Nottingham University Press, Nottingham, U.K., pp. 403-418.

Geor, R.J. 2005. Nutritional considerations for the colic patient. In: Proc. Am Assoc Equine Pract Focus Meeting, Quebec, pp. 55-64.

Geor, R.J. 2007. Feeding management of horses recovering from colic. Comp. Cont. Educ. Pract. 2(6):344-355.

Goodson, J., W.J. Tyznik, J.H. Clin, et al. 1988. Effects of an abrupt diet change from hay to concentrate on microbial numbers and physical environment in the cecum of the pony. Appl. Environ. Microbiol. 54:1946–1950.

Granot, N., J. Milgram, T. Bdolah-Abram, I. Shemesh, and A. Steinman. 2008. Surgical management of sand impactions in horses: A retrospective study of 41 cases. Aust. Vet. J. 86:404-407.

Griffiths, N.J., J.R. Walton, and G.B. Edwards. 1997. An investigation of the toxigenic types of *Clostridium perfringens* in horses with anterior enteritis: Preliminary results. Anaerobe 3:121–125.

Hackett, R.P. 2002. Displacement of the large colon. In: Manual of Equine Gastroenterology. T.S. Mair, T.J. Divers, and N.G. Ducharme, ed. W.B. Saunders, St. Louis, pp. 284-288.

Hallowell, G. 2008. Retrospective study assessing efficacy of treatment of large colonic impactions. Equine Vet. J. 40(4):411-413.

Hammock, P.D., D.E. Freeman, and G.J. Baker. 1998. Failure of psyllium mucilloid to hasten evacuation of sand from the equine large intestine. Vet. Surg. 27:547-554.

Hance, S.R., and R.M. Embertson. 1992. Colopexy in broodmares: 44 cases (1986-1990). J. Am. Vet. Med. Assoc. 201(5):782-787.

Hanson, R.R., J, Schumacher, J. Humberg, et al. 1996. Medical treatment of horses with ileal impaction: 10 cases (1990-1994). J. Am. Vet. Med. Assoc. 208:898-900.

Hanson, R.R., A.J. Nixon, M. Calderwood-Mays, et al. 1988a. Evaluation of three techniques for end-to-end anastomosis of the small colon in horses. Am. J. Vet. Res. 49:1613-1620.

Hanson, R.R., A.J. Nixon, M. Calderwood-Mays, et al. 1988b. Comparison of staple and suture techniques for end-to-end anastomosis of the small colon in horses. Am. J. Vet. Res. 49:1621-1628.

Hanson, R.R., J.C. Wright, J. Schumacher, et al. 1998. Surgical reduction of ileal impaction in the horse: 28 cases. Vet. Surg. 27:555-560.

Hardy, J., M. Minton, J.T. Robertson, et al. 2000. Nephrosplenic entrapment in the horse: A retrospective study of 174 cases. Equine Vet. J. Suppl. 32:95-97.

Hardy, J. 2008. Specific diseases of the large colon. In: The Equine Acute Abdomen. N.A. White, J.N. Moore, and T.S. Mair, ed. Teton New Media, Jackson, pp. 628-644.

Harrison, I.W. 1988. Equine large intestinal volvulus: A review of 124 cases. Vet. Surg. 77-81.

Hassel, D.M., D. Langer, J.R. Snyder, et al. 1999. Evaluation of enterolithiasis in horses: 900 cases (1973-1996). J. Am. Vet. Med. Assoc. 214:226-230.

Hassel, D.M., P.S. Schiffman, and J.R. Snyder. 2001. Petrographic and geochemic evaluation of equine enteroliths. Am. J. Vet. Res. 62:350–358.

Hassel, D.M. 2002. Enterolithiasis. Clin. Tech. Equine Pract. 1(3):143-147.

Hassel, D.M., P.C. Rakestraw, I.A. Gardner, et al. 2004. Dietary risk factors and colonic pH and mineral concentrations in horses with enterolithiasis. Am. J. Vet. Int. Med. 18:346-349.

Hassel, D.M., B.M. Aldridge, C.M. Drake, and J.R. Snyder. 2008. Evolution of dietary and management risk factors for enterolithiasis among horses in California. Res. Vet. Sci 85:476-480.

Hassel, D.M. S.J. Spier, B.M. Aldridge, M. Watnik, R.A. Argenzio, J.R. Snyder. 2009a. Influence of diet and water supply on mineral content and pH within the large intestine of horses with enterolithiasis. Vet. J. 182:44-49.

Hassel, D.M., P.A. Smith, J.E. Nieto, P. Beldomenico, and S.J. Spier. 2009b. Di-tri-octahedral smectite for the prevention of post-operative diarrhea in equids with surgical disease of the large intestine: Results of a randomized clinical trial. Vet. J. 182:210-214.

Hawley, P.R., W.P. Faulk, and T.K. Hunt, et al. 1970. Collagenase activity in the gastrointestinal tract. Brit. J. Surg. 57:896-900.

Hillyer, M.H., F.G.R. Taylor, C.J. Proudman, et al. 2002. Case control study to identify risk factors for simple colonic obstruction and distension colic in horses. Equine Vet. J. 34(5):455-463.

Hintz, H.F., and R.G. Loy. 1966. The effects on pelleting on the nutritive value of horse rations. J. Anim. Sci. 25:1059-1062.

Hintz, H.F., R.A. Argenzio, and H.F. Schryver. 1971. Digestion coefficients, blood glucose levels and molar percentage of volatile acids in intestinal fluid of ponies fed varying forage-grain ratios. J. Anim. Sci. 33:992–995.

Hintz, H.F., J.E. Lowe, P. Livesay-Wilkins, et al. 1988. Studies on equine enterolithiasis. In: Proc. Am. Assoc. Equine Pract. 24:53-59.

Hintz, H.F., T.M. Hernandez, V. Soderholm, et al. 1989. Effect of vinegar supplementation on pH of colonic fluid. In: Proc. Equine Nutr. Physiol. Symp.11:116–118.

Hintz, H.F. 2000. Equine nutrition update. In: Proc. Am. Assoc. Equine Pract. 46:62-79.

Hoffman, R.M., J.A. Wilson, W.L. Kronfeld, et al. 2001. Hydrolyzable carbohydrates in pasture, hay, and horse feed: Direct assay and seasonal variation. J. Anim. Sci. 79:500-506.

Hotwagner, K., and C. Iben. 2008. Evacuation of sand from the equine intestine with mineral oil, with and without psyllium. J. Anim. Physiol. An. N. 92:86-91.

Hubert, J.D., J. Hardy, S.J. Holcombe, et al. 2000. Cecal amputation via a right ventral colon enterotomy for correction of nonreducible cecocolic intussusception in 8 horses. Vet. Surg. 29:317-325.

Hudson, J.M., N.D. Cohen, P.G. Gibbs, et al. 2001. Feeding practices associated with colic in horses. J. Am. Vet. Med. Assoc. 219:1419-1425.

Hughes, F.E., and D.E.J. Slone. 1997. Large colon resection. Vet. Clin. N. Am.-Equine 13:341-350.

Hunter, L.C., J.K Miller, and I.R. Poxton. 1999. The association of *Clostridium botulinum* type C with equine grass sickness: A toxicoinfection? Equine Vet. J. 31:492-499.

Huskamp, B. 1985. Diagnosis of gastroduodenitis and its surgical treatment by a temporary duodenocaecostomy. Equine Vet. J. 17(4):314-316.

Huskamp, B. 1987. Displacement of the large colon. In: Current Therapy in Equine Medicine, Second Edition. N.E. Robinson, ed. W.B. Saunders, Philadelphia, pp. 60-65.

Huskamp, B., and W. Scheidemann. 2000. Diagnosis and treatment of chronic recurrent caecal impaction. Equine Vet. J. Suppl. 32:65-68.

Hussein, H.S., L.A. Vogedes, G.C. Fernandez, et al. 2004. Effects of cereal grain supplementation on apparent digestibility of nutrients and concentrations of fermentation end-products in the feces and serum of horses consuming alfalfa cubes. J. Anim. Sci. 82:1986–1996.

Husted, L., L.C. Sanchez, S.N. Olsen, K.E. Baptiste, and A.M. Merritt. 2008. Effect of paddock versus stall housing on 24 hour gastric pH within the proximal and ventral equine stomach. Equine Vet. J. 40(4):337-341.

Johnston, J.K., and D.D. Morris. 1987. Comparison of duodenitis/proximal jejunitis and small intestinal obstruction in horses: 68 cases (1977-1985). J. Am. Vet. Med. Assoc. 191:849-854.

Jonsson, H., and A. Egenvall. 2006. Prevalence of gastric ulceration in Swedish Standardbreds in race-training. Equine Vet. J. 38:209-213.

Julliand, V., A. de Fombelle, C. Drogoul, et al. 2001. Feeding and microbial disorders in horses: 3. Effects of three hay:grain rations on microbial profiles and activities. J. Equine Vet. Sci. 21:543-546.

Kalsbeek, H.C. 1989. Further experiences with nonsurgical correction of nephrosplenic entrapment of the colon in the horse. Equine Vet. J. 6:442-443.

Kendall, A., C. Ley, A. Egenvall, and J. Brojer. 2008. Radiographic parameters for diagnosing sand colic in horses. Acta. Vet. Scand. 20:17-23.

Kienzle, E., S. Radicke, E. Landes, et al. 1994. Activity of amylase in the gastrointestinal tract of the horse. J. Anim. Physiol. An. N. 72:234-241.

Kirker-Head, C., and R. Stekel. 1988. Volvulus of the small colon in a horse. Mod. Vet. Pract. 69:14-16.

Korolainen, R., and M. Ruohoniemi. 2002. Reliability of ultrasonography compared to radiography in revealing intestinal sand accumulations in horses. Equine Vet. J. 34(5):499-504.

Lee, R.D., G. Harris, and T.R. Murphy. 2010. Bermudagrasses in Georgia. In: University of Georgia Cooperative Extension Service Bulletin, number B911. Athens, GA, pp. 1-11.

le Jeune, S.S., J.E. Nieto, J.E. Dechant, and J.R. Snyder. 2009. Prevalence of gastric ulcers in Thoroughbred broodmares in pasture: A preliminary report. Vet. J. 181:251-255.

Lester, G.D., J.R. Bolton, L.K. Cullen, et al. 1992. Effects of general anesthesia on myoelectric activity of the intestine in horses. Am. J. Vet. Res. 52:1553-1557.

Lester, GD. 2004. Gastrointestinal diseases of performance horses. In: Equine Sports Medicine and Surgery. K.W. Hinchcliff, A.J. Kaneps, and R.J. Geor, ed. Saunders Elsevier, Philadelphia, pp. 1037-1043.

Lewis, L.D. 1995. Sick horse feeding and nutritional support. In: Equine Clinical Nutrition. L.D. Lewis, ed. Lea and Febiger, Baltimore, p. 396.

Little, D., W.R. Redding, and A.T. Blikslager. 2001. Risk factors for reduced postoperative fecal output in horses: 37 cases (1997-1998). J. Am. Vet. Med. Assoc. 218:414-420.

Little, D., and A.T. Blikslager. 2002. Factors associated with development of ileal impaction in horses with surgical colic: 78 cases (1986-2000). Equine Vet. J. 34(5):464-468.

Lieb, S. 1997. Sand removal from the GI tract of equine. In: Proc. Fifteenth Equine Nutr. Physiol. Symp., p. 335.

Lloyd, K., H.F. Hintz, J.D. Wheat, et al. 1987. Enteroliths in horses. Cornell Vet. 77:172-186.

Longland, A.C., A.J. Cairns, and M.O. Humphreys. 1999. Seasonal and diurnal changes in fructan

concentration in *Lolium perenne*: Implications for the grazing management of equine predisposed to laminitis. In: Proc. Equine Nutr. Physiol. Soc. Symp., pp. 258-259.

Longland, A.C., and B.M. Byrd. 2006. Pasture nonstructural carbohydrates and equine laminitis. J. Nutr. 136:2099S–2102S.

Lopes, M.A., G.S. Moura, and J.D. Filho. 1999. Treatment of large colonic impaction with enteral fluid therapy. In: Proc. Am. Assoc. Equine Pract., pp. 99-102.

Lopes, M.A., N.A. White, and M.V. Crisman, et al. 2004. Effects of feeding large amounts of grain on colonic contents and feces in horses. Am. J. Vet. Res. 65:687-694.

Lorenzo-Figueras, M., S.M. Morisset, J. Morisset, J, et al. 2007. Digestive enzyme concentration and activities in healthy pancreatic tissue of horses. Am. J. Vet. Res. 68:1070-1072.

Lores, M., and A. Ortenburger. 2008. Use of cecal bypass via side-to-side ileocolic anastomosis without ileal transaction for treatment of cecocolic intussusception in three horses. J. Am. Vet. Med. Assoc. 232:574-577.

Luthersson, N., K. Hou Nielsen, P. Harris, and T.D.H. Parkin. 2009. Risk factors associated with equine gastric ulceration syndrome (EGUS) in 201 horses in Denmark. Equine Vet. J. 41:625-630.

Lybbert, T., P. Gibbs, N. Cohen, et al. 2007. Feeding alfalfa hay to exercising horses reduces the severity of gastric squamous mucosal ulceration. In: Proc. Am. Assoc. Equine Pract., pp. 525-526.

Markel, M.D. 1989. Prevention of large colon displacements and volvulus. Vet. Clin. N. Am.-Equine 5(2):395-405.

Martin, B.B.J., D.E. Freeman, M.W. Ross, et al. 1999. Cecocecal and cecocolic intussusceptions in horses: 30 cases (1976-1996). J. Am. Vet. Med. Assoc. 214:80-84.

McCarthy, H.E., N.P. French, G.B. Edwards, et al. 2004. Equine grass sickness is associated with low antibody levels to *Clostridium botulinum*: A matched case-control study. Equine Vet. J. 36:123-129.

McClave, S.A., R.G. Martindale, V.W. Vanek, et al. 2009. Guidelines for the provision and assessment of nutrition support therapy in the adult critically ill patient. Society of Critical Care Medicine (SSCM) and American Society for Parenteral and Enteral Nutrition (A.S.P.E.N.). Jpen-Parenter. Enter. 33:277-316.

McConnico, R.S. 2004. Duodenitis-proximal jejunitis (anterior enteritis, proximal enteritis). In: Equine Internal Medicine, 2nd ed., S.M. Reed, W.M. Bayly, and D.C. Sellon, ed. Saunders Elsevier, St. Louis, pp. 873-878.

Medina, B., I.D. Girard, E. Jacotot, et al. 2002. Effect of a preparation of *Saccharomyces cerevisiae* on microbial profiles and fermentation patterns in the large intestine of horses fed a high fiber or a high starch diet. J. Anim. Sci. 80:2600-2609.

Mehdi, S., and V. Mohammad. 2006. A farm-based prospective study of equine colic incidence and associated risk factors. J. Equine Vet. Sci. 26(4):171-174.

Merritt, A.M., J. Robbins, and B. Brewer. 1982. Is salmonella infection a cause of the acute gastric dilitation/ileus syndrome in horses? In: Proc. Equine Colic Research Symposium, pp. 119-124.

Merritt, A.M. 2003. The equine stomach: A personal perspective. In: Proc. Am. Assoc. Equine Pract. 49:75-102.

Morris, D., J. Morris, and S. Ward. 1986. Comparisons of age, breed, history and management in 229 horses with colic. Equine Vet. J. Suppl. 7:129-133.

Murray, R.C., G.M. Constantinescu, E.M. Green. 1992. Equine enteroliths. Comp. Cont. Educ. Pract. 14:1104-1113.

Murray, M.J., and G.F. Schusser. 1993. Measurement of 24-h gastric pH using an indwelling pH electrode in horses unfed, fed, and treated with ranitidine. Equine Vet. J. 25:417-421.

Murray, M.J. 1994. Equine model of inducing ulceration in alimentary squamous epithelial mucosa. Digest. Dis. Sci. 12:2530-2535.

Murray, M.J., and E.S. Eichorn. 1996. Effects of intermittent feed deprivation, intermittent feed deprivation with ranitidine administration, and stall confinement with ad libitum access to hay on gastric ulceration in horses. Am. J. Vet. Res. 57(11):1599-1603.

Nadeau, J.A., F.M. Andrews, A.G. Mathew, et al. 2000. Evaluation of diet as a cause of gastric ulcers in horse. Am. J. Vet. Res. 61:784-790.

Nadeau, J.A., F.M. Andrews, C.S. Patton, et al. 2003a. Effects of hydrochloric, acetic, butyric and propionic acids on pathogenesis of ulcers in the non-glandular portion of the stomach of horses. Am. J. Vet. Res. 64(4):404-412.

Nadeau, J.A., F.M. Andrews, S.C. Patton, et al. 2003b. Effects of hydrochloric, valeric and other volatile fatty acids on pathogenesis of ulcers in the non-glandular portion of the stomach of horses. Am. J. Vet. Res. 64:413–417.

O'Connor, T.P., M.M. Lam, and J. Diamond. 1999. Magnitude of functional adaptation after intestinal resection. Am. J. Physiol. 276:R1265-R1275.

Parks, A.H, R.E. Doran, N.A. White, et al. 1989. Ileal impaction in the horse: 75 cases. Cornell Vet. 79:83-91.

Plummer, A., P.C. Rakestraw, J. Hardy, et al. 2007. Outcome of medical and surgical treatment of cecal impaction in horses: 114 cases (1994-2004). J. Am. Vet. Med. Assoc. 231(9):1378-1385.

Plummer, A. 2009. Impactions of the small and large intestines. Vet. Clin. N. Am.-Equine 25:317-327.

Potter, G.D., F.F. Arnold, D.D. Householder, et al. 1992. Digestion of starch in the small or large intestine of the equine. Pferdeheilkunde 1:107-111.

Poxton, I.R., L.C. Hunter, R. Brown, et al. 1997. *Clostridia* and equine grass sickness. Rev. Med. Microbiol. 8:S49-S51.

Prange, T., S.J. Holcombe, J.A. Brown, et al. 2010. Resection and anastomosis of the descending colon in 43 horses. Vet. Surg. 39:748-753.

Proudman, C.J., and G.B. Edwards. 1993. Are tapeworms associated with equine colic? A case control study. Equine Vet. J. 25:224-226.

Proudman, C.J., N.P. French, and A.J. Trees. 1998. Tapeworm infection is a significant risk factor for spasmodic colic and ileal impaction colic in the horse. Equine Vet. J. 30:194-199.

Pugh, D.G., and J.T. Thompson. 1992. Impaction colics attributed to decreased water intake and feeding coastal Bermuda grass hay in a boarding stable. Equine Pract. 14:9-14.

Quigley, E.M., and J.S. Thompson. 1993. The motor response to intestinal resection: Motor activity in the canine small intestine following distal resection. Gastroenterology 105:791-798.

Quinteros, D.D., J.M. Garcia-Lopez, and P.J. Provost. 2010. Complete caecal bypass without ileal transaction for caecal impaction in horses: Seven clinical cases (1997-2007). Aust. Vet. J. 88:434-438.

Rabuffo, T.S., E.S. Hackett, N. Grenager, R. Boston, and J.A. Orsini. 2009. Prevelance of gastric ulcerations in horses with colic. J. Equine Vet. Sci. 29(6):540-545.

Ragle, C.A., D.M. Meagher, C.A. Lacrox, et al. 1989a. Surgical treatment of sand colic: results in 40 horses. Vet. Surg.18:48-51.

Ragle, C.A., D.M. Meagher, J.L. Schrader, and C.M. Honnas. 1989b. Abdominal auscultation in the detection of experimentally induced gastrointestinal sand accumulation. J. Vet. Int. Med. 3(1):12-14.

Rakestraw, P.C., and J. Hardy. 2006. Large intestine. In Equine Surgery, 3rd ed. J.A. Auer and J.A. Stick, ed. Saunders, St. Louis, pp. 436-478.

Ralston, S.L., K.E. Sullins, and T.S. Stashak. 1986. Digestion in horses after resection of ischemic insult to the large colon. Am. J. Vet. Res. 47(10):2290-2293.

Reese, R.E., and F.M. Andrews. 2009. Nutrition and dietary management of equine gastric ulcer syndrome. Vet. Clin. N. Am.-Equine 25:79-92.

Reeves, M.J., M.D. Salman, and G. Smith. 1996. Risk factors for equine acute abdominal disease (colic): Results from a multi-center case-control study. Prev. Vet. Med. 26:285-301.

Rhoads, W., M. Barton, and A.H. Parks. 1999a. Comparison of medical and surgical treatment for impaction of the small colon in horses: 84 cases (1986-1996). J. Am. Vet. Med. Assoc. 214(7):1042-1047.

Rhoads, W.S., and A.H. Parks. 1999b. Incarceration of the small colon through a rent in the gastrosplenic ligament in a pony. J. Am. Vet. Med. Assoc. 214:226-228.

Robb, J., D.L. Doxey, E.M. Milne, et al. 1997. The isolation of potentially toxigenic fungi from the environment of horses with grass sickness and mal seco. In: Grass Sickness, Equine Motor Neuron Disease and Related Disorders. Proc. Internat Workshop. C. Hahn, V. Gerber, C. Herholz and I.G. Mayhew, ed. Newmarket, Equine Vet. J., pp. 52-541.

Roberts, C.T., and D.E. Slone. 2000. Caecal impactions managed surgically by typhlotomy in 10 cases (1988–1998). Equine Vet. J. Suppl. 32:74-76.

Roberts, M.C. 1974. Amylase activity in the small intestine of the horse. Res. Ve.t Sci. UK 17:400-401.

Ross, M.W., P.R. Stephens, and J.M. Reinmer. 1988. Small colon intussusception in a broodmare. J. Am. Vet. Med. Assoc. 192:372-374.

Ross, M.W., K.K. Cullin, and J.A. Rutkowski. 1990. Myoelectric activity of the ileum, cecum and right ventral colon in ponies during interdigestive, nonfeeding, and digestive periods. Am. J. Vet. Res. 51:561-566.

Ruggles, A., and M.W. Ross. 1991. Medical and surgical management of small colon impaction in horses: 28 cases (1984-1989). J. Am. Vet. Med. Assoc. 199(12):1762-1766.

Ruohoniemi, M., R. Kaikkonen, M. Raekallio, and L. Luukkanen. 2001. Abdominal radiography in monitoring the resolution of sand accumulations from the large colon of horses treated medically. Equine Vet. J. 33(1):50-64.

Sanchez, L.C. 2004. Diseases of the stomach. In: Equine Internal Medicine, 2nd Ed. S.M. Reed, W.M. Bayly, and D.C. Sellon, ed. Saunders Elsevier, St. Louis, pp. 863-873.

Santschi, E.M., D.E. Slone, and W.M. Frank. 1993. Use of ultrasound for diagnosis of left dorsal displacement of the large colon and monitoring its nonsurgical correction. Vet. Surg. 22(4):281-284.

Schumacher, J., T.L. Seahorn, and N.D. Cohen. 1994. Duodenitis/proximal jejunitis in horses. Comp. Cont. Educ. Pract. Vet. 16:1197-1206.

Schumacher, J., J. Mullen, R. Shelby, et al. 1995. An investigation of the role of *Fusarium moniliforme* in duodenitis/proximal jejunitis of horses. Vet. Hum. Toxicol. 37:39-45.

Schumacher, J., and T. Mair. 2002. Small colon obstruction in the mature horse. Equine Vet. Educ. 14(1):19-28.

Shirazi-Beechey, S.P. 1995. Molecular biology of intestinal glucose transport. Nutr. Res. Rev. 8:27-41.

Shirazi-Beechey, S.P. 2008. Molecular insights into dietary induced colic in the horse. Equine Vet. J. 40(4):414-421.

Smith, L.C.R., R.J. Payne, S.J. Boys Smith, A.P. Bathe, T.R.C. Greet. 2010. Outcome and long-term follow-up of 20 horses undergoing surgery for caecal impaction: A retrospective study (2000-2008). Equine Vet. J. 42(5):388-392.

Smith, L.J., and T.J. Mair. 2010. Are horses that undergo an exploratory laparotomy for correction of a right dorsal displacement of the large colon predisposed to post-operative colic, compared to other forms of large colon displacement? Equine Vet. J. 42(1):44-46.

Smyth, G.B., D.W. Young, and L.S. Hammon. 1988a. Effects of diet and feeding on past-prandial serum gastrin and insulin concentrations in adult horses. Equine Vet. J. Suppl 7:56-59.

Snyder, J.R., H.J. Olander, and J.R. Pascoe, et al. 1988. Morphologic alterations observed during experimental ischemia of the equine large colon. Am. J. Vet. Res. 49:801-809.

Snyder, J.R., J.R. Pascoe, H.J. Olander, et al. 1989. Strangulating volvulus of the ascending colon in horse. J. Am. Vet. Med. Assoc. 195:757-764.

Specht, T.E., and P.T. Colahan. 1988. Surgical treatment of sand colic in equids: 48 cases (1978-1985). J. Am. Vet. Med. Assoc. 193:1560-1564.

Stevens, C.E. 1978. Physiological implications of microbial digestion in the large intestine of mammals: relation to dietary factors. Am. J. Clin. Nutr. 31:S161-S168.

Tate, L.P., S.L. Ralston, C.M. Koch, et al. 1983. Effects of extensive resection of the small intestine in the pony. Am. J. Vet. Res. 44:1187-1191.

Taylor, F.G.R., and M.H. Hillyer. 1992. The differential diagnosis of hyperglycaemia in horses. Equine Vet. Educ. 4:135-138.

Tinker, M.K., N.A. White, C.D. Thatcher, et al. 1997. Prospective study of equine colic risk factors. Equine Vet. J. 29(6):454-458.

Tyler. R. 1992. Cecocolic intussusception in a yearling Thoroughbred filly and its surgical management by ileocolostomy. Equine Vet. Educ. 4:229-232.

Underwood, C., L.L. Southwood, and D. Knight. 2008. Complications and survival associated with surgical compared with medical management of horses with duodenitis-proximal jejunitis. Equine Vet. J. 40(4):373-378.

Vachon, A.M., and A.T. Fischer. 1995. Small-intestinal herniation through the epiploic foramen-53 cases (1987-1993). Equine Vet. J. 27:373-380.

Van Harreveld, P.D., J. Cox, and D.S. Biller. 1999. Phenylephrine HCl as a treatment of nephrosplenic entrapment in a horse. Equine Vet. Educ. 11(60):282-284.

Van Hoogmoed, L., J.R. Snyder, J.R. Pascoe, et al. 2000. Use of pelvic flexure biopsies to predict survival after large colon torsion in horses. Vet. Surg. 29:572-577.

Vatistas, N.J., R.L. Sifferman, J. Holste, et al. 1999. Induction and maintenance of gastric ulceration in horses in simulated race training. Equine Vet. J. Suppl. 29:40-44.

Videla, R., and F.M. Andrews. 2009. New perspectives in equine gastric ulcer syndrome. Vet. Clin. N. Am.-Equine 25:283-301.

Ward, J.L., and S.L. Fubini. 1994. Partial typhlectomy and ileocolostomy for treatment of nonreducible cecocolic intussusceptions in a horse. J. Am. Vet. Med. Assoc. 205:325-328.

Weese, J.S., N.M. Cote, and R.V. de Gannes. 2003. Evaluation of in vitro properties of di-tri-octahedral smectite on clostridial toxins and growth. Equine Vet. J. 35:638-641.

White, N.A., D.E. Tyler, and R.B. Blackwell. 1987. Haemorrhagic fibrinonecrotic duodenitis-proximal-jejunitis: 20 cases (1977-1984). J. Am. Vet. Med. Assoc. 190:311-315.

White, N.A. 1990. Epidemiology and etiology of colic. In: The Equine Acute Abdomen. N.A. White, ed. Lea and Febiger, Philadelphia, pp. 50-64.

White, N.A., and R.M. Dabareiner. 1997. Treatment of impaction colics. Vet. Clin. N. Am.-Equine 13:243-259.

White, N.A. 2005. Update on periopertive nutrition. In: Proc. ACVS Diplomate Resort Meeting, Bretton Woods, New Hampshire, pp. 58-61.

Yarbrough, T.B., D.L. Langer, J.R. Snyder, et al. 1994. Abdominal radiography for diagnosis of enterolithiasis in horses: 141 cases (1990-1992). J. Am. Vet. Med. Assoc. 205:592-595.

Yao-Zong, Y., L. Shi-Rong, and M. Delvaux. 2004. Comparative efficacy of dioctahedral smectite (Smecta) and a probiotic preparation in chronic functional diarrhea. Digest. Liver Dis. 36: 824-828.